Transcriptional regulation of effector-triggered immunity (ETI) in plants: from tissue to cells

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Chapter 1

General Introduction

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Overview of plant immunity

Plants are constantly exposed to a variety of pathogens which pose a serious threat to crop productivity. Plant pathogens have caused major crop disease outbreaks in the past with Great Irish Famine of 19th century being one of the most devastating examples which lead to emigration/death of around 2.5 million people[1]. On top of that, many food and cash crops, such as wheat, rice, maize and others are still under threat of many different types of diseases[2]. According to the Food and Agriculture Organization of the United Nations (FAO) plant diseases lead to approximately 20-40% of global crop loss annually, costing about US$220 billion[3]. In response, global agricultural pesticide use dominated by herbicides has increased steadily over recent decades, nearly doubling since 1990, with associated economic costs rising to almost USD 50 billion annually, reflecting a growing reliance on chemical disease management strategies which pose severe health risks on consumers[4]. These limitations underscore the urgent need for sustainable, disease management strategies that rely on the plant's own innate defense mechanisms. To defend themselves, plants have the inherent capability of responding to the pathogenic cues by eliciting a multitude of defense responses that are mediated through complex signaling networks[5]. Physical barriers on plant surface such as epidermal hairs, wax layers and the cell wall being the first line of defense to prevent pathogen invasion[6]. In addition, anti-microbial molecules in the apoplast acts as chemical barrier to limit pathogen progression[7]. However, some pathogens have evolved to by-bass the physical and chemical barriers to colonize the plants. Plant defense responses can then be initiated by recognition of general pathogen-associated molecular patterns (PAMPs) by cell-surface localized pattern-recognition receptors (PRRs) leading to pattern-triggered immunity (PTI) and that of specific pathogen effector molecules secreted into plant cells recognized by intracellular nucleotide-binding leucine-rich repeat receptors (NLRs) leading to effector-triggered immunity (ETI)[5]. Conceptually, PTI and ETI in plants share some classical features of innate immune mechanisms in animals, where conserved microbial features and intracellular danger signals are sensed by pattern-recognition receptors and NOD-like receptors, respectively[8,9]. However, unlike animals, plants lack specialized immune cells and adaptive immunity; instead, each cell autonomously perceives pathogens and mounts defense responses. Historically, plant immunity was described using a dichotomous framework of PTI, also termed as PAMP-triggered defense, and ETI, previously referred to as gene-for-gene resistance[10,11]. Emerging evidence has challenged this strict separation, leading to the view that PTI and ETI operate as overlapping, mutually reinforcing modules within a continuum of integrated immune signaling rather than as discrete defense layers[12,13]. These layers of immunity are fine-tuned by spatial, temporal, and molecular contexts. This complexity is further underscored by the roles of transcriptional regulators, calcium signaling, and chromatin dynamics in shaping immune outputs[14–16]. Additionally, recent advances in single-cell and spatial transcriptomics[17] are revealing unprecedented insights into how immunity is executed at the resolution of individual cell types challenging the notion of uniform immune responses across plant tissues.

1. Cell surface immunity

1.1 PRR immune receptor

Plant defense responses can be initiated by recognition of general PAMPs by cell-surface localized PRRs leading to PTI and that of specific pathogen effector molecules secreted into plant cells recognized by intracellular NLRs, activating ETI[10] (**Figure 1**). PRRs can be either receptor-like proteins (RLPs) or receptor-like kinases (RLKs). Both RLPs and RLKs possess an extracellular ectodomain (ECD) and a transmembrane domain, however RLPs lack a C-terminal intracellular kinase domain[5,18]. Based on their ECD they are classified into different subfamilies: leucine-rich repeat (LRR) domain, lysinemotifs (LysM), lectin domain, or epidermal growth factor (EGF)-like domain[19–21]. PRRs perceive a wide range of PAMPs such as peptides, lipids, peptidoglycans (PGs), and polysaccharides from prokaryotes (bacteria) and chitin and oligo-galacturonides (OGs) from eukaryotes (fungi and oomycetes)[18].

1.2 PRR signaling

Many PRRs acts together with co-receptors to transduce the downstream signaling. Upon ligand perception and binding LRR, RLKs recruit BRI1-ASSOCIATED RECEPTOR 2 KINASE (BAK1), a member of SOMATIC EMBRYOGENESIS RECEPTOR KINASES (SERKs) family[20]. For instance, LRR-RLK FLAGELLIN SENSING2 (FLS2), EF-TU RECEPTOR (EFR), and PEP RECEPTOR 1 (PEPRs) require the coreceptors BAK1 and BAK1-LIKE 1 (BKK1) both of which are members of the SOMATIC EMBRYOGENESIS RECEPTOR KINASE (SERK) family[20,22–24]. On the other hand RLP23 requires BAK1 and SUPPRESSOR OF BIR1 (SOBIR1) as co-receptor for promoting downstream signaling[25]. Additionally RLP23 is suggested being one of the PRR which might require lipase-like protein family to activate some downstream immune responses[26], although this mechanism needs further investigation.

Some RLKs, such as members of the BAK1-INTERACTING RECEPTOR-LIKE KINASE (BIR) family, negatively regulate immune signaling by sequestering SOBIR1 and BAK1, thereby preventing autoactivation[27,28]. Additionally, RLKs like FERONIA have been reported to suppress the interaction between FLS2 and BAK1 by interacting with RAPID ALKALINIZATION FACTORS (RALF23) thereby modulating immune signaling[29]. These findings suggest that PRRs possess both activating and inhibitory roles in modulating downstream responses. Upon ligand binding, heteromeric receptor complexes form between PRRs and their co-receptors, bringing their cytoplasmic kinase domains into close proximity. This proximity facilitates trans-phosphorylation and subsequent activation of receptor-like cytoplasmic kinases (RLCKs) such as BIK1, initiating downstream signaling cascades[30,31]. Upon PRR activation, a rapid series of cellular events is triggered, including the production of reactive oxygen species (ROS), MAPK cascade activation, cytosolic calcium influx, callose deposition, and extensive transcriptional reprogramming[323334]. Collectively, these physiological and cellular changes constitute pattern-triggered immunity (PTI).

2. Intracellular immunity

2.1 NLRs immune receptors

Adapted pathogens usually deliver a suite of effectors into the plants, which promotes pathogen virulence and results in effector-triggered susceptibility (ETS) in host plants[10]. Plant pathogenic bacteria deliver effectors into host cells using type III secretion systems (TTSS)[35]. Pathogen effectors are recognised by specific disease resistance (R) genes which encode NLR proteins[10,36]. *Arabidopsis thaliana* (Arabidopsis) have around 150 NLR coding genes which are broadly classified into 2 categories[37]. TIR-NB-LRR (TNL) group with an N-terminal Toll and interleukin-1 (TIR)-like domain such as RESISTANT TO PSEUDOMONAS SYRINGAE 4 (RPS4)/RESISTANCE TO RALSTONIA SOLANACEARUM 1 (RRS1) which recognize bacteria carrying AvrRps4[38,39], and CC-NB-LRR (CNL) group with an N-terminal coiled-coil domain such as RESISTANCE TO PSEUDOMONAS SYRINGAE PROTEIN 2 (RPS2) which recognize bacteria carrying AvrRps2[40]. Besides their division into TNLs and CNLs, NLRs can also be distinguished by their functional contribution to immune responses. Certain NLRs act as effector sensors, while helper NLRs (hNLRs) act downstream to relay signaling from these sensors and promote defense activation. In Arabidopsis most of TNLs and only some CNLs require hNLRs ACTIVATED DISEASE RESISTANCE 1 (ADR1s) and N REQUIREMENT GENE 1 (NRG1s) for mediating downstream immune signaling[41–44]. Downstream signaling also differs between NLR classes, TNLs (e.g., RPS4/RRS1) transmit defense signals through the ENHANCED DISEASE SUSCEPTIBILITY 1 (EDS1) pathway, which involves complex formation with lipase-like proteins PHYTOALEXIN DEFICIENT 4 (PAD4) and SENESCENCE ASSOCIATED GENE 101 (SAG101)[45,46]. On the other hand, classical CNL receptors such as RESISTANCE TO PSEUDOMONAS SYRINGAE PV MACULICOLA (RPM1) and RPS2 require NON-RACE-SPECIFIC DISEASE RESISTANCE 1 (NDR1) for full resistance output[45].

NLRs can detect effectors through direct binding like the TNL RECOGNITION TO PERONOSPORA PARASITICA (RPP1) from Arabidopsis recognizes the *Hyaloperonospora arabidopsidis* (Hpa) effector ARABIDOPSIS THALIANA RECOGNIZED1 (ATR1)[47]. On the other hand NLRs can detect the effectors by guarding host components called guard hypothesis[19]. For instance, Arabidopsis NLRs RPS2 and RPM1 monitor the status of the RPM1-INTERACTING PROTEIN 4 (RIN4) protein[48], which is targeted pathogen effectors. Additionally, the decoy model proposed later by that hosts can evolve alleles or gene copies (paralogs) that don't directly serve essential defense roles[49]. Instead, they act as deceptive copies, resembling real pathogen targets which can also be monitored by NLRs to trigger immune response.

2.2 Key signaling module in ETI: the EDS1 signaling

Upon activation TNLs come in close proximity upon oligomerization which in turn activates the NADase and 2′,3′-cAMP/cGMP synthase activity[9,50–52] which leads to the synthesis of some small molecules like v-cADPR/ADPR/NAM and 2',3'-cNMP that is proposed to activate the downstream signaling[50,53,54]. Upon TNL activation, the lipase-like proteins SAG101 and EDS1 interact with NRG1[55], while PAD4 and EDS1 associate with ADR1[43] (**Figure 1**). These interactions lead to the formation of heteromeric complexes, which then activate immune responses, including defense-related gene expression and the hypersensitive response (HR). Interestingly, it has been shown that these EDS1-SAG101-NRG1 and EDS1-PAD4-ADR1 have some unequal redundant roles during PTI+ETI activation[56]. More recently, it has been shown that activation and association of helper NLRs and lipase-like protein to form resistosomes, induce calcium influx to promote downstream defense responses[57,58] (**Figure 1**). In addition to these modules, a distinct class of helper NLRs termed NRCs (NLRs required for cell death), predominantly found in Solanaceae species, act as central signaling hubs downstream of multiple sensor NLRs. Upon activation, NRCs oligomerize to form resistosome-like calcium channels that are sufficient to trigger cell death and immune signaling[59,60], highlighting both conserved and lineage-specific strategies for ETI signal execution. Although much progress has been made in dissecting the signaling pathway. However, the contribution of crucial immune regulators in governing the ETI specific responses remains inadequately understood.

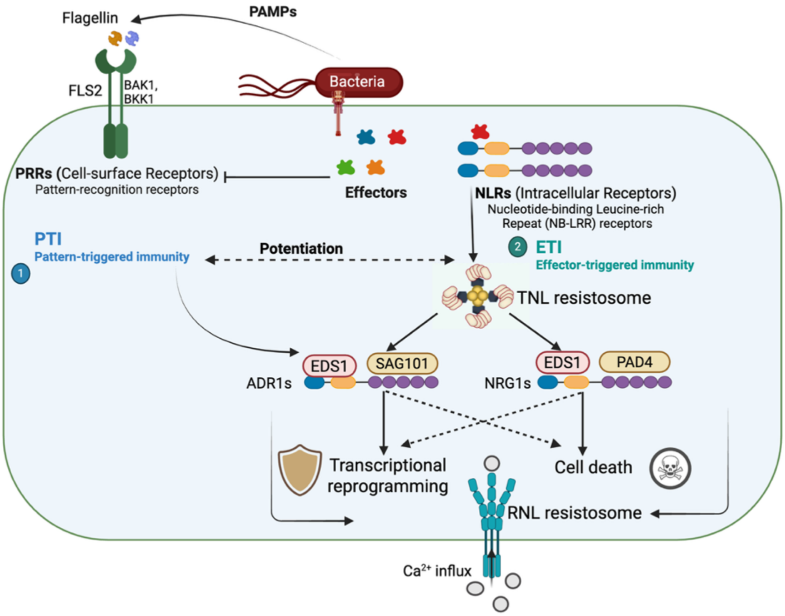

Figure 1. Layered plant immune signaling pathways coordinated by cell-surface and intracellular receptors. The schematic illustration of layered immune system in plants, highlighting how cell-surface and intracellular receptors coordinate immune responses upon pathogen recognition. Pattern-recognition receptors (PRRs), such as FLAGELLIN-SENSING 2 (FLS2), detect conserved pathogen-associated molecular patterns (PAMPs) such as bacterial flagellin and initiate pattern-triggered immunity (PTI). Upon PAMP binding, PRRs form complexes with co-receptors like BRI1-ASSOCIATED RECEPTOR 2 KINASE (BAK1) and BAK1-LIKE 1 (BKK1), leading to activation of early defense responses. To overcome PTI, pathogens deliver effectors into host cells. These effectors are recognized by intracellular nucleotide-binding leucine-rich repeat receptors (NLRs), which activate effector-triggered immunity (ETI). TIR-NLR activation promotes resistosomes that trigger the ENHANCED DISEASE SUSCEPTIBILITY 1 (EDS1) signaling pathway. EDS1 associates with PHYTOALEXIN DEFICIENT 4 (PAD4) and ACTIVATED DISEASE RESISTANCE 1 (ADR1), or with SENESCENCE ASSOCIATED GENE 101 (SAG101) and N REQUIREMENT GENE 1 (NRG1), to form two distinct signaling modules: EDS1-PAD4-ADR1 and EDS1–SAG101–NRG1. Both modules contribute to immune

responses via the formation of helper NLRs (hNLRs) resistosomes, leading to calcium (Ca^{2+}) influx, transcriptional reprogramming, and hypersensitive cell death. The diagram emphasizes that both ADR1 and NRG1 contribute to hNLRs resistosome assembly, with the ADR1 module playing a greater role in defense gene activation, and the NRG1 module being more involved in promoting cell death.

3. Transcriptional regulation of plant innate immunity

3.1 Role of transcription factors

The timing and efficiency of immune transcriptome activation are critical determinants of a plant's ability to mount an effective defense against invading pathogens. Among the various cellular components involved in transcriptional reprogramming, transcription factors (TFs) play a central and well-characterized role. Extensive genetic and functional studies have demonstrated that mutations in key TF families including WRKYs, TGAs, NACs, CALMODULIN-BINDING PROTEIN 60 (CBP60s), ETHYLENE RESPONSE FACTORS (ERFs), BASIC LEUCINE ZIPPER (bZIPs), BASIC HELIX-LOOP-HELIX (bHLHs), MYBs, CALMODULIN-BINDING TRANSCRIPTION ACTIVATOR (CAMTAs), and TCPs can profoundly alter disease resistance phenotypes across a wide range of plant-pathogen interactions[61–67]. These TFs are responsible for activating and fine-tuning the expression of numerous immune-related genes, often integrating upstream signals such as salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) to coordinate context-specific responses.

The central role of TFs in immunity makes them frequent targets of pathogen effectors, which have evolved to manipulate host transcription in order to suppress defense and promote infection[68–70]. Numerous bacterial, fungal, and oomycete pathogens deliver effector proteins directly into host cells to interfere with transcriptional regulators including TFs, transcriptional co-activators, repressors, and components of the

Mediator complex **(Table 1).** Many of these effectors specifically manipulate hormone signaling pathways, especially those involving SA[71,72], JA[73], or the crosstalk between the two[74–77]. By hijacking these signaling hubs, pathogens enhance host susceptibility in a manner tailored to their lifestyle for example, by repressing SA responses to benefit biotrophs or suppressing JA signaling to assist necrotrophs. Overall, this growing body of evidence underscores the vulnerability and importance of transcriptional regulation as both a defensive strength and a strategic weak point in plant pathogen interactions.

Table 1. Pathogen effectors and their host targets that are involved in transcriptional regulation during plant immunity. This table summarizes some well-studied effectors secreted by different pathogens that hijack diverse transcriptional regulators of the host plant, including transcription factors, and transcriptional (co-) activators and repressors, to facilitate infection.

Pathogens	**Effector**	**Function of host target**	**Host target**	**Host species**	**Ref.**
Ralstonia solanacearum	RipAB	Transcription factor (TF)	TGAs	*Arabidopsis thaliana*	[78]
Xanthomonas campestris pv *v esicatoria*	XopD	Transcription factor (TF)	MYB30	*Arabidopsis thaliana*	[79]
Xanthomonas campestris pv *v esicatoria*	XopS	Transcription factor (TF)	WRKY40	*Capsicum annuum*	[77]
Phytoplasma	Phyllogen	Transcription factor (TF)	MADS-box	*Arabidopsis thaliana*, *Oryza sativa*	[80]

Hyaloperonospora arabidopsidis (*Hpa*)	HaRxL44	Mediator complex	MED19a	*Arabidopsis thaliana*	[75]
Hyaloperonospora arabidopsidis (*Hpa*)	HaRxL21	Transcriptional Co-repressor	TOPLESS (TPL)	*Arabidopsis thaliana*	[81]
Pseudomonas syringae	HopZ1 and HopX1	Transcriptional repressor	JASMONATE-ZIM DOMAIN (JAZ)	*Arabidopsis thaliana*	[74,76]
Laccaria bicolor	MiSSP7	Transcriptional repressor	JASMONATE-ZIM DOMAIN (JAZ)	*Populus trichocarpa*	[73]
Psuedomonas syringae	AvrPtoB	Transcriptional Co-activator	NONEXPRESSER OF PR GENES1 (NPR1)	*Arabidopsis thaliana*	[72]
Phytopthora capsici	RxLR48	Transcriptional Co-activator	NONEXPRESSER OF PR GENES1 (NPR1)	*Arabidopsis thaliana*	[71]

3.2 Multi-level regulation of transcriptional control

Plant immune transcriptional regulation is orchestrated through a complex, multi-level network involving both general and highly specific responses (**Figure 2**). Early immune responses often involve a general stress response, which is activated by both biotic and abiotic stressors[82–

[85], followed by more tailored transcriptional programs specific to the eliciting pathogen or molecular pattern[82,83,86–92]. This dynamic regulation is mediated by several layers, including post-translational modifications of transcription factors (TFs) such as phosphorylation[93,94], SUMOylation[95,96], and ubiquitination[97–101] which modulate TF activity, DNA binding, and stability. Together, these regulatory strategies provide plants with the flexibility and precision required to interpret diverse immune cues and mount appropriately scaled defense responses.

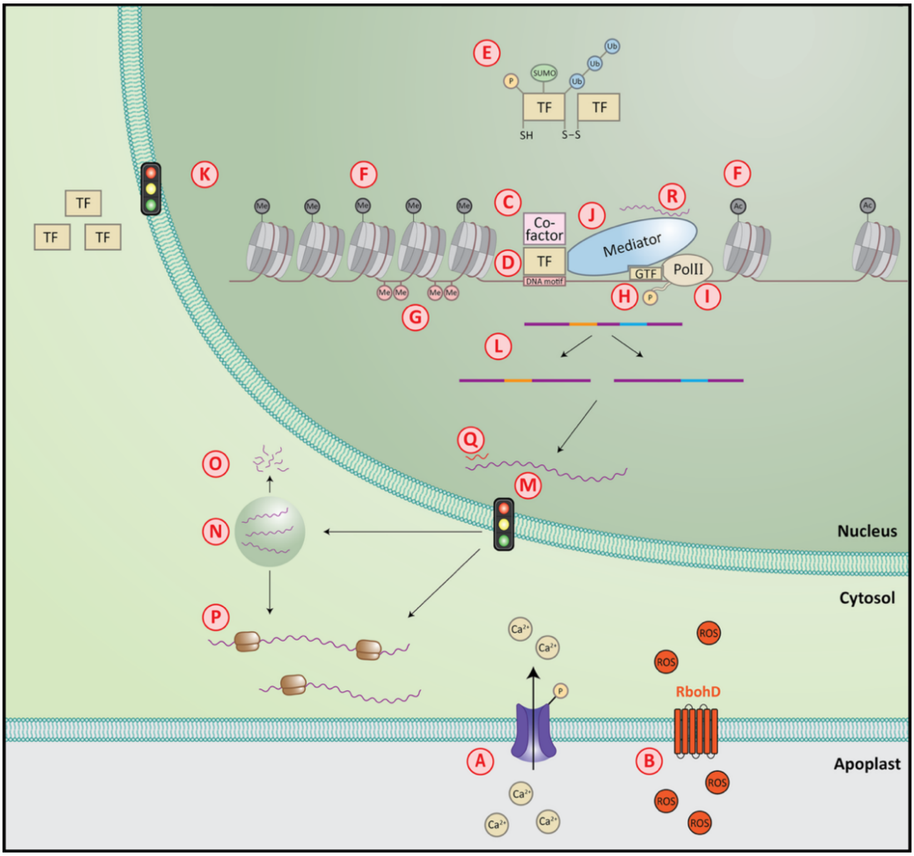

Figure 2. Regulation of transcriptional control occur at multiple levels. Mechanisms involved in the regulation of immune-related transcription. (A) regulation of calcium (Ca^{2+}) influx, which may lead to post-translational modifications of TFs; (B) generation of ROS by RbohD, which may lead to post-translational modifications of TFs (see also Figure 2E); (C) co-factors that may contribute to regulation of transcription; (D) TFs regulate transcription by binding to a motif; (E) post-translational modifications of TFs, such as phosphorylation (P), sumoylation (SUMO), ubiquitination (Ub) and forming of oligomers through S-S bridges depending on the redox state; (F) modifications of histones (methylation [Me] or acetylation [Ac]) to regulate the chromatin state; (G) methylation of DNA; (H) phosphorylation of the C-terminal domain of RNA-polymerase II (PolII) promotes transcription; (I) PolII may initiate transcription at alternative transcription start sites; (J) the Mediator complex forms the bridge between specific TFs, general TFs (GTF) and PolII; (K) selective import of TFs or other proteins; (L) alternative splicing; (M) selective retention of mRNAs in the nucleus; (N) temporary storage of mRNAs in stress granules or P-bodies; (O) degradation of mRNAs from P-bodies; (P) release of mRNAs from stress granules or P-bodies into the cytosol, followed by translation; (Q) post-transcriptional gene silencing by small RNAs; (R) long non-coding RNAs can regulate transcription in different ways, depicted here is modulation of MED19a by ELENA1.

3.3 Chromatin and transcription initiation

Chromatin accessibility plays a critical role in shaping transcriptional outcomes during immune activation. This is governed by histone modifications, nucleosome repositioning, and histone variant exchange, which together influence the accessibility of regulatory DNA regions[102–107]. Notably, compelling evidence shows that transcriptional changes across different immune responses are tightly correlated with chromatin accessibility[16]. In parallel, dynamic DNA methylation and demethylation, mediated by enzymes such as ROS1 and DEMETER (DME), fine-tune promoter accessibility and thereby modulate immune gene expression[108–110]. At the level of the transcriptional machinery, phosphorylation of RNA

Polymerase II's C-terminal domain by immune-activated kinases promotes transcriptional initiation, while phosphatases like CPLs act as negative regulators[111,112]. Additional regulatory precision is achieved through alternative transcription initiation and the activity of the Mediator complex, which can recruit Pol III, Pol IV, or Pol V depending on the transcriptional context and hormonal signaling environment [113–119]. Finally, nuclear import and export mechanisms contribute to the spatial regulation of immune signaling by controlling the localization of key transcriptional regulators such as NPR1, TOPLESS-RELATED1 (TPR1), and CONSTITUTIVE EXPRESSER OF PATHOGENESIS-RELATED GENES 5 (CPR5)-dependent immune factors[120–123].

4. Post-transcriptional regulation

Post-transcriptionally, immune gene expression is fine-tuned by alternative splicing, nuclear retention of mRNAs, and storage or decay in stress granules and P-bodies[124–132]. For instance, P-body components like DECAPPING1 (DCP1) are directly regulated by MPK signaling to promote decay of specific mRNAs during PTI[131,132]. Non-coding RNAs including microRNAs and long non-coding RNAs (lncRNAs) add yet another regulatory dimension. miRNAs participate in post-transcriptional gene silencing and are epigenetically controlled during infection[133,134], while lncRNAs such as ELENA1 enhance immune gene expression by modulating Mediator complex interactions and chromatin dynamics (**Figure 2**)[135,136]. Collectively, these interconnected layers of transcriptional and post-transcriptional regulation enable plants to execute robust, precisely coordinated immune responses while maintaining developmental and metabolic homeostasis.

5. Ca^{2+} signaling and role of CBP60 family in plant immunity

A rapid influx of calcium and a change in redox status are vital parts of plant immunity and they play intertwined roles in PTI and ETI[137]. Calcium influx is induced immediately upon perception of PAMPs and effectors, which has been coupled to classical calcium channels[15,138], but also to recently identified calcium channels formed by helper NLR-based resistosomes[57,139] (**Figure 1**). Intracellularly, the calcium signal is decoded by calcium-binding proteins like calmodulin (CaM) and Ca^{2+}-dependent protein kinases (CDPKs). These can directly activate TFs, such as the defense-regulating CaM-binding TF family CAMTAs and CBP60s, or WRKY28, WRKY33 and WRKY48, which are phosphorylated by CPK5 and CPK6. This leads to altered defense-related transcription by these TFs, which influences resistance to diverse pathogens[93,94,140,141]. Although in general positive effects of calcium signaling on immunity have been reported, this is not always the case. For example, the Ca^{2+}-activated CAMTA3 (or AtSR1) TF represses SA signaling[142]. Moreover, several other CaM-regulated and CaM-like proteins like CBP60a, CALMODULIN-LIKE 46 (CML46) and CML47 negatively impact SA-related gene expression and accordingly, mutant lines are enhanced resistant to virulent *P. syringae*[143].

The Arabidopsis CBP60 family consists of eight members, several of which have emerged as important regulators of plant defense[62,144]. While most members positively influence immunity, CBP60a is a notable exception, acting as a negative regulator of SA-mediated responses[144]. Among the family, CBP60g and SYSTEMIC ACQUIRED RESISTANCE DEFICIENT 1 (SARD1) are the best characterized and function as central transcriptional hubs in immune signaling. Both are rapidly induced upon pathogen recognition and bind directly to the promoters of numerous defense-related genes[140,145], including those involved in SA and NHP (N-hydroxypipecolic acid) biosynthesis, PTI, ETI-specific genes (**Figure 3**).

Importantly, CBP60g and SARD1 act redundantly, loss of one can be partially compensated by the other, but simultaneous disruption of both severely compromises immunity[62,140]. Beyond activating positive regulators, emerging evidence suggests that CBP60g and SARD1 also bind to and repress specific negative regulators, enabling them to fine-tune immune output and prevent inappropriate or excessive defense activation. However, a comprehensive study systematically linking the roles of CBP60g and SARD1 to resistance against diverse pathogens and across distinct immune pathways (PTI and ETI) has been lacking. Moreover, a global transcriptional dissection uncoupling their dual roles in activation and repression is needed to fully understand how these transcription factors sit at the heart of a balanced immune regulatory framework, coordinating both pathogen resistance and immune-associated cell death. In addition, it is also important to determine how CBP60g/SARD1-dependent programs are deployed across distinct cellular contexts.

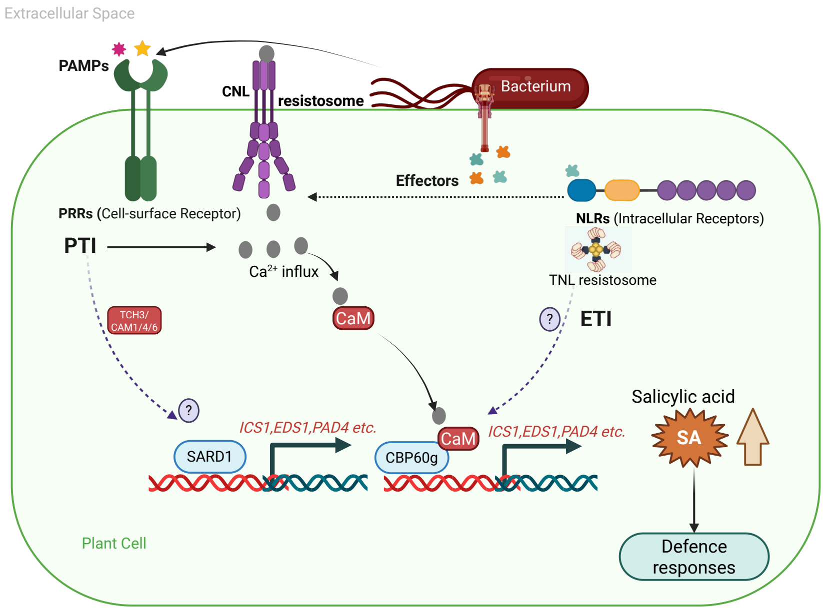

Figure 3. Overview of CBP60g and SARD1 in plant innate immunity. Pathogen recognition by cell-surface receptors and intracellular receptors lead to activation of PTI and ETI. Activation of plant immunity leads to production of reactive oxygen species, callose deposition, cytosolic Ca^{2+} flux, kinase activation, induction of CBP60g and SARD1. CBP60g is activated by Ca^{2+}-dependent calmodulin (CaM) binding (TCH3, CAM1/4/6) and SARD1 is activated by an unknown mechanism, which both activates the expression of *ENHANCED DISEASE SUSCEPTIBILITY 1* (*EDS1*), *PHYTOALEXIN DEFICIENT 4* (*PAD4*), *ISOCHORISMATE synthase 1* (*ICS1*) for promoting SA accumulation and the downstream SA-dependent defense responses.

6. Cell-autonomous immunity across diverse cell states

Understanding how individual plant cells respond to pathogenic threats is essential for unravelling the spatial and functional complexity of plant immunity. Unlike animals, plants lack mobile immune cells and therefore rely on cell-autonomous immune responses. Effector-triggered immunity (ETI), a potent intracellular defense mechanism mediated by nucleotide-binding leucine-rich repeat (NLR) receptors upon effector recognition, is central to this response[11]. However, traditional bulk transcriptomic approaches often obscure the cellular heterogeneity of immune responses, masking how different cells maintain the potential to mount immune activation, and what defines their shared versus cell-type-specific defense responses when pathogen exposure occurs stochastically across a tissue.

The advent of single-cell RNA sequencing (scRNA-seq) has enabled unprecedented resolution in dissecting these responses, revealing that while immune perception can be broadly uniform, the transcriptional execution of ETI is highly shaped by cell identity, developmental context, and chromatin accessibility meaning that the same immune signal may be interpreted differently depending on the cellular landscape in which it is triggered[17]. Importantly, pathogen encounters are rarely synchronized across all tissues. In natural infections, only a subset of cells perceive pathogen, while surrounding cells experience indirect immune cues. This creates spatially layered immune zones, where infected cells, adjacent bystander cells, and distal tissues may enter distinct transcriptional and metabolic states. Such spatial compartmentalization also highlights the importance of cell-to-cell communication, including local propagation of immune signals and longer-range systemic signaling that primes distal tissues. Recent single-cell studies have revealed that immune activation can organize into “hotspots,” including rare immune-active populations such as PRIMER cells, which occupy the center of immune domains and are surrounded by transcriptionally distinct neighbouring cells[146]. Similar spatial immune patterning has also been observed in other plant species, reinforcing that tissue immunity emerges not only from individual defense responses but also from their coordination across cellular neighborhoods[147].

These findings position scRNA-seq as a transformative tool in plant immunity research, particularly when coupled with spatial omics technologies can help in mapping immune states back to their anatomical context, capturing how immune outputs propagate from infection sites to adjacent and distal tissue regions. In addition, integrating scRNA-seq with other single-cell omics, such as single-cell Assay for transposase-accessible chromatin using sequencing (ATAC-seq), enables the inference of gene regulatory networks (GRNs) that link transcriptional outputs to underlying chromatin accessibility. This multi-omics framework can reveal how cell identity and regulatory state constrain immune competence and shape ETI execution across tissues.

Lastly, disentangling intrinsic immune potential from infection-driven variability remains a major challenge. Synthetic systems that can uniformly activate immune signaling in the absence of pathogens offer a powerful approach to disentangle intrinsic cell-type immune potential from pathogen-induced variability. By synchronizing immune activation across all cells, these systems enable a controlled view of how different cell types interpret and execute the same immune signal.

Together, these approaches open new avenues to unravel the logic of immune execution across plant tissues and lay the groundwork for designing next-generation strategies to engineer precise, robust, and spatially optimized disease resistance in crops.

7. Thesis outline

Despite extensive knowledge of plant immune signaling, many questions remain unanswered:

1. How are ETI outputs like resistance and cell death genetically separated through EDS1-containing modules?
2. How is effector-triggered immunity executed across distinct leaf cell-types?
3. How do master regulators like CBP60g and SARD1 orchestrate transcriptional dynamics to optimize immune outputs?

To address these questions, this thesis is structured into five chapters:

Chapter 1 provides a general introduction to the field of plant immunity, with a focus on the architecture and signaling logic of effector-triggered immunity (ETI). It outlines how plants, in the absence of mobile immune cells, rely on cell-autonomous defense strategies mediated by intracellular nucleotide-binding leucine-rich repeat (NLR) receptors. The chapter introduces key signaling modules such as EDS1-PAD4 and EDS1-SAG101 and discusses how these modules contribute differently to immune outputs.

Chapter 2 explores how two key EDS1-containing signaling modules EDS1-PAD4 and EDS1-SAG101, acts together with the helper NLRs (hNLRs) and differentially contribute to effector-triggered immunity (ETI) outcomes in plants. Through inducible immune activation, we demonstrate that the EDS1-PAD4-ADR1 module primarily governs disease resistance and immune-associated growth arrest, whereas the EDS1-SAG101-NRG1 module predominantly triggers hypersensitive response (HR)-mediated cell death. These findings reveal that distinct ETI outputs can be genetically uncoupled, offering a framework to investigate their respective transcriptional consequences. Additionally, this chapter introduces the concept of disease priming and discusses its potential application in enhancing plant defense, particularly in cases where immune responses are even partially compromised.

Building on the modular framework of effector-triggered immunity (ETI), **chapter 3** employs single-cell RNA sequencing (scRNA-seq) to profile immune responses across all major cell types in the Arabidopsis leaf, using a synthetic, inducible ETI activation system. We show that while ETI perception is uniform across cells, its execution is highly context-dependent, shaped by cell-type-specific chromatin accessibility and transcription factor networks. Notably, we uncover a spatial organization of immune regulators, with distinct transcriptional modules acting in specific cell types. For example, the transcription factors SARD1 and CBP60g are preferentially expressed in epidermal cells, and we demonstrate their functional necessity in restricting pathogen entry at the leaf surface. These findings highlight the spatial specialization of immune responses and underscore the importance of cell identity in orchestrating effective defense programs.

Chapter 4 centres on the transcription factors CBP60g and SARD1, which serve as master regulators of salicylic acid (SA)-mediated immunity in plants. Using mutant analyses, inducible ETI systems, and transcriptomic profiling, we reveal that these transcription factors not only promote disease resistance but also play a critical role in fine-tuning the balance between immune activation and cell death. Notably, the *cbp60g sard1* double mutant emerges as a valuable genetic resource for uncoupling resistance from hypersensitive response (HR). These mutants display exaggerated HR yet show compromised disease resistance, all without apparent growth defects challenging the long-held view that HR is a prerequisite for effective immunity. Furthermore, our data indicate that CBP60g and SARD1 modulate both positive and negative regulators of immune signaling, shaping a finely balanced transcriptional network during ETI.

The final **chapter 5** of this thesis summarizes the key findings and conclusions of my research, placing them within the broader context of plant immune regulation. I discuss the significance of these results in advancing our understanding of cell-type-specific immune responses, and reflect on the new questions that have emerged, highlighting areas for future investigation. Additionally, I outline potential applications and future perspectives, emphasizing how insights from this work could contribute to more sustainable and targeted strategies for combating plant diseases.

Together, these studies advance our understanding of the transcriptional and spatial logic underlying plant innate immunity and highlight the modular strategies used by plants to fine-tune defense.

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