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Transcriptional events defining plant immune responses

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Rapid and massive transcriptional reprogramming upon pathogen recognition is the decisive step in plant–phytopathogen interactions. Plant transcription factors (TFs) are key players in this process but they require a suite of other context-specific co-regulators to establish sensory transcription regulatory networks to bring about host immunity. Molecular, genetic and biochemical studies, particularly in the model plants Arabidopsis and rice, are continuously uncovering new components of the transcriptional machinery that can selectively impact host resistance toward a diverse range of pathogens. Moreover, detailed studies on key immune regulators, such as WRKY TFs and NPR1, are beginning to reveal the underlying mechanisms by which defense hormones influence the function of these factors. Here we provide a short update on such recent developments.

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Introduction

Plant innate immunity is a major research area both for scientists, trying to understand its molecular principals, and those with a keen interest in improving crop resistance toward phytopathogens. The plant innate immune response depends on two main recognition systems to detect invaders. One system recognizes non-self molecules termed MAMPs (microbe-associated molecular patterns) via dedicated plasma membrane localized receptors to trigger a complex signaling cascade leading to a basal defense response termed MAMP-triggered immunity (MTI) [1]. The second system involves intracellular host receptors encoded by major resistance (*R*) genes to detect pathogen-derived effector molecules within the host cell. Resistance established by this means is called effector-triggered immunity (ETI). ETI leads to a more robust and stronger immune response yet it shares many components with MTI [2]. A major consequence of both MTI and ETI signaling is a rapid and massive transcriptional reprogramming with a substantial overlap between the genes showing altered expression upon MTI or ETI [3].

Over the past two years excellent reviews have appeared dealing with different aspects related to transcriptional regulation of host immunity [4–9]. Thus, in this short review we merely present an update on recent discoveries related to this research field. We do not address the important role of small RNAs and chromatin modifiers in regulating plant defenses since these have recently been covered elsewhere [10,11].

Transcription factors involved in immunity

Because of the distinct strategies employed by the diverse phytopathogens attempting to gain access to their hosts, the plant surveillance system is highly sophisticated and comprises a complex interconnected signal transduction network ultimately ensuring a properly timed transcriptional output response. In general, TFs exert their functions by binding to defined DNA motifs within the regulatory regions of target genes thereby positively or negatively affecting expression. Certain large plant TF families including AP2/ERF, bHLH, NAC, TGA/bZIP and WRKY appear to be prominent regulators of host defense [9]. Numerous individual TFs have been identified that play critical roles in modulating and fine-tuning the host transcriptional immune response [6,7,9,12,13] (Table 1), but in most cases it remains unclear how their target genes contribute to establishing immunity. Below we summarize recent key findings involving specific TF family members.

TEOSINTE BRANCHED1/CYCLOIDEA/PCF (TCP) TFs play vital roles in development and in modulating hormone activities [14]. A large-scale protein–protein interaction study identified TCP13, TCP14, TCP15, TCP19 and TCP21 as central regulatory nodes of various signaling pathways targeted by pathogen-derived effectors [15]. TCP8, TCP14 and TCP15 functions positively effect ETI mediated by several *R* genes [16]. Two TCP factors, TCP4 and TCP20, acted antagonistically to regulate *LIPOXYGENASE2* (*LOX2*) expression encoding a key biosynthetic enzyme of the defense signaling hormone jasmonic acid (JA) [17]. Moreover, TCP8 promoter binding positively regulated the expression of *ISOCHORIMATE SYNTHASE1* (*ICS1*) required for salicylic acid (SA) biosynthesis, another key defense signaling

Table 1

Recent reports on plant transcription factors modulating plant innate immunity

Genes and species	Targets	Protein interactions	Features	Ref.
AtTCP8, 9	*ICS1*	WRKY28, NAC019, SARD1, SRFR1	*tcp8 tcp9* double mutant attenuated resistance to *Psm* ES4326	[16,18]
AtTCP13, 14, 19		Effectors from *G. orontii*, *H. arabidopsidis* (*Hpa*), *P. syringae* (*Pst*)	Effector-TCP13, 14, 19 interactions led to susceptibility phenotypes with *Hpa* and *G. orontii*, but to resistance phenotypes with *Pst*	[15]
AtERF014			Positively influences resistance to *Pst*, negatively to *B. cinerea*	[21•]
AtERF15			Resistance to *Pst* DC3000 and *B. cinerea*	[20]
AtERF96	*PDF1.2a, PR3, 4, ORA59*		Positively influences resistance to *B. cinerea* and *P. carotovorum*. Positive regulator of ABA response	[19,73]
AtCAMTA3	*EDS1*	EDS1, ICS1, SARD1, CBP60g, DREB1A, NTL9, ZAT12, CBF1, 2, SRS, CIPK14, BON1, CM2, ICE1, XLG2, NDR1, EIN3	CAMTA3 mutants show constitutive resistance to *Pst* DC3000, enhanced resistance to *B. cinerea*, *G. cichoracearum*, but elevated susceptibility to the herbivore *Trichopulsiani*	[23–27]
AtNAC032	*MYC2, NIMIN1, PDF1.2A*		Positively influences resistance to *Pst* DC3000	[28•]
AtWRKY22			Positively influences susceptibility to green peach aphid *M. persicae*; modulates SA-JA interplay	[38]
AtWRKY57	*JAZ1, 5*	SIB1, 2	Negatively affects resistance to *B. cinerea*. Competes with AtWRKY33 for common targets.	[35•]
AtWRKY11, 70			*wrky11 wrky70* double mutant lost *Bacillus cereus* AR156-triggered ISR to *Pst* DC3000.	[40]
AtWRKY33	*ACS2,6, 318 target genes*	MPK3	Glutathione-induced ACS expression via WRKY33. ChIP-seq identified 1576 *in vivo* genomic targets of WRKY33	[33,34••]
AtWRKY46		MPK3	Activator of MTI. *WRKY46* phoshorylation by MPK3	[74]
OsMYC2	*OsJAZ10, OsMADS1*	OsJAZ proteins	Positive regulator of early JA signaling. Overexpression of *OsMYC2* leads to increased resistance to *Xoo*	[31]
OsWRKY45			Important but contrasting roles in resistance to pathogens and herbivores. TE-siR815 and two alleles of *OsWRKY45* play distinct roles in resistance to *Xoo* and *Magnaportha oryzae*	[42,43•]
OsWRKY51	*OsPR10a*		Positively impacts resistance to *Xoo*	[75]
OsWRKY53		OsMPK3, 6	Negatively influences resistance to striped stem borer larva. Negatively modulates MPK activity	[76]
OsWRKY62, 76		OsWRKY62, 76	*WRKY62* and *WRKY76* resistance to *Xoo* and *M. oryzae* depends on alternative splicing	[43•]
CabZIP63	*CabZIP63, CabWRKY40*		VIGS silencing of *CabZIP63* attenuated resistance to *Ralstonia solanacearum*	[77]
AtNTL9, CHE	*ICS1*		NTL9-mediated SA synthesis essential for stomatal immunity. CHE regulator of daily and SAR SA levels	[78]

At, *Arabidopsis thaliana*; *Os*, *Oryza sativa*; *Ca*, *Capsicum annuum*.

hormone. Genetic studies revealed that TCP9 is also required for *ICS1* expression and for resistance toward the bacterium *Pseudomonas syringae* pv. *maculicola* ES4326 [18].

Members of the large APETALA2/ETHYLENE RESPONSE FACTOR (AP2/ERF) TF family, notably ERF1, ORA59, and ERF6, play essential roles in regulating the defense transcriptome [6]. Arabidopsis *ERF96*, when overexpressed, positively modulated resistance toward the necrotrophic fungus *Botrytis cinerea* and the bacterium *Pectobacterium carotovorum* [19]. Using appropriate mutants *B. cinerea*-induced expression of *ERF96* was shown to be dependent on JA and ethylene (ET). Chromatin Immunoprecipitation coupled with quantitative polymerase chain reaction (ChIP-qPCR) performed in an *ERF96*-overexpressor line identified *PR-3*, *PR-4* and *PDF1.2* as targets of ERF96.

Arabidopsis *ERF15*-overexpressor lines showed enhanced resistance against *B. cinerea* and the virulent bacterium *Pseudomonas syringae* pv. *tomato* (*Pst*) DC3000,