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REVIEW ARTICLE

Plant Immunity: A Plastic System Operated Through Cell-Fate Transition

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Abstract
Plants are continuously exposed to pathogen challenges. To defend themselves, plants have developed sophisticated innate and induced immune responses. The recognition of invading pathogens by membrane-localized or intracellular receptors triggers a local immune response, but plants often also establish a systemic immunity throughout the entire plant body to confer broad-spectrum and long-lasting resistance to secondary infections. Both the local and systemic immune responses are regulated by several phytohormones, including jasmonic acid, ethylene, and salicylic acid, which induce genome-wide transcriptional reprogramming to elicit effective immune responses. During this transcriptional reprogramming, epigenetic mechanisms are engaged to modulate chromatin structure and the accessibility of *cis*-elements to transcription factors. In this review, we first describe how the model plant *Arabidopsis thaliana* recognizes invading pathogens to trigger local and systemic immune responses. Next, we describe how phytohormones mediate transcriptional responses, establishing immunity. Finally, we review recent findings in the epigenetic aspect of immunity in Arabidopsis.

Keywords Local immunity · Systemic immunity · Salicylic acid · NPR1 · Transcriptional reprogramming · Epigenetic mechanisms

Introduction

Plants are potential hosts for a wide range of pathogens and continuously confront diverse pathogen challenges during their lifetime. Unlike vertebrates, plants lack specialized cells and circulatory systems developed for immunity; however, plants do have a capacity to trigger innate immune responses. Phytopathogens have developed various strategies to enhance their invasion, survival, and proliferation within host plants, to which plants have responded by developing sophisticated immune strategies.

The innate immunity of plants relies on large numbers of receptors for the surveillance of pathogen attacks. Plant cells recognize pathogen invasions using cell-surface receptors that detect the conserved molecular structures present in pathogens, and intracellular receptors that sense specific molecules secreted by pathogens. Signals initiated from pathogen recognition are conveyed to hormone-mediated signaling pathways, which induce massive transcriptional reprogramming to turn on the expression of defense-related genes and repress growth-promoting genes. In addition to eliciting local immunity, plant cells exposed to pathogen challenges generate mobile signals that trigger a systemic immunity to enable the whole plant body to be prepared for subsequent pathogen challenges. Through this local and systemic immune signaling, plants convert their cells into an immune-equipped state, and thus operate a plastic rather than a permanently specified immune system.

Local Immunity

In plants, local immunity is composed of a two-tiered innate immune response: pathogen-associated molecular pattern (PAMP)-triggered immunity (PTI) and effector-triggered immunity (ETI) (Fig. 1). PAMPs, such as flagellin, the bacterial elongation factor Tu, and chitin, are small molecular

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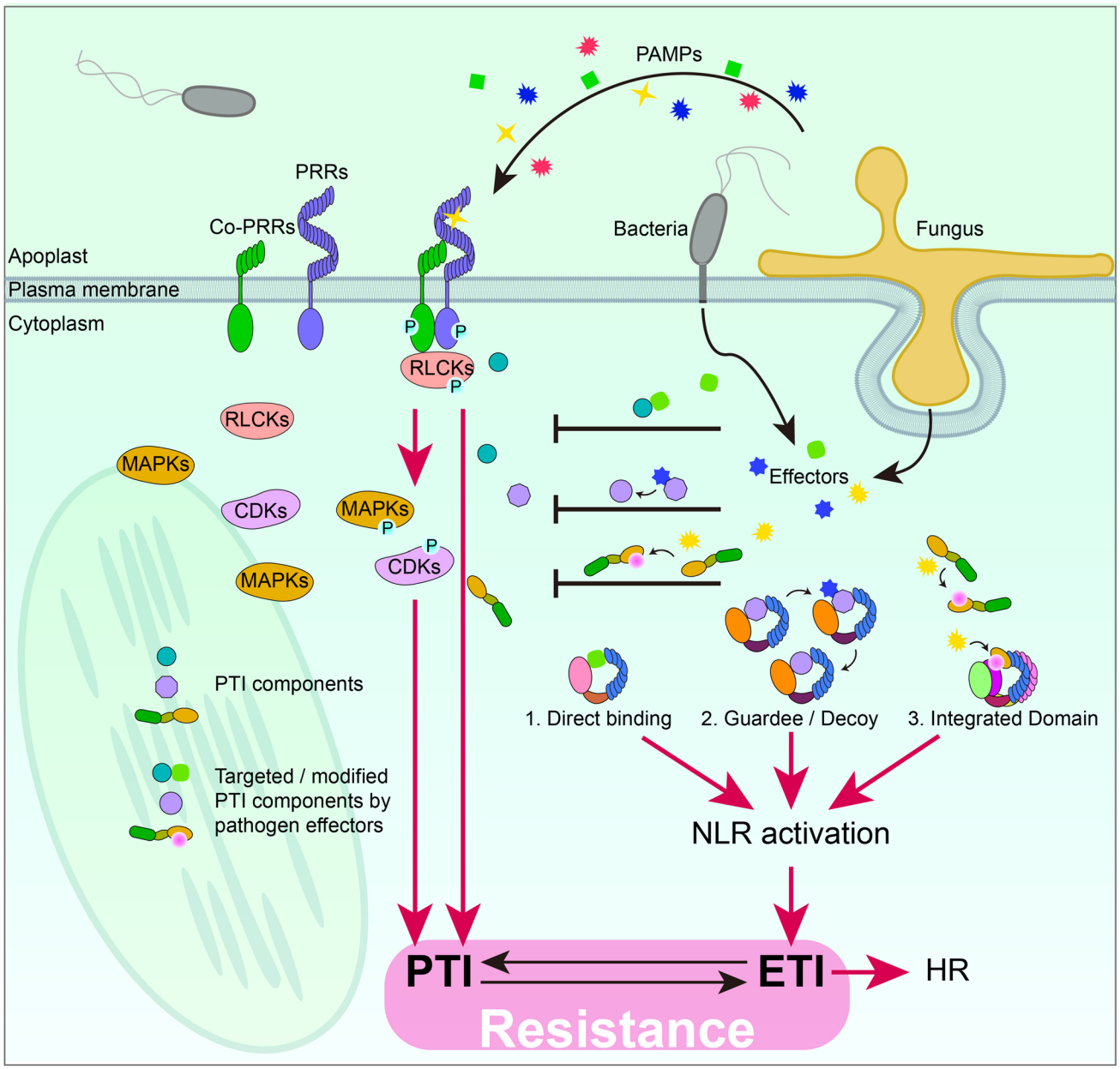


Fig. 1 Schematic of pathogen-associated molecular pattern (PAMP)-triggered immunity (PTI) and effector-triggered immunity (ETI) in Arabidopsis. In PTI, PAMPs derived from invading pathogens are perceived by PAMP recognition receptors (PRRs) in the plasma membrane, which also leads to the interaction between PRRs and co-PRRs. Activated PRR complexes transmit signals through phosphorylation relays: receptor-like cytoplasmic kinases (RLCKs) are thought to relay PRR-derived signals to multiple downstream signaling pathways, including those involving mitogen-activated protein kinase (MAPK) cascades and calcium-dependent kinases (CDKs). To suppress PTI, phytopathogens secret effector molecules into host cells. Pathogen effectors bind to host proteins and can induce conformational changes or post-translational modifications; however, host nucleotide-binding domain leucine-rich-repeat receptors (NLRs) can specifically recognize pathogen effectors either directly or indirectly and disable their activity. In indirect perception mechanisms, NLRs interact with guardees or decoys to monitor the changes in their conformations or modifications caused by effectors. In some cases, NLRs contain integrated domains, which are subject to modulation by effectors. NLRs become activated when they sense the presence of effectors, triggering ETI. A programmed cell death called the hypersensitive response (HR) occurs only during ETI; however, PTI and ETI do exhibit substantial similarities in various immune responses. Intricate crosstalk between PTI and ETI is likely responsible for these similarities, and could enhance disease resistance

motifs that are conserved among a class of pathogens. PTI is induced when cell membrane-localized PAMP recognition receptors (PRRs) detect PAMPs. To suppress PTI and enhance their virulence, pathogens secrete effector molecules into host cells via the type III secretion system. ETI is induced when pathogen effectors are recognized by host intracellular receptors called disease resistance (R) proteins, which directly bind with the effectors or detect changes in host proteins induced by pathogen effectors.

Although PTI and ETI are differently triggered, these two immune responses eventually elicit various immune responses that are similar to each other, including calcium influx, reactive oxygen species (ROS) burst, callose deposition, stomatal closure, the production of antimicrobial secondary metabolites, the activation of mitogen-activated protein kinase (MAPK) cascades, and transcriptional reprogramming. The existence of an intricate crosstalk between PTI and ETI was recently described (Ngou et al. 2021; Pruitt et al. 2021; Yuan et al. 2021), which likely enables the two immune response pathways to coordinate and share various downstream events. Nonetheless, ETI is known to be activated more quickly and result in more robust immune responses than PTI. Furthermore, during ETI but not PTI, a programmed cell death called the hypersensitive response (HR) is developed to restrict the growth and spread of pathogens (Fig. 1).

Pathogen-Associated Molecular Pattern (PAMP)-Triggered Immunity (PTI)

PTI relies on PRRs that are localized in the plasma membrane. PRRs interact with co-receptors in a PAMP-induced manner to activate downstream signaling pathways (Fig. 1); for example, FLAGELLIN-SENSITIVE2 (FLS2) and EF-TU RECEPTOR (EFR), which are receptors for bacterial flg22 and EF-Tu, respectively, interact with the co-receptor BRI1-ASSOCIATED KINASE1 (BAK1) (Chinchilla et al. 2007). The PAMP-induced interaction between BAK1 and FLS2 or EFR is facilitated by FERONIA (FER) acting as a scaffold protein (Stegmann et al. 2017). In its basal state, BAK1 is sequestered from FLS2 or EFR by directly binding with BAK1-INTERACTING RECEPTOR-LIKE KINASE2 (BIR2) and BIR3 (Halter et al. 2014; Imkampe et al. 2017).

Similar to FLS2 and EFR, LYSM-CONTAINING RECEPTOR-LIKE KINASE5 (LYK5), a well-known chitin receptor, requires the co-receptor CHITIN ELICITOR RECEPTOR KINASE1 (CERK1) to trigger antifungal immunity (Cao et al. 2014). Interestingly, bacterial elicitors also induce BAK1-mediated CERK1 phosphorylation, which results in enhanced antifungal immunity (Gong et al. 2019). This example suggests that bacterial pathogen-induced PRR complexes might prime the PRR complexes to function in antifungal immunity.

Signals initiated from PAMP-induced PRR complexes are conveyed to receptor-like cytoplasmic kinases (RLCKs), including BOTRYTIS-INDUCED KINASE1 (BIK1), through auto- and trans-phosphorylation events. After its phosphorylation by BAK1, BIK1 directly phosphorylates the NADPH oxidase RESPIRATORY BURST OXIDASE HOMOLOGUE D (RbohD) to trigger a ROS burst (Li et al. 2014). Moreover, the BIK1-mediated phosphorylation of CYCLIC NUCLEOTIDE GATED CHANNEL4 (CNGC4) activates a heteromeric calcium channel composed of CNGC2 and CNGC4, resulting in a calcium influx into cytosol (Tian et al. 2019). In addition, PAMP-induced PRR complexes activate multiple downstream signaling pathways, including calcium-dependent kinases (CDKs) and MAPKs (Fig. 1).

Effector-Triggered Immunity (ETI)

ETI relies on intracellular receptors known as R proteins, most of which belong to a subfamily of nucleotide-binding domain leucine-rich-repeat receptors (NLRs) (Fig. 1). Plant NLRs contain either Toll/interleukin-1 receptor (TIR) domain, a coiled-coil (CC) domain, or a resistance to powdery mildew8-like (RPW8) domain at the N-terminus, resulting in their division into three classes: TIR-type NLRs (TNLs), CC-type NLRs (CNLs), and RPW8-type NLRs (RNLs), respectively.

NLRs recognize effectors secreted from pathogens using a variety of strategies. Some NLRs physically interact with the effectors; however, more commonly NLRs sense conformational changes or post-translational modifications of host proteins that are induced by the targeting of pathogen effectors. The indirect perception mechanisms of pathogen effectors by plant NLRs have been described in guardee and decoy models (van der Hoorn and Kamoun 2008), in which the guardee is a host protein playing a role in plant immunity and the decoy is a mimic of host protein targeted by the pathogen effector. Guardees or decoys are directly modified by pathogen effectors, and cognate NLRs recognize the changes of guardees or decoys to trigger ETI (Fig. 1).

The guardee protein RESISTANCE TO *P. SYRINGAE* PV MACULICOLA1 (RPM1)-INTERACTING4 (RIN4) is itself a negative regulator of PTI (Kim et al. 2005), and is targeted by the pathogen effectors AvrRpt2, AvrRpm1, and AvrB. To monitor these effectors, RESISTANT TO *P. SYRINGAE*2 (RPS2) and RPM1, the CNL class members, interact with RIN4 in the plasma membrane. While RPM1 senses AvrRpm1- and AvrB-induced RIN4 phosphorylation, RPS2 senses the AvrRpt2-mediated reduction in RIN4 and triggers the RPM1- or RPS2-dependent signaling pathways, respectively (Mackey et al. 2002; Axtell and Staskawicz 2003).

PROBABLE SERINE/THREONINE-PROTEIN KINASE2 (PBL2) acts as a BIK1 decoy in ETI. BIK1 uridylylation by an effector AvrAC suppresses BIK1-mediated PTI (Feng et al. 2012), while by contrast PBL2 uridylylated by AvrAC forms a complex with HOPZ-ACTIVATED RESISTANCE1 (ZAR1) via interacting with RESISTANCE RELATED KINASE1 (RKS1) to trigger ZAR1-mediated ETI (Wang et al. 2015). ZAR1-mediated ETI is also induced when ZAR1 senses the HopZ1a-mediated acetylation of HOPZ-ETI-DEFICIENT1 (ZED1) acting as a decoy (Lewis et al. 2013). ZAR1 forms a pentameric funnel-shaped structure, called the ZAR1 resistosome, on the plasma membrane (Wang et al. 2019), which when activated functions as a calcium-permeable channel (Bi et al. 2021).

A subset of plant NLRs carry integrated domains that mimic the binding targets of pathogen effectors; for instance, RESISTANT TO *RALSTONIA SOLANACEARUM*1 (RRS1) contains an integrated WRKY domain to which the AvrRps4 and PopP2 effectors bind. RPS4 interacts with RRS1 to sense AvrRps4 targeting to the WRKY domain and the Pop2-mediated acetylation of the WRKY domain (Sarris et al. 2015). WRKY proteins are DNA-binding transcription factors acting in the regulation of defense genes; thus, the two NLRs (RPS4 and RRS1) with RRS1 as a helper NLR act as a pair to detect effectors that interfere with the functions of the WRKY transcription factors.

In addition to effector sensing, helper NLRs act downstream of NLR-mediated signaling pathways. N REQUIREMENT GENE1 (NRG1) functions together with a heterodimer complex composed of ENHANCED DISEASE SUSCEPTIBILITY1 (EDS1) and SENESCENCE-ASSOCIATED GENE101 (SAG101) to trigger HR (Rietz et al. 2011), and ACTIVATED DISEASE RESISTANCE1 (ADR1) acts together with a heterodimer composed of EDS1 and PHYTOALEXIN-DEFICIENT4 (PAD4) to promote the biosynthesis of salicylic acid (SA), a key phytohormone in plant immunity (Dong et al. 2016). NRG1 and ADR1 associate with the EDS1/SAG101 and EDS1/PAD4 complex, respectively, following the activation of the TNL-mediated signaling pathways (Sun et al. 2021), which are converged on a lipase-like protein, EDS1 (Rietz et al. 2011).

Taken together, plant NLRs generally detect self-molecules perturbed by pathogen effectors rather than non-self-molecules, suggesting that plants with limited defense genes effectively sense diverse effectors sharing target molecules.

Systemic Immunity

Besides local immunity at infection sites, plants trigger systemic immunity to protect the rest of the plant body from subsequent pathogen attack. This phenomenon is called systemic acquired resistance (SAR), and confers long-lasting and broad-spectrum resistance. It is established by a slight increase in the levels of SA in uninfected tissues and is accompanied by the expression of the SA marker *PATHOGENESIS-RELATED* (*PR*) genes (Yalpani et al. 1991; Gaffney et al. 1993; Wildermuth et al. 2001). The establishment of SAR is severely impaired by mutations in *NON-EXPRESSOR OF PATHOGENESIS-RELATED GENES1* (*NPR1*) (Cao et al. 1994), suggesting that NPR1 acts as an essential regulator of SAR. Even though both SA accumulation and SA-mediated signaling are required for SAR, SA itself is unlikely to act as a mobile signal (Vernooij et al. 1994; Ryals et al. 1995). The roles of SA and NPR1 in local immunity will be further explained later in this review.

Instead of SA, methyl salicylate (MeSA) was once proposed to be a phloem-mobile signal that could induce the systemic immune response upon hydrolysis to active SA in systemic tissues, as well as being an airborne signal that can induce disease resistance in neighboring plants (Park et al. 2007). This molecule is unlikely to be the main systemic immunity signal however, as it was later reported that most MeSA accumulated in the pathogen-infected leaves is emitted into the atmosphere, and SAR is not affected by mutations in an SA methyltransferase gene (Attaran et al. 2009).

Recent studies have suggested that a new pathway involving pipecolic acid (Pip) → nitric oxide (NO) ↔ ROS → azelaic acid (AzA) → glycerol-3-phosphate (G3P) acts in parallel with the SA pathway for the establishment of SAR. Pip is increased both in the petiole exudates of infected and uninfected tissues upon pathogen infection, and an exogenous application of Pip induces SAR (Navarova et al. 2012). The Pip-mediated SAR is dependent on FLAVIN-DEPENDENT-MONOOXYGENASE1 (FMO1) catalyzing the conversion of Pip to N-hydroxypipecolic acid (NHP) (Hartmann et al. 2018). The de novo biosynthesis of Pip in uninfected but not in infected tissues is dependent on both SA and G3P, and Pip functions upstream of the NO/ROS-mediated signaling pathway in both uninfected and infected tissues (Wang et al. 2018). Following the Pip-induced accumulation of NO/ROS, several ROS act in an additive manner to induce AzA biosynthesis, which results in G3P biosynthesis (Wang et al. 2014). The biosynthesis of G3P is dependent on two lipid-transfer proteins, DEFECTIVE IN INDUCED RESISTANCE1 (DIR1) and AZELAIC ACID INDUCED1 (AZI1), which interact with each other (Yu et al. 2013a). G3P may act as a mobile signal to induce SAR, and its translocation to uninfected tissues is interdependent with DIR1 (Chanda et al. 2011).

Once systemic immunity is established, plants are primed to induce more rapid and robust immune responses upon subsequent pathogen challenges. Defense priming is a type of immune memory that plants adopt as an adaptive strategy to survive their continuous exposure to surrounding pathogens. It was reported that bacterial pathogen- or herbivore-induced

defense priming could be inherited to subsequent generations (Luna et al. 2012; Rasmann et al. 2012; Slaughter et al. 2012); however, Yun et al. recently reported that they did not find evidence of the transgenerational inheritance of defense priming using a variety of methods used for pathogen infection and multiple developmental stages of host plants undergoing repetitive pathogen challenges (Yun et al. 2022). Thus, the transgenerational effect of defense priming should be more carefully reassessed in the context of the underlying molecular mechanisms.

Systemic immunity is also activated by beneficial microorganisms such as mycorrhizal fungi and plant growth-promoting rhizobacteria. This phenomenon is termed induced systemic resistance (ISR) to be distinguished from SAR. While SAR is dependent on SA-mediated signaling, ISR is dependent on jasmonic acid (JA)/ethylene (ET)-mediated signaling.

Phytohormone-Mediated Signaling Pathways

Initiated from pathogen recognition, defense signals are transmitted in the form of phytohormones for signal amplification and transcriptional reprogramming. JA, ET, and SA are key hormones that play central roles in plant immunity. JA and ET are essential for defense against necrotrophic pathogens and herbivores, which destroy host cells to obtain nutrients and cause wounding to host plants, respectively. On the contrary, SA is responsible for defense against biotrophic pathogens, which derive nutrients from living host cells, and hemibiotrophic pathogens, which display an initial biotrophic phase before moving to a necrotrophic phase.

It has been documented that JA- and ET-mediated signaling pathways are synergistic, whereas the JA/ET- and SA-mediated signaling pathways are mutually antagonistic (Li et al. 2019); however, recent findings showed that the JA/ET- and SA-mediated signaling pathways are not always antagonistic. For instance, an exogeneous treatment of the ethylene precursor 1-aminocyclopropane-1-carboxylic acid (ACC) enhanced the SA-induced expression of *PR1*, and this effect was not shown in the *ethylene insensitive2-1* (*ein2-1*) mutant (De vos et al. 2006). By analyzing gene expression levels, another study reported that the synergistic effect of SA and JA is revealed when both hormones are applied at low concentrations, whereas an antagonistic effect is observed at high concentrations (Mur et al. 2006). It was also reported that during ETI involving SA-mediated signaling, the JA-responsive genes required for de novo JA biosynthesis are induced in an NPR3 and NPR4-dependent manner, and the increased JA levels contribute to HR induction (Liu et al. 2016). Mine et al. showed that a co-treatment with flg22 and methyl jasmonic acid (MeJA) enhanced resistance to a biotrophic pathogen in a *delayed dehiscence2* (*dde2*) *pad4* double mutant to a greater extent than the flg22 treatment alone (Mine et al. 2017). Collectively, hormone-mediated signaling pathways in plant immunity are coordinated by a variety of cross-talks, constituting a complex signaling network.

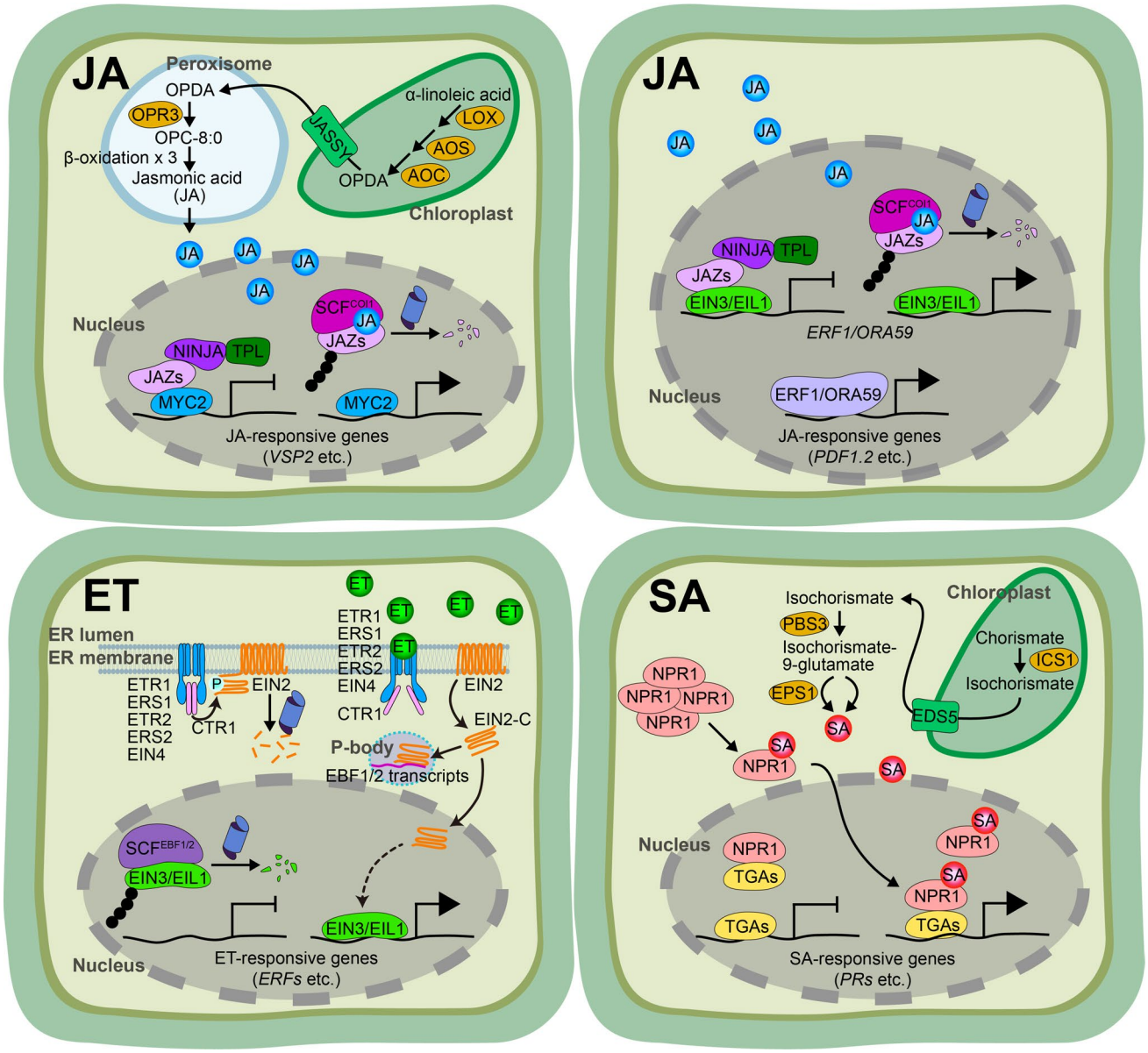
Jasmonic Acid (JA)-Mediated Signaling Pathway

JA is biosynthesized in the chloroplasts and peroxisomes via the octadecanoid pathway involving several enzymatic steps. The biosynthesis of JA is initiated in the chloroplasts, where α-linoleic acid is converted into oxophytodienoic acid (OPDA). The export of OPDA into the cytosol is facilitated by JASSY proteins localized on the outer membrane of the chloroplasts (Guan et al. 2019). After their import into the peroxisome, OPDA is reduced by OPDA REDUCTASE3 (OPR3) and then converted into JA through three cycles of β-oxidation (Schaller et al. 2000) (Fig. 2).

CORONATINE-INSENSITIVE1 (COI1) acts as a receptor for the bioactive form of JA, jasmonoyl-L-isoleucine (JA-Ile), and is the F-box subunit of the SKP1-CULLIN1-F-BOX-TYPE (SCF) ubiquitin ligase complex SCF^{COI1} (Katsir et al. 2008; Yan et al. 2009). JA-Ile mediates the binding of COI1 to the JASMONATE ZIM-DOMAIN (JAZ) proteins (repressors of JA-responsive genes) and, thus, results in the 26S proteasome-mediated degradation of JAZs (Chini et al. 2007; Thines et al. 2007) (Fig. 2).

In their basal state, JAZs suppress the function of the transcriptional activators of the JA-responsive genes by forming a transcriptional co-repressor complex with NOVEL INTERACTOR OF JAZ (NINJA), TOPLESS (TPL), and the TPL-RELATED PROTEINS (TPRs) (Pauwels et al. 2010). MYC2 is a binding target of the JAZs and acts as a key transcriptional activator of the JA-induced genes (Kazan and Manners 2013); thus, the JA-induced degradation of JAZs causes the release of MYC2 from its suppression, leading to the transcriptional activation of the JA-responsive genes (Fig. 2). This MYC2 branch of JA-mediated immunity is known to be induced by herbivores and mechanical wounding.

On the contrary, the ERF branch of JA-mediated immunity is induced by necrotrophic pathogens. The ERF branch is dependent on the OCTADECANOID-RESPONSIVE ARABIDOPSIS AP2/ERF59 (ORA59) and ETHYLENE RESPONSE FACTOR1 (ERF1) transcription factors. *ORA59* and *ERF1* expression is activated by ETHYLENE INSENSITIVE3 (EIN3) and EIN3 LIKE1 (EIL1), with which the JAZs interact to suppress their activities (Zhu et al. 2011). Thus, the JA-triggered degradation of JAZs results in the EIN3/EIL1-induced transcriptional activation of *ORA59* and *ERF1* (Fig. 2). The ERF branch shows a synergistic interaction with ET-mediated signaling but an



antagonistic interaction with the MYC2 branch. The antagonistic interaction between the ERF and MYC2 branches is fulfilled in such a way that EIN3 and MYC2 mutually inhibit their transcriptional activities by binding to each other (Song et al. 2014).

Ethylene (ET)-Mediated Signaling Pathway

The gaseous hormone ET is produced in a two-step enzymatic reaction. In *Arabidopsis thaliana*, multiple ET receptors including ETYLENE RESPONSE1 (ETR1), ETHYLENE RESPONSE SENSOR1 (ERS1), ETR2, ERS2, and EIN4 have been identified. All of these ET receptors are localized in the membrane of the endoplasmic reticulum (ER).

Upon ET perception, the fragment generated by EIN2 cleavage at the C-terminus (EIN2-C) is translocated into the nucleus. By contrast, under normal conditions, CONSTITUTIVE TRIPLE RESPONSE1 (CTR1)-mediated EIN2 phosphorylation not only prevents the cleavage but also promotes EIN2 degradation (Ju et al. 2012; Qiao et al. 2012). When generated by ET signaling, the EIN2-C directly binds to the 3′ untranslated regions (UTRs) of the *EIN3-BINDING F BOX PROTEIN1* (*EBF1*) and *EBF2* transcripts to repress their translation (Li et al. 2015; Merchante et al. 2015). While the two F-box proteins, EBF1 and EBF2, mediate the degradation of EIN3/EIL1, which are essential transcription factors required for ET-induced gene expression under normal conditions (Potuschak et al. 2003), EIN3 and EIL1 become stabilized by ET signaling and trigger the

◂**Fig. 2** Phytohormone-mediated signaling pathways leading to transcriptional reprogramming and immunity in Arabidopsis. For jasmonic acid (JA) biosynthesis, α-linoleic acid is first converted to oxophytodienoic acid (OPDA) by a series of reactions catalyzed by various enzymes, including LIPOXYGENASE (LOX), ALLENE OXIDE SYNTHASE (AOS), and ALLENE OXIDE CYCLASE (AOC), in the chloroplasts. OPDA is then exported through JASSY from the chloroplast to the cytoplasm and subsequently imported into the peroxisome. Within the peroxisome, OPDA is reduced by OPDA REDUCTASE3 (OPR3), and the reduced product is shortened through three cycles of β-oxidation to generate JA. JA is perceived by CORONATINE-INSENSITIVE1 (COI1), which comprises the F-box subunit of the SKP1-CULLIN1-F-BOX-TYPE (SCF) ubiquitin ligase complex SCF^{COI1}. This complex mediates the degradation of the JASMONATE ZIM-DOMAIN (JAZ) proteins through 26S proteasome. In the absence of JA, JAZs interact with MYC2 to suppress its transcriptional activity by forming a co-repressor complex with NOVEL INTERACTOR OF JAZ (NINJA) and TOPLESS (TPL). The JA-induced degradation of the JAZs enables MYC2 to induce the transcriptional activation of the JA-responsive genes, including *VEGETATIVE STORAGE PROTEIN2* (*VSP2*). The JAZs also repress the transcription of *ETHYLENE RESPONSE FACTOR1* (*ERF1*) and *OCTADECANOID-RESPONSIVE ARABIDOPSIS AP2* (*ORA59*) by interacting with ETHYLENE INSENSITIVE3 (EIN3) and EIN3 LIKE1 (EIL1). Upon JA-induced degradation of the JAZs, EIN3 and EIL1 induce the transcription of *ERF1* and *ORA59*, resulting in the subsequent expression of JA-responsive genes including *PLANT DEFENSIN1.2* (*PDF1.2*). In the absence of ethylene (ET), CONSTITUTIVE TRIPLE RESPONSE1 (CTR1) interacts with multiple ET receptors, including ETYLENE RESPONSE1 (ETR1), ETHYLENE RESPONSE SENSOR1 (ERS1), ETR2, ERS2, and EIN4, and directly phosphorylates the C-terminal domain of EIN2 within the membrane of the endoplasmic reticulum (ER). EIN2 phosphorylation by CTR1 prevents the cleavage of the EIN2-C fragment but facilitates the degradation of EIN2 through the 26S proteasome. In addition, EIN3 and EIL1 are degraded by the $SCF^{EBF1/2}$ in the absence of ET in the nucleus. When ET receptors in the ER membrane perceive ET, the kinase activity of CTR1 becomes inactivated. As a result, EIN2-C is cleaved, binds with the *EBF1/2* transcripts, and targets them to the cytoplasmic processing body (P-body) for their translational repression. As a result, EIN3 and EIL1 are released from $SCF^{EBF1/2}$-mediated degradation upon ET signaling. EIN2-C is also translocated into the nucleus and activates the transcription of the ET-responsive genes, including the *ERF*s, through the activity of the undegraded EIN3 and EIL1. Salicylic acid (SA) is biosynthesized through the isochorismate pathway upon pathogen infection. In the chloroplasts, chorismate is converted to isochorismate by ISOCHORISMATE SYNTHASE1 (ICS1). Isochorismate is then exported from the chloroplast to the cytoplasm by ENHANCED DISEASE SUSCEPTIBILITY5 (EDS5). Next, AVRPPHB SUSCEPTIBLE3 (PBS3) conjugates glutamate to isochorismate, generating isochorismate-9-glutamate. Finally, isochorismate-9-glutamate is converted into SA by ENHANCED PSEUDOMONAS SUSCEPTIBLITY1 (EPS1) or through spontaneous decay. SA induces the conversion of oligomeric NONEXPRESSOR OF PATHOGENESIS-RELATED GENES1 (NPR1) proteins into their monomeric form. The monomerized NPR1 proteins are then translocated into the nucleus. Although NPR1 forms a complex with TGACG motif-binding transcription factors (TGAs) even in the absence of SA, the TGAs but not the NPR1–TGA complex are targeted to and repress the transcription of the SA-responsive genes, including the *PR*s. When NPR1 perceives SA via its direct binding, the NPR1–TGA complex is targeted to and activates the transcription of the SA-responsive genes

ET responses by inducing the expression of numerous ET-responsive genes (Fig. 2).

Salicylic Acid (SA)-Mediated Signaling Pathway

Two SA biosynthetic pathways have been identified in plants, one involving ISOCHORISMATE SYNTHASE1 (ICS1) and the other involving PHENYLALANINE AMMONIA-LYASE (PAL) (Lefevere et al. 2020). In Arabidopsis, SA is mainly biosynthesized through the isochorismate pathway involving ICS1 upon pathogen infection, whereas the PAL pathway only has a minor effect on SA-mediated immunity.

ICS1 catalyzes the conversion of chorismate to isochorismate in the chloroplast. The expression of *ICS1* is activated by two transcriptional activators, CALMODULIN BINDING PROTEIN 60-LIKE G (CBP60g) and its homolog SYSTEMIC ACQUIRED RESISTANCE DEFICIENT1 (SARD1) (Zhang et al. 2010). EDS5 was proposed to export isochorismate from the chloroplasts into the cytoplasm on the basis that *EDS5* encodes a multidrug and toxin extrusion (MATE) transporter protein and SA accumulation is compromised in an *eds5* mutant (Nawrath et al. 2002). After export from plastids, isochorismate is conjugated to glutamate by AVRPPHB SUSCEPTIBLE3 (PBS3), after which isochorismate-9-glutamate decays into SA either spontaneously or facilitated by ENHANCED PSEUDOMONAS SUSCEPTIBLITY1 (EPS1) (Torrens-Spence et al. 2019) (Fig. 2). SA is directly perceived by NPR1 (Wu et al. 2012; Ding et al. 2018) and its homologs, NPR3 and NPR4 (Fu et al. 2012; Ding et al. 2018).

Nonexpresser of Pathogenesis-Related Genes1 (NPR1) in SA-Mediated Immunity

NPR1 is a *bona fide* SA receptor (Wu et al. 2012; Ding et al. 2018) (Fig. 2). The direct binding of SA induces a conformational change in the NPR1 protein, resulting in the release of the C-terminal transactivation domain from the inhibitory effect of the N-terminal BTB/POZ domain (Wu et al. 2012). Mutations in *NPR1* cause failures in the development of SA-triggered immunity and impairments in SA- and pathogen-induced *PR* gene expression (Cao et al. 1994, 1997; Delaney et al. 1995; Shah et al. 1997). In addition, SA-induced transcriptional reprogramming is impaired in *npr1* mutants. According to microarray-based and RNA sequencing-based analyses, *npr1* mutations affect the expression of 99% of the benzothiadiazole S-methylester (called BTH; a functional SA analog)-inducible genes (Wang et al. 2006) and 71% of the 2,6-dichloroisonicotinic acid (called INA; a functional SA analog)-inducible genes (Jin et al. 2018), respectively; therefore, NPR1 acts as the master transcriptional regulator in the transmission of SA signals to transcriptional reprogramming.

The mechanism by which NPR1 regulates SA-mediated changes in gene expression has been extensively studied. An SA-triggered redox change induces NPR1 monomerization, and the monomeric NPR1 is translocated from the cytoplasm to activate the *PR* genes (Mou et al. 2003) by interacting with TGACG motif-binding transcription factors (TGAs) at the *PR* promoters (Zhang et al. 1999; Zhou et al. 2000; Després et al. 2003; Shearer et al. 2009) (Fig. 2). Based on cryo-electron microscopy and crystal structure analyses, it was recently proposed that the SA-induced structural change of NPR1 facilitates the recruitment of an unknown regulator for transcriptional activation (Kumar et al. 2022), suggesting that NPR1 might require other transcriptional activators for SA-induced gene expression. Indeed, a recent study demonstrated that NPR1 forms a transcriptional co-activator complex with CBP/p300-family histone acetyltransferases, HISTONE ACETYLTRANSFERASE OF THE CBP FAMILY1 (HAC1) and HAC5 (HAC1/5), as well as TGAs, and that the HAC-NPR1-TGA complex mediates histone H3 acetylation in the *PR1* promoter and its transcriptional activation upon SA signaling (Jin et al. 2018).

The SMALL UBIQUITIN-LIKE MODIFIER3 (SUMO3)-mediated sumoylation of NPR1 has been known to be a mechanism for NPR1 turnover (Saleh et al. 2015). This protein modification is induced by SA accumulation but inhibited by NPR1 phosphorylation at Ser55/Ser59 under normal conditions. The SA-induced NPR1 sumoylation promotes NPR1 phosphorylation at Ser11/Ser15, and the Ser11/Ser15 phosphorylation in turn enhances the NPR1 sumoylation and NPR1 activity. The SUMO3-mediated NPR1 sumoylation also facilitates proteasome-mediated NPR1 degradation; thus, it was proposed that the proteasome-mediated turnover of active NPR1 might facilitate NPR1-induced gene expression through a promoter-refreshing mechanism (Spoel et al. 2009).

NPR3 and NPR4 in SA-Mediated Immunity

The transcriptional co-activator role of NPR1 is suppressed by its homologs, NPR3 and NPR4 (NPR3/4). NPR3/4 act as SA receptors with affinities to SA that differ from that of NPR1, but also function as adaptors for the Cullin3 ubiquitin E3 ligase which mediates the turnover of NPR1 protein in an SA concentration–dependent manner (Fu et al. 2012). Moreover, functionally redundant NPR3/4 target several genes that are also SA-dependent NPR1 targets and repress their expression in the basal state (Ding et al. 2018). NPR3/4 also induces the transcriptional activation of the JA-responsive genes required for de novo JA biosynthesis during ETI-induced SA accumulation by directly mediating the degradation of JAZ repressors, resulting in enhanced ETI and HR that are associated with SA-triggered immunity (Liu et al. 2016).

Epigenetic Regulation of Plant Immunity

When attacked by pathogens, plants induce a massive transcriptional reprogramming to elicit an effective immune response. As eukaryotic DNA is organized into chromatin, changes in chromatin structure are prerequisites for massive transcriptional reprogramming. Chromatin structure is regulated by epigenetic mechanisms, including DNA methylation, histone modification, chromatin remodeling, and non-coding RNAs. These epigenetic mechanisms enable plants to differentially use the genetic information in their DNA and adapt to and survive various pathogen challenges.

Role of DNA Methylation

The methylation of the fifth position of the pyrimidine ring of cytosine (5-methylcytosine) is the most abundant type of DNA methylation in plants, animals, and yeast. Whereas DNA methylation in animals mostly occurs at CG dinucleotides, DNA methylation in plants is deposited in three different cytosine sequence contexts: CG, CHG, and CHH (where H is A, T, or C). The genome-wide DNA methylation level has an impact on pathogen resistance in plants. Both the *methyltransferase1* (*met1*) single and *domains rearranged methylase1* (*drm1*) *drm2 chromomethylase3* (*cmt3*) triple mutants, which have reduced genome-wide CG and CHG/CHH methylation levels, respectively, were more resistant to a bacterial pathogen known to induce SA-mediated immunity (Dowen et al. 2012). In addition, several mutants defective in the RNA-directed DNA methylation pathway, including *nuclear rna polymerase e1* (*nrpe1*), *nuclear RNA polymerase d2* (*nrpd2*), *rna-dependent rna polymerase2* (*rdr2*), *defective in rna-dependent dna methylation1* (*drd1*), *argonaute4* (*ago4*), and the double *drm1 drm2* mutant, were more susceptible to necrotrophic pathogens known to induce JA-mediated immunity (López et al. 2011). Some mutants defective in DNA methylation, including *decreased dna methylation1* (*ddm1*), *nrpe1*, *drd1*, and *cmt3*, displayed enhanced resistance to a biotrophic pathogen, while exhibiting diminished resistance to a necrotrophic pathogen (López Sánchez et al. 2016). Contrasting phenotypes to the above were observed in the *repressor of silencing1* (*ros1*) mutant, which has genome-wide hyper DNA methylation (López Sánchez et al. 2016).

DNA methylation is dynamically altered in response to infection by virulent and avirulent pathogens, and the SA content changes with a context-specific pattern (Dowen et al. 2012). More recently, it was reported that rapid DNA demethylation, which is dependent on ROS1,

DEMETER-LIKE PROTEIN2 (DML2), and DML3, is induced by flg22 and the *ros1 dml2 dml3* triple mutant exhibits compromised flg22-triggered immunity (Huang et al. 2022). A pathogen attack induced DNA hypomethylation at centromeric and pericentromeric regions (Pavet et al. 2006), while the SA-induced hypomethylation at several transposable elements was positively correlated with the derepression of these transposable elements (Dowen et al. 2012). Thus, it has, been hypothesized that DNA methylation levels at transposons or other repetitive sequences near or within defense genes might affect the expression of these genes. The TNL-encoding gene *RESISTANCE METHYLATED GENE1* (*RMG1*) contains two helitron-related repeats in its promoter. Consistent with this hypothesis, the DNA methylation level at the repeat proximal to the transcriptional start site was increased in all cytosine contexts, and both the basal and flg22-induced transcript levels of *RMG1* were compromised in a *ros1* mutant (Yu et al. 2013b).

Role of Histone Modifications

Eukaryotic DNA is wrapped around a histone octamer, forming a nucleosome, which is the basal repeating unit of chromatin. A histone octamer consists of two copies of the histone proteins H2A, H2B, H3, and H4. In addition, the histone H1 protein acts as a linker, and this linker activity is essential for higher-order chromatin organization. The N-terminal tail of each nucleosome histone protein protrudes from the nucleosome core, enabling histone modifiers easy access to the histone tails and post-translational modifications. The covalent modification of histones can affect the chromatin structure by altering the electric charge and structure of the histone tails. In addition, each covalent histone modification catalyzed by specific 'writers' can provide a binding platform for modification-specific 'readers', which eventually recruit 'effectors' that affect chromatin structure and transcription. Histone modifications therefore influence plant immunity mainly through regulating transcriptional output. Now, there is considerable evidence that numerous defense genes are regulated by histone modifications as written below.

The acetylation of lysine residues within histones is regulated by the opposing activities of histone acetyltransferases (HATs) and histone deacetylases (HDACs). Several HATs and HDACs have roles in the regulation of plant immunity via their histone acetyltransferase and deacetylase activities, respectively. HISTONE ACETYLTRANSFERASE OF THE GNAT FAMILY1 (HAG1), a member of the GNAT-family HATs, regulates the histone H3 lysine 14 (H3K14) acetylation levels in the 5′ and 3′ ends of its target genes, which is associated with the downregulation of genes acting in SA-mediated immunity and the upregulation of genes acting as inhibitors of SA biosynthesis (Kim et al. 2020). Another GNAT-family member, ELONGATA3 (ELO3), also known as ELONGATOR PROTEIN3 (ELP3) or HAG3, is the catalytic subunit of an elongator complex. The HAT activity of ELO3 was found to be required for SA-mediated immunity (Defraia et al. 2013); however, it remains unclear whether ELO3 directly targets and acetylates histones at SA-responsive gene loci to induce their transcription.

Both *HAC1* and *HAC5* (*HAC1/5*), members of the CBP/p300-family HATs, have positive roles in SA-mediated immunity with functional redundancy and *HAC1* dominance. Dozens of SA-responsive genes, including *PR* genes, were induced in the presence of SA through HAC1/5-dependent histone H3 acetylation, and a subset of those genes were co-regulated by NPR1 (Jin et al. 2018). Furthermore, to induce the expression of the JA-responsive genes, HAC1 acetylated H3K9 at their promoters by forming a complex with MEDIATOR SUBUNIT25 (MED25) and MYC2 (An et al. 2017). Notably, HAC1 interacted with NPR1 and MYC2, which play essential roles in SA- and JA-mediated immunity, respectively, suggesting that HAC1 might be a central regulator of multiple immunity pathways in Arabidopsis.

HISTONE DEACETYLASE19 (HDA19) and HDA6, members of the REDUCED POTASSIUM DEFICIENCY3 (RPD3)/HISTONE DEACETYLASE1 (HDA1) family, function as positive regulators in JA-mediated immunity (Zhou et al. 2005; Wu et al. 2008), but as negative regulators in SA-mediated immunity (Choi et al. 2012; Wang et al. 2017). In the regulation of JA-responsive genes, the opposing activities of HDA6 and HAG1 jointly maintain the acetylation homeostasis of a co-repressor, TOPLESS (TPL) (An et al. 2022). The *HDA6* transcript level is increased in response to JA, thus facilitating TPL deacetylation which suppresses the TPL-dependent repression of JA-induced genes. This case therefore indicates that antagonistic activities of histone modifiers control the expression of defense genes through the modification of a non-histone protein.

In addition to HATs and HDACs, several histone methyltransferases and demethylases are known to be involved in the regulation of defense genes. A histone methyltransferase ARABIDOPSIS HOMOLOG OF TRITHORAX1 (ATX1) is targeted to *WRKY70* in response to bacterial pathogens and deposits H3K4 trimethylation (H3K4me3) to activate the expression of the target gene (Alvarez-Venegas et al. 2007). Another histone methyltransferase, SET DOMAIN GROUP8 (SDG8), affects both SA- and JA/ET-mediated immunity by regulating the H3K36me3-associated transcription of defense genes; for example, the H3K36me3-associated transcription of a gene encoding an RPS4-like R protein is dependent on SDG8 (Palma et al. 2010). SDG8 also positively regulates both H3K36me3 enrichment and the transcription of SA- or JA/ET-responsive marker genes upon pathogen infection or exogenous hormone treatment

(Berr et al. 2010; Zhang et al. 2020). An H3K4 methyltransferase, SDG25, plays a positive role in the regulation of genes encoding TNLs (Xia et al. 2013). Mutations in the *JUMONJI DOMAIN-CONTAINING PROTEIN14* (*JMJ14*) or *JMJ27* histone demethylase gene cause a reduced disease resistance to a bacterial pathogen associated with SA-mediated immunity (Dutta et al. 2017; Li et al. 2020).

Role of Chromatin-Remodeling Complexes (CRCs)

Chromatin-remodeling complexes (CRCs) are large and multi-component complexes that affect the position and/or composition of the nucleosomes. CRCs contain an ATPase/helicase subunit of the SWITCHING DEFECTIVE2/SUCROSE NON-FERMENTING2 (SWI2/SNF2) family, and energy derived from ATP hydrolysis allows the CRCs to modify nucleosomes. The SWI2/SNF2-family CRCs are evolutionarily conserved and categorized into four subfamilies: SWI/SNF, IMITATION SWITCH (ISWI), CHROMODOMAIN HELICASE DNA-BINDING (CHD), and INOSITOL REQUIRING 80 (INO80). In comparison with other epigenetic mechanisms, little is known about the role of CRCs in plant immunity.

The SWI2/SNF2-RELATED1 (SWR1) complex belonging to the INO subfamily catalyzes the replacement of canonical H2A with the H2A.Z variant. PHOTOPERIOD-INDEPENDENT EARLY FLOWERING1 (PIE1) is the catalytic subunit of the SWR1 complex. Mutations in both *PIE1* and the genes encoding the subunits of the SWR1 complex and H2A.Z affects plant immunity. In the *pie1*, *sef*, and *hta9 hta11* mutants, the basal transcript levels of SA-responsive genes and basal resistance to biotrophic pathogens were increased (March-Diaz et al. 2008); however, by contrast, a more recent study showed that basal resistance to biotrophic pathogens was reduced in the mutants of genes encoding the SWR1 complex, including *pie1*, *swc6*, and *hta9 hta11* (Berriri et al. 2016). The same study also reported that ETI and JA/ET-mediated immunity were compromised in the *pie1* and *swc6* mutants (Berriri et al. 2016). A SWI/SNF subfamily member, SPLAYED (SYD), is required for the resistance against necrotrophic but not biotrophic pathogens, and targets several marker genes of JA/ET-mediated immunity to induce their transcription (Walley et al. 2008).

Role of Long Noncoding RNAs (lncRNAs)

LncRNAs are transcripts with more than 200 nucleotides and no protein-coding capacity. LncRNAs are pervasively transcribed from various genomic regions, including intergenic sequences, enhancers, introns, and some regions of protein-coding sequences in either sense or antisense orientations. This heterogeneity makes lncRNAs versatile regulators that are involved in diverse biological processes using a variety of molecular mechanisms. LncRNAs may regulate genes in *cis* and/or *trans*; *cis*-acting lncRNAs regulate the expression of genes located at or near their own loci of transcription, while *trans*-acting lncRNAs regulate the expression of genes at distant loci. LncRNAs may act as scaffolds for the assembly of RNA–protein complexes or sometimes recruit epigenetic or transcriptional factors to specific loci through their sequence complementarity to DNA or RNA. Recently, lncRNAs have emerged as important regulators of plant immunity.

The *ELF18-INDUCED LONG NONCODING RNA1* (*ELENA1*) recruits MED19a to the *PR1* promoter and induces its expression upon PAMP treatment, thereby acting as a positive regulator in PAMP-triggered SA-induced immunity (Seo et al. 2017). More recently, ELENA1 was reported to evict FIBRILLARIN2 (FIB2) from MED19a on the *PR1* promoter, resulting in *PR1* derepression (Seo et al. 2019).

A lncRNA, *SALICYLIC ACID BIOGENESIS CONTROLLER1* (*SABC1*), balances immunity and growth by regulating SA biosynthesis (Liu et al. 2022). Under normal conditions, *SABC1* suppresses immunity and promotes growth by recruiting POLYCOMB REPRESSIVE COMPLEX2 (PRC2) to *NAC3* to repress its transcription via H3K27me3 deposition; however, upon pathogen infection, the transcript level of *SABC1* in decreased, resulting in the transcriptional activation of *NAC3* and the subsequent derepression of immunity and growth inhibition.

Concluding Remarks

The co-evolutionary arms race between plants and phytopathogens has resulted in various types of virulence factors in phytopathogens and pathogen-receptors in plants. Research over the past few decades has revealed that plants and phytopathogens use a variety of strategies for successful pathogen recognition and enhanced virulence, respectively; however, our understanding of the underlying molecular mechanisms is limited to only a few plant receptors and pathogen effectors (Kourelis and van der Hoorn 2018; Xin et al. 2018). Recent studies that have described the existence of intricate cross-talks between PTI and ETI further show the complexity of the plant immune responses against pathogens (Ngou et al. 2021; Pruitt et al. 2021; Yuan et al. 2021). Although PTI and ETI share various downstream events, the mechanisms by which ETI activates the more rapid and robust immune responses than PTI and the reason why HR is specific to ETI signaling should be answered in greater detail.

Crosstalks in plant immunity are also found among the phytohormone-mediated signaling pathways. It was proposed that the JA- and ET-mediated signaling pathways are

synergistic, whereas the SA- and JA/ET-mediated signaling pathways are antagonistic to each other (Li et al. 2019); however, evidence from recent studies has indicated that the SA- and JA/ET-mediated signaling pathways are not necessarily antagonistic (De vos et al. 2006; Mur et al. 2006; Liu et al. 2016; Mine et al. 2017). Considering that the SA- and JA/ET-triggered immune responses are responsible for defense against different types of pathogens, the activation of one of the immunity pathways by a type of pathogen may make host plants vulnerable to other types of pathogens which trigger the other immunity pathway. To avoid this risk, plants might balance and orchestrate various signaling pathways through cross-talks to achieve an optimal immunity to defend against potential attacks as well as responding to immediate attacks.

Unlike animals, plants do not have permanently differentiated immune cells. However, plants can induce immune-equipped cells upon pathogen attack. Based on this nature of plant immunity, which is operated through cell-fate transitions from normal to immune-equipped cells, massive transcriptional reprogramming is necessary and fundamental in plant immunity. Therefore, epigenetic mechanisms and regulators that enable transcriptional reprogramming are to be important in plant immunity also. However, given that epigenetic regulators usually have multiple and broad targets and participate in various biological processes, pleiotropic effects from the misexpression of epigenetic regulators are to be expected and considered when the role of epigenetic components in immunity is assessed. For this reason, the molecular mechanisms as well as phenotypic effects of the epigenetic components must be elucidated in detail. As one epigenetic component-related aspect, it would be of interest to identify new epigenetic partners targeting the master transcriptional regulators of plant immunity, including NPR1, MYC2, and EIN3/EIL1 involved in SA-, JA- and ET-triggered immunity, respectively, and reveal their underlying regulatory molecular mechanisms.

Studies of systemic immunity in plants are of great value due to their major application potentials for enhancing immune response. Despite extensive studies to identify the mobile signals that establish systemic immunity, *bona fide* mobile signals are yet to be confirmed. In addition to identifying the mobile signals, future studies on systemic immunity should address how the mobile signals are generated in local tissues and perceived in systemic tissues. Systemic immunity enables plants to prime an enhanced resistance against broad-spectrum pathogens, and is likely to occur at the physiological, metabolic, and transcriptional levels. Epigenetic mechanisms, including DNA methylations and histone modifications, may provide mechanisms for defense priming at the transcriptional level. In this regard, it would be of interest to elucidate how an initial exposure to a pathogen is memorized at the transcriptional level through epigenetic mechanisms.

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Declarations

Conflict of Interest The authors declare no conflict of interest.

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