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Resistance-Gene-Mediated Defense Responses against Biotic Stresses in the Crop Model Plant Tomato

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Abstract

Complex series of defense response activation, consistent with the studies conducted in the model plant *Arabidopsis thaliana*, has been demonstrated in tomato during incompatible pathogen/pest interactions. During the past two decades, numerous tomato genes have been identified conferring resistance to diverse pathogens/pests in a gene-for-gene manner. A few of these cloned resistance (*R*)-genes (*Cf* and *Pto*) have been extensively studied and excellent existing reviews describe *R*-gene function, interacting proteins and the mechanism of Avirulence effector perception. Recent comprehensive gene expression analysis of tomato responses to biotic stresses resulted in identification of genes and potential molecular processes that are associated with several of the tomato *R*-gene-mediated resistance responses. The purpose of this review is to provide an overview of tomato *R*-gene-mediated defense responses to different pathogens/pests together with the components involved in the organization of this highly complex network of plant defenses.

Keywords: Resistance-gene; Biotic stresses; *Arabidopsis thaliana*; Defense response; Tomato

Introduction

Plants are subjected to various biotic stresses throughout their sedentary life cycle. These continuous stressful conditions have prompted development of a range of defense responses including physical barriers [1-4], chemical weapons [5,6], and resistance (*R*)-gene acquisition [7-10]. Tomato (*Solanum lycopersicum*) is the second most important vegetable crop next to potato. About 170 million tons of tomatoes were produced worldwide in 2014 [11]. Due to the high nutritional value of its fruit, high yield, short life cycle, and diverse varieties and cultivars, tomato is widely grown all year round under both outdoor and indoor conditions. However, this worldwide cultivation is challenged by an abundance of diseases caused by microbial pathogens composed of fungi, bacteria, and viruses, as well as insect and nematode pests.

Two principal immune mechanisms operate against biotic stresses in plants. The first line of defense is triggered by a class of immune receptors upon recognition of pathogen associated molecular patterns (PAMPs), chemical signatures that appear to be widely conserved among certain pathogen clades [12,13]. This interaction is referred to as PAMP-triggered immunity (PTI). As part of the continuous arms race between plants and pathogens, the later have evolved to acquire effector molecules to counteract the plant PTI mechanism and ensure pathogenicity. This weakened plant immune response is known as compatible interaction. This prompted plants in turn to develop specific *R*-proteins that recognize the pathogen/pest effector(s) and initiate the second principle immune mechanism termed effector-triggered immunity (ETI) [14]. This interaction is also referred to as incompatible interaction and is generally characterized by a vast transcriptional reprogramming after recognition of the pathogen/pest effector molecule(s) [15]. The recognized effector is termed as Avirulent (Avr) and the recognition could be indirect or directly by an *R*-gene. *R*-Avr interaction typically results in a hypersensitive cell death response (HR) at the site of infection.

Starting early nineties, extensive research led to the cloning of a number of tomato *R*-genes (Table 1). These *R*-genes together with those identified from additional plant species were assigned to different classes based on the presence of various structural motifs that can be extracellular, cytoplasmic or transmembrane [16,17]. Majority of the plant cloned *R*-genes encode for nucleotide-binding domain and leucine-rich repeats (NLR) proteins with variable amino- and carboxy-terminal domains that may contain Toll/interleukin-1 receptor (TIR)- or coiled-coil (CC)-domain (Table 1). Many *R*-genes belong to gene families and are organized in tandem arrays, clusters, and super-clusters [18,19]. Interestingly, these *R*-genes with low structural diversity were shown to confer resistance to diverse pathogens and pests *via* recognition of arsenal of effectors [7]. This means that, besides the common mechanisms underlying disease resistance signal transduction throughout the plant kingdom, individual resistance gene products can act in unique signaling pathways [20]. In many plant species, it has been shown that during the course of evolution, *R*-genes have undergone gene duplication and selection pressures leading to divergent evolution. Genome-wide identification and classification of Solanaceae NLRs have identified 267, 443, and 755 NL*R*-encoding genes in tomato, potato, and pepper genomes, respectively [21]. Heinz1706 tomato encodes 478 NLRs [22].

Most of our knowledge about plant defense originated from studies conducted in the model plant *Arabidopsis thaliana* [23]. Extensive genome-wide transcriptional profiling including cDNA-AFLP [24,25], suppression subtractive hybridization (SSH) [26,27], microarrays and RNA-sequencing technologies [28-30] provided valuable insights into plant-pathogen interactions at the cellular and molecular level. The identification of genes repressed or activated in plants assisted in making novel hypotheses concerning the biology of a given interaction (both compatible and incompatible). Further analysis of the differentially regulated genes, using gene inactivation, overexpression [31], and biochemical approaches, confirmed the crucial roles for some of these genes in the plant ETI responses.

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Resistant gene	Resistance against	Resistance Source	Reference
Asc-1	*Alternaria alternata* f. sp. *lycopersici*	*S. lycopersicum*	[77]
Am	*Alfalfa mosaic virus*	*S. habrochaites*	[168]
Bs4	*Xanthomonas campestris*	*S. lycopersicum*	[169]
Cmr	*Cucumber mosaic virus*	*S. chilense*	[170]
Cf-1	*Cladosporium fulvum*	*S. lycopersicum* var cerasiforme	[166]
Cf-2		*S. pimpinellifolium*	[46]
Cf-3		*S. pimpinellifolium*	[204]
Cf-4		*S. habrochaites*	[48]
Cf-4A			[171]
Cf-4E			[172]
Cf-5		*S. lycopersicum* var cerasiforme	[45]
Cf-9		*S. pimpinellifolium*	[47]
Cf-9B			[167]
Cf-ECP1		*S. pimpinellifolium*	[173]
Cf-ECP2		*S. pimpinellifolium*	[174]
Cf-ECP4		*S. pimpinellifolium*	[173]
Cf-ECP5			[175]
Cf-ECP6			[176]
Cf-ECP7			[176]
Cf-19		*S. lycopersicum*	[177]
Frl	*Fusarium oxysporum* f.sp. *radicis-lycopersici*		[178]
Hero	*Globodera rostochiensis*	*S. pimpinellifolium*	[80]
I	*Fusarium oxysporum* formae speciales *lycopersici*	*S. pimpinellifolium*	[179]
I-1		*S. pennellii*	[180]
I-2			[52]
I-3		*S. pennellii*	[205]
Lv	*Leveillula taurica*	*S. chilense*	[181]
Mi-1.2	Meloidogyne spp., *Macrosiphum euphorbiae*, *Bemisia tabaci*, *Bactericerca cockerelli*	*S. peruvianum*	[206]
Mi-9		*S. peruvianum*	[182]
Ol-1	*Oidium neolycopersici*	*S. habrochaites*	[183]
ol-2		*S. lycopersicum* var cerasiforme	[184]
Ol-3		*S. habrochaites*	[183]
Ol-4		*S. peruvianum*	
Ol-5		*S. habrochaites*	
Ol-6		Unknown origin	
Ph-1	*Phytophthora infestans*	*S. pimpinellifolium*	[185]
Ph-2		*S. pimpinellifolium*	[186]
Ph-3		*S. pimpinellifolium*	[187]
ph-4			[188]
ph-5			M.R. Foolad et al., unpublished data
Prf	*Pseudomonas syringae* pv *tomato*		[20]
Pto		*S. pimpinellifolium*	[189]
Py-1	*Pyrenochaeta lycopersici*	*S. peruvianum*	[190]
Pot-1	*Potato virus Y*, *Tobacco etch virus*	*S. habrochaites*	[191]
Sm	*Stemphyllium solani*	*S. Pimpinelifolium*	[192]
Sw-1a	*Tomato spotted wilt virus*		
Sw-1b			
sw-2			
sw-3			
sw-4			
*Sw-5**	*Tomato spotted wilt virus*, *tomato chlorotic spot virus*, *groundnut ringspot virus*	*S. peruvianum*	[193]
Sw-6	*Tomato spotted wilt virus*	*S. peruvianum*	[194]
Sw-7		*S. chilense*	[195]
Ty-1	*Tomato yellow leaf curl virus*	*S. chilense*	[196]
Ty-2		*S. habrochaites*	[197]
Ty-3	*Tomato yellow leaf curl virus*, *Tomato mosaic virus*	*S. chilense*	[198]
Ty-4	*Tomato yellow leaf curl virus*	*S. chilense*	[197]
Ty-5		*S. peruvianum*	[199]
tcm-1	*Tomato chlorotic mottle begomovirus*	*S. lycopersicum*	[200]
tgr-1	*Tomato leaf curl virus*	*S. chilense*	[201]
*Tm-1**	*Tomato mosaic virus*	*S. habrochaites*	[202]
tm-1	*Tobacco mild green mosaic virus*, *Pepper mild mottle virus*	*S. habrochaites*	[98]
*Tm-2**	*Tomato mosaic virus*	*S. peruvianum*	[202]
Tm-22*		*S. peruvianum*	[102]
Ve1	*Verticillium dahliae*	*S. lycopersicum*	[204]

Table 1: Comprehensive list of tomato resistant genes cloned or characterized by virus-induced gene silencing.

Global transcriptome profiling is an important initial step for dissecting biological systems particularly with systems where not much is known about the molecular basis of the resistance response. The enrichment of the tomato EST databases initiated several genome-wide profiling studies [32-35]. This facilitated significant progress in the characterization of tomato incompatible responses to Avr pathogens, contributing to future gene identification and to the understanding of the potential molecular processes that are associated with the different tomato *R*-gene-mediated resistances [33]. With the advent of next generation sequencing technologies, and the tomato genome sequence [36], additional genome wide studies have been conducted. With a genome sequence and a high-density linkage and molecular maps [37], combined with being a host for numerous pathogens and pests, tomato has emerged as a powerful model system for crop plant defense response studies. Moreover, the introgression of *R*-genes from wild species into cultivated tomato provides a unique opportunity to study different resistance mechanisms against very diverse biotic agents in a single plant. In this review, we summarize the current understanding of *R*-genes in tomato and the downstream signaling components that are critical for activating defense responses. In addition, we discuss the current and future technologies that will significantly enhance our knowledge about tomato-pathogen interactions and will provide alternative strategies to develop a sustainable resistance.

Tomato Resistance Genes and their Modes of Action

Host resistance is an important component of a sustainable disease management system [38]. It is an environmentally benign method that can be used as an alternative to chemicals, as their applicability

is becoming limited due to adverse environmental and human health effects [39,40] and the emergence of resistant pathogen/pest strains [41]. Cultivated tomato, *S. lycopersicum,* has a narrow genetic base and is consequently vulnerable to many diseases and pests. On the other hand, a repertoire of genetically diverse wild tomato species represents a rich source of *R*-genes. Over the past 50 years, several race-specific disease resistant genes have been identified in wild tomato species (Table 1), and extensive tomato breeding programs have been based on the transfer of *R*-genes from wild accessions into cultivated tomato. So far, majority of the identified tomato *R*-genes conferring resistance to diverse pathogens and pests belongs the NLR class. An array of mechanisms in tomato *R*-gene-mediated resistances has been documented depending on the particular *R*-gene and pathogen/pest combination [42,43].

Fungi

Cladosporium fulvum–tomato pathosystem is a well-established model system that complies with the gene-fo*r*-gene concept first described by [44]. Elegant experiments demonstrated the involvement of pathogen effectors or Avrs in the induction of ETI post recognition by the *Cf* genes, resulting in incompatible interaction [45-48]. The *Cf* genes belong to family of LR*R*-RLP (Recepto*r*-Like Protein) encoding *R*-genes and mediate resistance against the apoplast-colonizing foliar fungal pathogen *C. fulvum.* The *Cf*-mediated resistance involves formation of cell wall appositions, callose deposition and phytoalexin accumulation. Moreover, the tomato resistance phenotype against *C. fulvum* is accompanied by HR, typically described as necrotic brown spots near the site of infection that limits further growth and spread of the pathogens [49]. About five *Avr* genes (*Avr2*, *Avr4*, *Avr4E*, *Avr5*, and *Avr9*) have been cloned and characterized from *C. fulvum*, and are recognized by the corresponding *Cf-2*, *Cf-4*, *Cf-4E*, *Cf-5*, and *Cf-9* genes (Table 1). Thus, *Cf*-mediated resistance phenotype is the combined result of HR and other defense responses. Another well-known tomato fungal pathosystem is the xylem colonizing *Fusarium oxysporum* formae speciales *lycopersici* (Fol). Resistance to Fol is mediated by *I* (Immunity)-genes that mainly involves callose deposition, accumulation of phenolics and formation of tyloses (outgrowths of xylem contact cells) and gels in the infected vessels [50]. Of the three cloned *I*-genes, only *I-2* encodes for CC-NLR (CNL) while the remaining two encode membrane associated receptor-like kinase (RLK), such as *I-3* which encodes a S-RLK, or RLP, and *I-7* encodes a LR*R*-RLP [51-53]. Three Fol effectors, Avr1 (Six4), Avr2 (Six3) and Avr3 (Six1) are recognized by *I* (and the non-allelic *I-1*), *I-2* and *I-3* genes respectively [54-56]. *I-7* confers resistance to Fol races 1, 2 and 3 and *I-7*-mediated resistance is not suppressed by Avr1 [53]. The Avr effector that recognizes *I-7* is yet to be identified. Unlike *Cf*-mediated resistance, *I*-gene-mediated resistance lacks the classical HR described above. In the vicinity of the *I-2* locus another resistance locus *Ty-1,* against *Tomato yellow leaf curl* geminivirus (TYLCV), is also mapped [57]. The *I-2* locus on chromosome 6 is one of the most divergent *R*-gene loci in tomato, partly due to gene duplications among the homologs. This diversity is also attributed to micro RNAs (miRNAs), specifically miR6024 that triggers phasiRNAs from *I-2* homologs in tomato [58]. Ouyang et al., 2014 performed deep sequencing from resistant and susceptible tomato cultivar to identify miRNAs that correlate with Fol resistance. Interestingly, they found that two miRNAs (slmiR482f and slmiR5300) were repressed in the resistant plants and these miRNAs targeted four genes with full or partial NB domains, however, *I-2* was not among these targets [59]. This suggests that there could be more *R*-genes involved in the immune signaling against Fol.

In tomato, *Ve* is a single dominant locus that confers resistance against *Verticillium.* The Ve locus contains two closely linked and inversely oriented genes, *Ve1* and *Ve2*, both encoding a RLP-type cell surface receptor. *Ve1 R*-gene provides resistance against race 1 isolates of *Verticillium* [60], by recognition of the Ave1 effector from race 1 strains of *V. dahliae* [61]. The detailed mechanism of tomato resistance against *Verticillium* wilt mediated by Ve1 recognition of Ave1 is not well understood. However, domain-swapping analysis of *Ve1* and *Ve2* identified the domains essential for *Ve1* functionality in tomato [62]. It has been suggested that HR is not absolutely required for *Verticillium* wilt resistance, and may occur as a consequence of escalated signaling upon Ave1 recognition in both tomato and tobacco [63]. Transgenic tomato expressing Ave1 induced various defense genes including *PR-1*, *PR-2* and peroxidases, independently of *Ve1* [64]. Homologs of tomato *Ve1* have also been reported from other plant species including tobacco, potato, wild eggplant, hop and cotton suggesting a conserved recognition mechanism [65,66]. Host-induced gene silencing (HIGS) has been successfully used in tomato, Arabidopsis and cotton plants to suppress *Verticillium* wilt disease by targeting various virulence effectors of *V. dahliae* [67,68].

Distinct resistant mechanisms associated with the *Ol*-genes against the powdery mildew species *Oidium neolycopersici* have been demonstrated using nea*r*-isogenic lines (NIL) [69]. The dominant resistance genes (*Ol-1*, *Ol-3, Ol-4, Ol-5*, and *Ol-6*) hamper the fungal growth *via* classical HR of the host epidermal cells, while the recessive gene *ol-2* confers resistance *via* papilla formation [70,71]. By performing complementation experiments using transgenic tomato lines as well as virus-induced gene silencing (VIGS) assays it was demonstrated that the *ol-2*–mediated powdery mildew resistance is due to loss of *SlMlo1* (*mildew resistance locus O*) function [72]. *Ol-1*-mediated resistance to powdery mildew in tomato requires enzymes glutathione *S*-Transferase [73] and acetolactate synthase [74]. More recently, 15 other *SlMlo* homologs were identified and characterized for their structural organization, phylogenetic analysis and expression profiles [75]. In the future, it would be interesting to investigate the possible roles of these homologs in tomato defense against other powdery mildew species including *Erysiphe orontii* and *Leveillula taurica*.

As opposed to the specific response to pathogen-encoded effectors in gene-fo*r*-gene host-pathogen interactions, the mode of action of *Asc-1*-mediated resistance to the late blight disease causing fungus *Alternaria alternaria* formae speciales *lycopersici* is based on insensitivity to sphinganine-analog mycotoxins (SAMs) [76]. Consequently, *Asc-1* has no homology to any published plant disease resistance gene but is homologous to the *Saccharomyces cerevisiae LAG1* that has been associated with life span in yeast. Thus, the mechanism of *Asc-1*-mediated resistance is by preventing apoptosis in resistant plants by the restoration of EGGAP transport [77]. Overexpression of *Asc-1* gene also confers resistance to *Alternaria* in *Nicotiana umbratical* [78].

Nematodes and Insects

In nematodes two *R*-genes have been cloned so far including *Mi* and *Hero*. Differences in resistance mechanisms or incompatible responses to nematodes are also evident in tomato. *Hero*-mediated resistance against potato cyst nematodes (PCNs) (*Globodera* spp.) is often described as a "hypersensitive-like" or "delayed hypersensitive" response that appears after syncytium (feeding structure) induction, leading to slow deterioration or abnormal development of the feeding site [79]. Although PCNs and similar cyst-forming nematodes are able to invade and develop on resistant plants, however, their reproduction is severely compromised [80]. *Hero* encodes a NLR protein and confers

resistance to all pathotypes of *G. rostochiensis* and partial resistance to *G. pallida* [81]. *Hero* gene is not only expressed in roots but also in aerial parts including, stems, leaves, and flower buds, its expression is upregulated in roots in response to PCN infection and correlates with the timing of syncytium death [80]. Interestingly, inoculation of tomato leaves with PCN also leads to HR indicating that *Hero*-mediated resistance response is not tissue-specific [82].

In contrast to *Hero*, *Mi-1.2*-mediated resistance against root-knot nematodes (RKN) (*Meloidogyne* spp.) is early and involves HR. As a result, the invading juvenile is not able to induce a feeding site and becomes surrounded and embedded among necrotized cells [83]. The *Mi-1.2* gene also confers resistance to certain potato aphid isolates (*Macrosiphum euphorbiae*), whitefly (*Bemisia tabaci*) and to some extend to psyllids (*Bactericerca cockerelli)* [84-88] *via* yet unidentified mechanism(s) that does not involve HR. Although *Mi-1* is an effective source of RKN resistance, *Mi-1*-mediated resistance is inactive above 28°C soil temperature [89]. More recently, another nematode resistant gene *Mi-9,* from the wild species *Solanum arcanum,* was genetically characterized and identified as a homolog of *Mi-1* that conferred heat-stable resistance to RNK [90]. Interestingly, silencing *Mi-1* homologs in tomato lines carrying *Ol-4* and *Ol-6* compromised the resistance to *O. neolycopersici* in those lines, suggesting that *Ol-4* and *Ol-6* are *Mi-1* homologs [91]. About 59 *Mi-1* homologs have been identified in the genome of the cultivated potato species *S. tuberosum* and *S. phureja* [92]. The evolutionary history of *Mi-1* and another *R*-gene family member *Sw5* (CNL protein that provides resistance to tomato spotted wilt virus (TSWV) [93,94]) is analyzed in closely related *Solanaceae* family members *S. tuberosum* and *S. lycopersicum* [95]. In this study, the authors reported that the potato genome carries larger *R*-gene families than tomato and this could due to sequential duplications in the potato genome or recurrent gene losses in tomato. Further, they observed that *Sw5* and *Mi-1* gene families had dissimilar evolutionary histories. Overall, this work suggests that gene clusters are more prone to duplication and translocation, which may occur through unequal crossing overs or errors in the replication or recombination processes. Interestingly, a recent study reported that *Mi-1.2* has direct negative effects on a zoophytophagous biocontrol agent *Orius insidiosus* [96]. Taken together, these findings suggests that a single dominant *R*-gene mediated resistance can impact organisms belonging to very diverse feeding guilds.

Besides conferring resistance against *C. fulvum*, the *Cf-2* also mediates resistance to the root parasitic nematode *G. rostochiensis* and this resistance requires Rcr3pim protein of *S. pimpinellifolium* [97]. A tomato root cDNA library was screened in a yeast two-hybrid assay, by using *G. rostochiensis* effector G*r*-VAP1 as bait. In this screen, it was found that G*r*-VAP1interacts with apoplastic papain-like cysteine proteases Rcr3pim. Tomato plants that lack the *Cf-2* gene but has the functional *Rcr3pim* allele have higher number of nematodes than the *Cf-0/Rcr3lyc*and *Cf-0/rcr3-3* plants suggesting that Rcr3pim is the virulence target of *G. rostochiensis*. Transient expression of G*r*-VAP1 in tomato plants harboring *Cf-2* and Rcr3pim triggers an HR response [97].

Viruses

Plant viruses cause disease and severe losses in tomato. Similar to other classes of pathogens, tomato plants have acquired a series of *R*-genes against these viruses. Tomato virus can spread by different ways such as transmission *via* contaminated seeds or insect borne transmission. Tomato mosaic virus (ToMV) is a seed borne virus that can be spread by human activities for instance agricultural workers with contaminated hands, tools, and clothing, however transmission by insects is rare. Tomato *Tm-1*-mediated resistance against ToMV involves direct or indirect binding of the *Tm-1* gene to replication proteins of ToMV, thus, inhibiting RNA replication even before formation of the active replication complexes on the membranes, however there is no HR [98]. The Tm-1 protein is predicted to have the TIM barrel structure but there are no clues about their cellular functions. Interestingly, the product of the *Tm-1* (allelic to *Tm-1*) gene found in the ToMV susceptible tomatoes can neither bind to ToMV replication proteins nor inhibit ToMV multiplication but have been shown to bind to the replication proteins of non-host viruses tobacco mild green mosaic virus (TMGMV) and pepper mild mottle virus (PMMoV) and inhibit their RNA replication *in vitro* resulting in non-host resistance [98-100]. Another tomato *R*-gene, *Tm-22*, confers resistance to ToMV by the recognition of the carboxy terminus of the ToMV movement protein and interfering with viral cell-to-cell movement in plants [101]. Tm-22 belongs to the CNL class of resistance proteins [102]. Transgenic tobacco plants expressing *Tm-22* gene become resistant against infection with ToMV [103]. Similarly, transgenic potato plants over expressing *Tm-22* gene confers resistance to multiple viruses like tobacco mosaic virus, ToMV, potato virus X (PVX) and PVY [104].

An example of virus transmitted by insects mainly thrips is TSWV. Tomato *Sw5* gene confers resistance against TSWV [93,94]. The Avr determinant of tomato Sw-5 protein is the NSm movement protein of TSWV [105]. Transient expression of the NSm protein in tomato and generation of transgenic *N. benthamiana* harboring the *Sw5-b* gene triggers an HR [106]. Eight TSWV *R*-genes (*Sw1a, Sw1b, Sw2, Sw3, Sw4, Sw-5, Sw-6* and *Sw-7*) have been reported to date [107].

TYLCV belongs to the class of DNA viruses that are transmitted *via* whiteflies and affects tomato production worldwide. There are total six TYLCV resistance genes *Ty-1* to *Ty-6*. *Ty-1* and *Ty-3,* both derived from *Solanum chilense* and are allelic. The *Ty-1/Ty-3*-mediated defense against TYLCV is somehow different from tomato defense against other viruses as TYLCV shows low levels of viral replication and systemic spread but with moderate (as with *Ty-3*) or no (as with *Ty-1*) visual symptoms [108]. *Ty-1* and *Ty-3* are allelic and represents a unique category of *R*-genes that encode for RNA-dependent RNA polymerases (RdRp) unlike most of the *R*-genes discussed so far that belongs to NLR family. *Ty-1* and *Ty-3* are proposed to confer resistance to TYLCV by amplifying the RNAi signal [108]. The catalytic domain of the *Ty-1/Ty-3* allele is characterized by a five-amino acid motif, DFDGD [108]. As compared to susceptible tomato plants, *Ty-1/Ty-3* plants have higher levels of siRNA amplifications and *Ty-1* plants also show higher levels of TYLCV DNA methylation [109]. Interestingly, *Ty-1*-mediated resistance is also effective against the bipartite tomato severe rugose begomo virus, suggesting enhanced transcriptional gene silencing, however, a mixed infection of TYLCV with a RNA virus such as cucumber mosaic virus (CMV) compromised the resistance leading to a decrease in *Ty-1*–mounted anti-geminiviral RNAi response [109]. Under natural field conditions with the occurrences of mixed viral infection *Ty-1*-mediated resistance might not be very effective. Unlike *Rx*-mediated resistance that results in extreme resistance (ER) against potato virus X [110], TYLCV mediated resistance results in virus tolerance rather than immunity. Functional *Ty-1/Ty-3*-like alleles are also present in several other *S. chilense* wild type tomato accessions, shown by fine mapping and VIGS [111]. Additionally, the DFDGD catalytic domain of the *Ty-1*and *Ty-3* genes is conserved among *Solanum* species [111]. In a recent study, *Ty-2* and Ty-3 genes were used to develop a series of *R*-gene pyramided tomato lines and the linked markers were evaluated for their diagnostic value and utility in pyramiding *Ty* genes [112].

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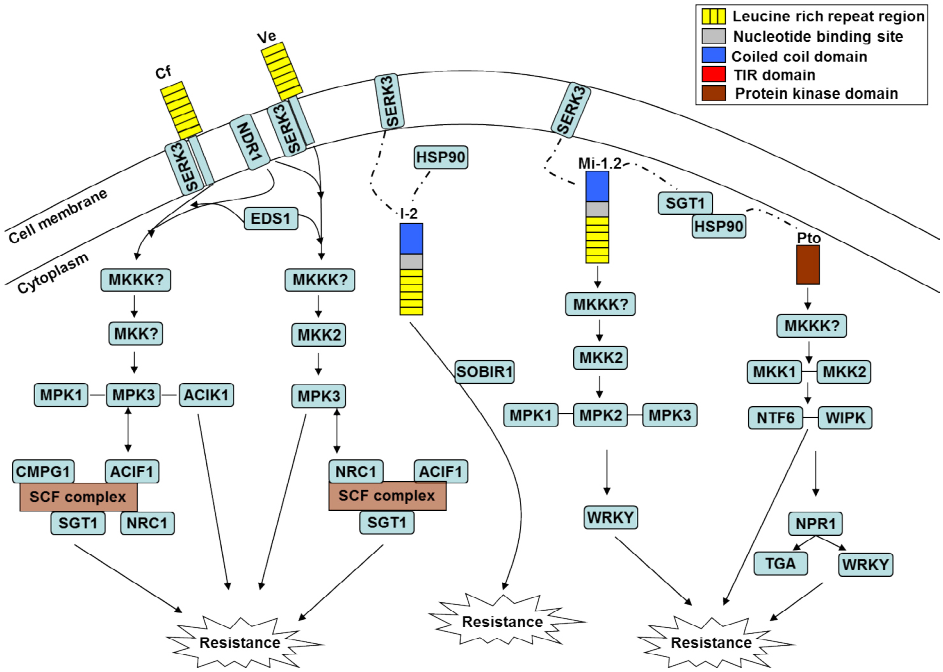

Figure 1: Downstream signaling components of tomato *R*-genes.

Bacteria

Pseudomonas syringae pv. *tomato* (*Pst*) causes bacterial speck of tomato and the major sources of *Pst* infection can be seed and infected crop debris. In tomato a serine-threonine protein kinase *Pto* gene confers resistance to *Pst* strains carry the avirulence gene *AvrPto* [113]. *Prf* that encodes an NLR resides in the middle of the *Pto* gene cluster [20]. In tomato, *Prf*-mediated resistance against *Pst* involves recognition of secreted effectors (AvrPto or AvrPtoB) by two highly homologous tomato protein kinases Pto and Fen [114-117]. Changes in these kinases upon binding to the effectors are detected by *Prf,* resulting in HR at the sites of attempted infection.

Signaling Components Acting Downstream of Tomato Resistance Genes

Early understanding of host-pathogen interaction came from studies conducted in Arabidopsis. Identification and characterization of host components underlying ETI revealed both common and specific signaling components in *R*-gene mediated resistances against different biotic stresses [118-120]. Some of the important components of *R*-gene mediated downstream signaling from Arabidopsis include Non-Race-Specific Disease Resistance1 (NDR1), Enhanced Disease Susceptibility1 (EDS1), Phytoalexin Deficient 4 (PAD4), Nonexpresser of PR genes 1 (NPR1), Suppressor of the G2 allele of SKP1 (SGT1), Required for Mla 12 Resistance (RAR1), RAR2, AvrPphB susceptible 3 (PBS3), Heat Shock Protein (HSP90) [42,121]. Additional signaling components include, the mitogen-activated protein (MAP) kinases, one of the largest group of plant kinases that function in the regulation of complex plant defense reactions by altering the activity of the different signal transduction pathways through phosphorylation/dephosphorylation of proteins [122]. Defense associated phytohormones including jasmonic acid (JA), ethylene (ET) and salicylic acid (SA) regulate plant responses to a wide range of pests and pathogens. There are excellent reviews focusing on the complex network of defense signaling pathways that involve these three phytohormones [123].

Tomato became another ideal model for studying host-pathogen interaction as it is natural host of many pests and pathogens as well as possesses a repertoire of *R*-genes. The application of VIGS, transient reverse genetics approach, has been successfully used to study the function of certain tomato genes [124]. To analyze the function of some of tomato *R*-genes that produce HR and to identify their downstream signaling components and mechanisms many groups have performed experiments in tomato. However, given the moderate efficiency of VIGS in tomato, large-scale random screens have been conducted in the heterologous system *N. benthamiana*, where VIGS is more effective. Many of the functional studies in *N. benthamiana* have been performed by using an auto active tomato *R*-gene and by transient expression of corresponding Avr, to consistently and uniformly activate the host system and thus avoiding variations caused by the infecting organisms.

Tomato and *C. fulvum* interaction is a model to study the receptor-mediated resistance [125]. Using VIGS in tomato and/or *N. bethamiana* or *N. tobaccum* the different components of this interaction have been identified (Figure 1), including Cf-9-interacting thioredoxin (CITRX) [126], Avr9/Cf-9 induced kinase 1 (ACIK1) [127], the NLR protein required for HR-associated cell death 1 (NRC1) [128], the U-box protein CMPG1 [129], the LeMPK1, LeMPK2, and LeMPK3 [130], Avr9/Cf-9–Induced F-Box1 (ACIF) [131], members of the phospholipase C family [49], Suppressor of BAK1-Interacting RLK1 1 (SOBIR1) [132], Somatic Embryogenesis Receptor Kinase1 (SERK3)/BAK1 [133], endoplasmic reticulum residing chaperones including HSP70 binding proteins (BiPs) and a lectin-type calreticulin (CRT) [134].

Likewise, using VIGS in tomato the signaling cascade downstream of *Ve1* is shown to require several components including EDS1, NDR1, NRC1, ACIF, MEK2 and SERK3/BAK1 (Figure 1) [135]. To identify additional components involved in *Ve1*-mediated signaling, a GFP-tagged version of Ve1 protein was overexpressed in *N. benthamiana* leaves, followed by mass spectrometry. This resulted in the identification of BiPs and CRT as Ve1 interacting proteins. VIGS mediated knockdown of BiP*s and* CRT*s* in tomato resulted in compromised

Ve1-mediated resistance to *V. dahliae* in most cases, showing that these chaperones play an important role in Ve1 functionality [136]. Furthermore, by using VIGS it has been demonstrated that SOBIR1 and SERK3/BAK1 are also required for I/Avr1-dependent necrosis in *N. benthamiana* [137]. In a genetic based screening F2 tomato seedlings, those homozygous for the eds1 mutation (eds1/eds1) and those that were heterozygous (EDS1/eds1), were chosen for a disease assay and were inoculated with Fol race 3. Samples were screened for the disease resistance and it was found that EDS1 is required for *I-7* mediated resistance [53].

By applying VIGS in tomato plants it has been shown that *Mi-1.2*-mediated resistance against nematodes and aphids requires *Hsp90*, *Sgt1*, members of the MAP kinase cascade and WRKY transcription factors (Figure 1) [138-141]. In addition, by utilizing transgenic tomato plants expressing NahG (encodes for an enzyme that metabolizes SA) a role for SA in *Mi-1*–mediated resistance to potato aphids was identified [141]. In a VIGS screen performed in *N. benthamiana* to identify the components of *Mi*-signaling that can suppress HR triggered by a constitutively active form of Mi-1, Mi-DS4, SERK1 was identified as an important player [142,143].

To identify the tomato proteins that interact with ToMV movement protein or Tm-22-LRR yeast two-hybrid screens were performed and tomato cDNA library was screened, by using ToMV movement protein and Tm-22-LRR as respective baits [144]. In these screens Rubisco small subunit (RbCS) was identified as interacting with ToMV movement protein and SGT1 as interacting with Tm-22, in addition MP-Interacting Protein 1 (MIP1), a group of type I J-domain proteins was found to interact with both ToMV movement protein and Tm-22. By using VIGS and other *in vitro* and *in vivo* functional analysis in *N. benthamiana,* it was shown that MIP1s are required for both virus infection and plant immunity [144]. Furthermore, transgenic *N. benthamiana* plants expressing *Tm-22*, provides extreme resistance to ToMV, and VIGS mediated silencing of *NbRbCS* compromised *Tm-22*-dependent resistance, suggesting that RbCS of *N. benthamiana* plays an important role in ToMV movement and plant antiviral defenses [145]. To identify the genes involved in TYLCV resistance a reverse genetic approach was used where the susceptible and resistance tomato inbred lines from the same breeding program were inoculated with TYLCV [146]. cDNA libraries from inoculated and non-inoculated plants were compared and a trans membranal transporter protein Permease I-like was found to be preferentially expressed in resistant plants and VIGS mediated silencing of Permease gene in tomato led to decrease in resistance [146]. Furthermore, VIGS mediated silencing of hexose transporter *LeHT1*, resulted in plant growth inhibition and enhanced virus accumulation and spread and also resulted in a necrotic response along the stem and petioles of infected *LeHT1*-silenced R plants [147].

Pto-mediated resistance involves several components including kinases MEK1 andMEK2, wound-induced protein kinase (WIPK), NTF6, two transcription factors TGA1a and TGA2.2 and NPR1 (Figure 1) [118]. Furthermore, using stable RNAi/CaMV transient overexpression/VIGS about 25 genes were identified to play a role in *Pto*-mediated ETI as reviewed by [148].

Current and Future Perspective

Plants are continuously being challenged by new pathogen and pest races/strains, some of which being able to overcome the plant *R*-gene mediated defenses. One of the main goals of agricultural research is to develop technologies to overcome resistance breaking to prevent disease. In the past, few decades use of molecular markers has facilitated identification, mapping, characterization and transfer of many important traits in tomato including the traits for disease resistance [149,150]. With the recent advances in molecular biology and genetic approaches, several *R*-genes have been cloned (as discussed above). A broad-spectrum application for crop improvement and managing resistance that has gained great attention is non-host resistance [151]. Other alternatives include functional stacking of *R*-genes that has been successfully used in potato and tomato [152-154] and targeting the susceptible genes can result in a more broad-spectrum and durable type of resistance [155]. Furthermore, there has been increase resistance against some pathogens in tomato by transferring the *R*-genes from other plant species like pepper and potato [156,157].

Apart from the breeding technologies, a deeper understanding of plant innate immune perception and signaling is equally important. Here comes the role of model plants *A. thaliana* and easily amenable plant species such as *Nicotiana* species [158,159]. RNAi based approaches including siRNAs, miRNAs and *Agrobacterium*-mediated transient expression of dsRNA have been used against viruses, insects, and fungal pathogens [160]. Spray-induced gene silencing strategy utilizing dsRNAs and small RNAs targeting pathogen genes has also been successful against *Botrytis cinerea* [161]. More recently genome-editing technologies such as TALENs and CRISPR/Cas9 have been used in plant crop improvement, plant-breeding and enhanced pathogen resistance [162-164]. CRISPR/Cas9 has been successfully used to target TYLCV genome. Guide RNAs specific for coding and non-coding sequences of TYLCV were delivered *via* tobacco rattle virus into *N. benthamiana* plants stably overexpressing the Cas9 endonuclease. Subsequent challenge of these plants with TYLCV lead to a significant reduction in TYLCV accumulation and disease symptoms [165]. Recently, CRISP*R*-Cas9 system has been also used to inactivate tomato *SlDMR6*-1 (downy mildew resistance 6) resulting in disease resistance against different pathogens, including *P. syringae*, *P. capsici* and *Xanthomonas* spp. with no significant effect on plant growth and development [166]. Overall suggesting that these new technologies can be utilized for multiplex targeting of the pathogen virulence genes as well as plant susceptibility genes. Thus, there is a potential to enhance plant resistance by targeting newly evolved effectors and generating a platform for dissecting natural resistance and immune functions. At the same time, it will provide biotechnologists with a powerful tool for producing crop plants resistant to multiple viral infections.

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