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REVIEW

Plant Innate Immunity: Crosstalk of Signaling Pathways

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Abstract—The innate immunity of plants is a dynamic, multilevel system traditionally divided into pattern-triggered immunity (PTI) and effector-triggered immunity (ETI). Despite being activated by different types of receptors localized in different cell compartments, PTI and ETI are currently considered interdependent components of a single defense system. This view suggests that, due to various positive interactions between these two pathways, the innate immunity of plants is more than the sum of PTI and ETI. Available data indicate that PTI and ETI enhance each other synergistically, increasing the concentration of signaling molecules, such as components of kinase cascades, reactive oxygen species, calcium ions, and phytohormones. This leads to the activation of defense genes, providing a local response to pathogens and the development of systemic plant resistance.

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INTRODUCTION

Plants are exposed to attacks by pathogenic bacteria, fungi, and viruses, for which a plant organism serves as a natural habitat and a source of nutrients required for growth. As a result of the long-term coevolution of plants and phytopathogens, sophisticated molecular interaction mechanisms have emerged that enable plants to mount defense responses against infection while allowing pathogens to overcome such defenses [1]. In particular, plant evolution has led to the emergence of innate immunity, which is based on defense response genes that encode proteins capable of detecting attacks by pathogens and activating nonspecific protective responses, both local and systemic [2]. In addition, plants possess an adaptive immune system based on RNA silencing that provides sequence-specific protection against certain pathogens, predominantly viruses [3].

Traditionally, plant innate immunity has been considered as a two-layered system. The first layer, known as pattern-triggered immunity (PTI), is activated upon recognition of conserved pathogen-associated molecular patterns (PAMPs) or damage-associated molecular patterns (DAMPs) by pattern recognition receptors (PRRs) on the cell surface. To suppress PTI, many pathogens produce effector proteins. In response, plants activate a second, typically stronger immune response known as effector-triggered immunity (ETI), which involves the recognition of pathogen effectors by specific cytoplasmic receptors [4].

PTI and ETI had been initially considered as two distinct branches of plant innate immunity, a concept portrayed in the well-known “zigzag model” describing the development of plant immune responses [1]. However, subsequent studies have revealed extensive interconnections between PTI and ETI. Although these responses are mediated by different classes of receptors localized in distinct cellular compartments, both pathways converge on a common set of downstream

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defense responses, such as activation of mitogen-activated protein kinase (MAPK) cascades, calcium influx, production of reactive oxygen species (ROS), synthesis of phytohormones, and transcriptional reprogramming. In recent years, substantial progress has been made in understanding how PTI and ETI interact to establish effective immunity. Increasing evidence indicates that these two pathways are tightly interconnected and, in some cases, functionally inseparable across multiple levels of immune response, ranging from pathogen recognition to the activation of defense gene expression [1].

In this review, we summarize current insights into the mechanisms underlying PTI and ETI, as well as diverse modes of interaction between these components of plant innate immunity. We also discuss the development of local and systemic resistance to pathogens that arises upon activation of PTI and ETI.

PTI AND ETI: RECEPTORS AND CORECEPTORS

The classical distinction between PTI and ETI is largely based on the types of receptors involved in pathogen recognition. PTI is activated by PRRs located at the cell surface, which recognize PAMPs. Depending on the pathogen type, PAMPs may be further classified as virus-associated molecular patterns (VAMPs) in the case of viral infection or microbe-associated molecular patterns (MAMPs) in the case of bacterial or fungal infection. There are also damage-associated molecular patterns (DAMPs), which are plant-derived molecules, such as fragments of cell wall components generated during pathogen attack, that are capable of activating immune responses [1, 5]. PRRs contain a transmembrane α-helical domain and a variable extracellular domain (ECD) that mediates recognition of diverse PAMPs depending on the type of ECD. Some PRRs also possess a cytoplasmic kinase domain and are therefore classified as receptor-like kinases (RLKs), whereas PRRs lacking a structured cytoplasmic domain are referred to as receptor-like proteins (RLPs) [6, 7]. Based on the structure of their ECDs, PRRs can be divided into several classes. The largest group comprises receptors containing leucine-rich repeat (LRR) domains, while other classes include receptors with lysine motifs (LysM) and lectin, wall-associated kinase (WAK), S-locus, malectin-like, proline-rich, and cysteine-rich repeat domains [8]. Upon interaction with a pathogen, RLKs and RLPs recognize PAMPs via their extracellular domains, undergo conformational changes, and dynamically associate with their coreceptors and receptor-like cytoplasmic kinases (RLCKs), forming signaling complexes that initiate intracellular signaling cascades (Fig. 1). RLCKs lack extracellular and transmembrane domains, playing a central role in transducing signals from the cell surface to intracellular pathways that lead to pathogen resistance [7, 9].

In general, PTI-associated immune responses proceed as follows: activation of PRRs and their coreceptors upon PAMP recognition leads to phosphorylation of RLCKs, which subsequently phosphorylate downstream signaling components, thereby initiating intracellular signal transduction through activation of MAPK cascades, ion channels, and ROS production. These events result in large-scale transcriptional reprogramming and the activation of defense responses, including cell wall reinforcement, synthesis of antimicrobial compounds such as phytoalexins and phytoncides, and production of phytohormones that coordinate local and systemic responses, ultimately conferring resistance (Fig. 1) [8].

PRR coreceptors are most commonly represented by LRR-RLK receptors of the SERK family (SERK1-5, somatic embryogenesis receptor kinases) and by members of the NIK (NSP-interacting kinase) family (NIK1-3) [5, 7]. For example, recognition of bacterial flagellin or its conserved peptide fragment flg22 by the receptor FLS2 (flagellin sensing 2) in *Arabidopsis thaliana* results in rapid phosphorylation of the receptor kinase domain and its dimerization with the coreceptor SERK3, also known as BAK1 (BRI1-associated kinase 1). This complex subsequently interacts with the RLCK BIK1 (botrytis-induced kinase 1) [10]. Conformational changes lead to the release of phosphorylated BIK1 from the complex for activation of downstream signaling components [11].

Coreceptors of the NIK family have been shown to play a role in antiviral immunity. A mechanistic model of antiviral signaling via NIK1 suggests that viral PAMPs are recognized by yet unidentified PRRs, leading to phosphorylation and oligomerization of NIK1. Activated NIK1 mediates phosphorylation of ribosomal protein L10 (RPL10) at Ser104, thus promoting its translocation into the nucleus, resulting in global translational repression [12, 13]. Notably, in addition to its positive role in antiviral responses, NIK1 also participates in antibacterial immunity, where it functions as a negative regulator due to its constitutive association with FLS2 and BAK1. In *nik1*-knockout plants, flg22-induced activation of PTI is enhanced, including increased MAPK activity, elevated levels of ROS and salicylic acid (SA), and increased expression of the PR1 gene and other PTI components such as WRKY30, FRK1, and PP2C. Such effects can be explained by the constitutive interaction of NIK1 with FLS2 and BAK1. These interactions likely prevent autoimmune response in the absence of infection. Upon flg22 treatment, activation of FLS2 leads to phosphorylation of NIK1 at Thr474 by BAK1, thereby triggering antiviral signaling through

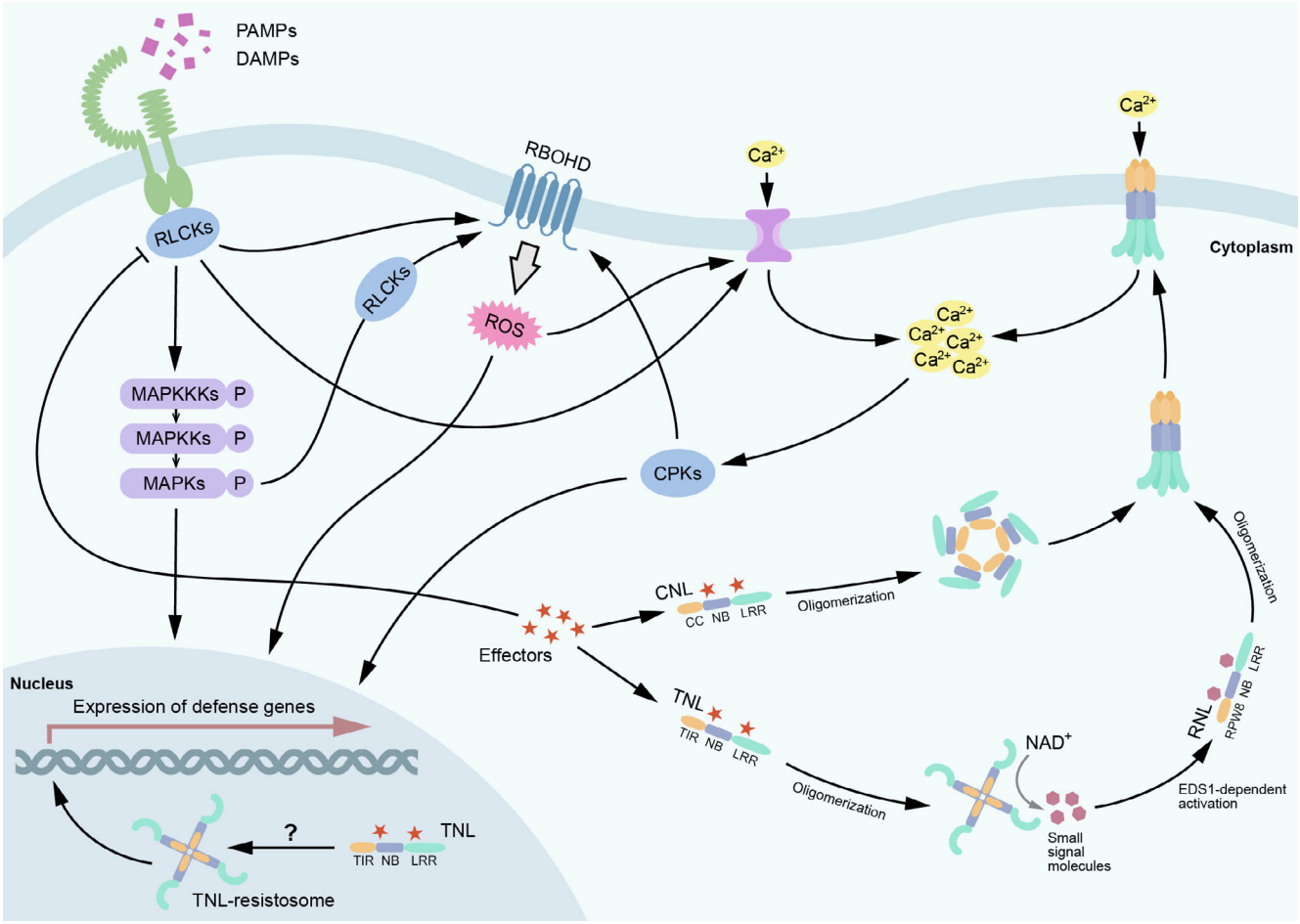

Fig. 1. Schematic representation of signaling pathways underlying PTI and ETI in plants. Recognition of PAMPs and DAMPs by cell-surface PRRs induces conformational changes in the receptors, their association with coreceptors, and phosphorylation of RLCKs. Activated RLCKs initiate multiple intracellular signaling cascades, including activation of MAPK cascades, phosphorylation of RBOHD leading to ROS burst, and activation of classical ion channels that mediate Ca^{2+} influx and downstream signaling via CPKs. Pathogen-derived effectors that suppress PTI are recognized by cytoplasmic NLR (NBS-LRR) receptors. Sensor NLRs (CNLs and TNLs) are responsible for effector recognition, whereas helper NLRs (RNLs) are activated downstream of sensor NLR signaling. NLR activation results in their oligomerization, resulting in formation of resistosomes. CNL- and RNL-resistosomes integrate into the plasma membrane and function as Ca^{2+} channels, promoting a robust calcium influx. TNL (TIR-NLR) resistosomes exhibit NADase activity, generating secondary signaling molecules that, through interaction with EDS1, lead to the activation of RNLs. The increase in cytosolic Ca^{2+} concentration that results from resistosome functioning activates CPKs, which then phosphorylate TFs and multiple signaling components, including RBOHD, thereby amplifying ROS production. ROS, in turn, activate Ca^{2+} channels, forming a positive feedback loop. Collectively, active Ca^{2+}, MAPK, and ROS signaling pathways lead to large-scale transcriptional reprogramming and activation of defense gene expression. Some CNLs and TNLs may also function directly in the nucleus; however, whether this occurs in the form of resistosomes remains to be determined.

the NIK1-mediated RPL10 phosphorylation and repression of translation-associated genes. Therefore, flg22 treatment can promote antiviral resistance in a NIK1-dependent manner [13]. Thus, NIK1 represents a unique example of a receptor kinase involved in autoimmune regulation, as well as in antiviral and antibacterial immunity.

Coreceptor kinases, such as SERK and NIK, are often targets of pathogen effectors [14]. Inhibition of these kinases suppresses PTI; however, in response to such effectors, plants activate the second layer of innate immunity – ETI. ETI receptors, which recognize pathogen effectors, are cytoplasmic proteins that contain LRR domains, nucleotide-binding sites (NBSs), and variable N-terminal domains. These receptors are collectively referred to as NBS-LRR proteins (NLRs). Based on the structure of their N-terminal domains, NLRs are classified into CNLs (coiled-coil domain), TNLs (Toll/interleukin-1 receptor domain), and RNLs (CC_{RPW8} domain) [15]. Functionally, NLRs can be divided into sensor NLRs (CNLs and TNLs), which recognize pathogen effectors, and helper NLRs (RNLs),

which do not directly recognize effectors, but rather transmit signals from sensor NLRs. Examples of helper NLRs include ADR1 (activated disease resistance 1), NRG1 (N requirement gene 1), and members of the NRC (NB-LRR protein required for HR-associated cell death) family. ADR1 can function with both CNL and TNL receptors, whereas NRG1 is associated with TNL-mediated signaling [16, 17]. NRC proteins are required for the function of sensor CNLs in the Solanaceae family [18].

Several models have been proposed to describe effector recognition by NLRs. In the direct recognition model, the NLR binds the effector via its LRR domain. In the guard model, NLRs detect modifications of host target proteins ("guardees") induced by pathogen effectors. In the decoy model, plants have evolved to express proteins that mimic genuine effector targets but lack their key immune functions. Modification of these decoys by effectors is recognized by NLRs, triggering NLR activation. Finally, in the integrated decoy model, decoy domains are incorporated directly into NLR proteins (e.g., HMA, WRKY, BED, and NOI domains); such domains are present in up to 10% of all NLRs [19-21].

While the C-terminal LRR domain is directly involved in the effector recognition, the NBS domain acts as a molecular switch that binds adenosine diphosphate (ADP) in the passive state and adenosine triphosphate (ATP) in the active state. Upon ligand recognition, the exchange of ADP for ATP in the NBS domain triggers conformational changes that lead to the formation of NLR oligomers called resistosomes. Pentameric and hexameric resistosomes formed by CNLs and RNLs can integrate into the plasma membrane and function as Ca^{2+}-permeable ion channels, whereas tetrameric TNL resistosomes function as holoenzymes bearing NADase activity and generate secondary signaling molecules derived from NAD^+ that activate RNL proteins (Fig. 1) [22, 23]. Proper functioning of the TNL/RNL signaling module also requires intermediate complexes with lipase-like proteins of the EDS1 (enhanced disease susceptibility 1) family [24]. Binding of TNL resistosome-derived signaling molecules to EDS1-PAD4 or EDS1-SAG101 heterodimers induces conformational changes in PAD4 (phytoalexin-deficient 4) and SAG101, promoting interaction with RNL proteins and their activation. The activated RNLs in turn undergo conformational rearrangements exposing N-terminal α-helices; this leads to dissociation of EDS1 and RNL oligomerization, resulting in the formation of membrane-associated resistosomes with ion channel activity [9, 25-28]. In addition, proper NLR function depends on HSP90 chaperones and RAR1/SGT1 co-chaperones, which form complexes with NLR proteins to ensure their correct folding and functioning [15, 20].

In general, upon effector recognition, activated NLRs oligomerize into resistosomes, triggering a strong Ca^{2+} influx into the cytoplasm. This activates nicotinamide adenine dinucleotide phosphate (NADPH) oxidases and ROS production, also triggering downstream hormonal signaling, transcriptional reprogramming, and programmed cell death associated with the hypersensitive response (HR), a hallmark of ETI. In some cases, activated NLRs can translocate to the nucleus and interact with transcription factors (TFs), directly modulating gene expression (Fig. 1). These processes ultimately lead to local and systemic immune responses, providing resistance at the site of infection and throughout the plant.

Despite the differences in initiation mechanisms, PTI and ETI activate overlapping and complementary reaction cascades, involve the same components of signaling pathways, and mutually regulate each other at various stages, including the receptor level. In fact, in recent years, increasing evidence suggest both the regulation of ETI by PTI receptors and the regulation of PTI by ETI receptors.

Many studies highlight the importance of PRR signaling for ETI. Since pathogens that induce ETI also contain PAMPs, it is challenging to dissect individual contributions of PTI and ETI. Notably, ETI-associated HR induced by *Pseudomonas syringae* effectors (AvrRpt2, AvrPphB, AvrRps4) is significantly reduced in PRR or coreceptor mutants, including fls2/efr, fls2/efr/cerk1, bak1-5/bkk1-1, and bak1-5/bkk1-1/cerk1. Furthermore, in Arabidopsis, PTI coreceptors BAK1 and BKK1 are required for restricting the infection by *Hyaloperonospora arabidopsidis*, whose effectors are recognized by TNL RPP2 and RPP4 [29]. Estradiol-dependent expression of bacterial effectors AvrRps4 and AvrRpt2 recognized by TNL RRS1/RPS4 and CNL RPS2, respectively, allowed to analyze ETI in the absence of PTI. Under these conditions, ETI responses were relatively weak and were not associated with ROS accumulation or HR induction [30, 31]. These findings indicate that PTI components are required for full ETI functionality and act to potentiate ETI-mediated pathogen restriction (Fig. 2) [31, 32]. Recent studies have revealed even tighter interdependence between PTI and ETI. RLCKs, key components of PTI signaling, have been shown to constitutively inhibit NLR oligomerization through phosphorylation. Upon PTI activation, RLCKs dissociate from NLRs and are recruited to activated PRRs, relieving ETI inhibition. Thus, PTI induction can enhance ETI signaling [33].

It has recently been discovered that PTI can also suppress ETI. This phenomenon, termed pre-PTI-mediated ETI suppression (PES), involves attenuation of AvrRpt2-induced ETI that occurs after the prior induction of a weak PTI-response by flg22 in *Arabidopsis* [22]. In the *dde2*/*ein2*/*pad4*/*sid2* mutant defective

in multiple hormone signaling pathways [including jasmonic acid (JA), ethylene (ET), PAD4 and SA branches], AvrRpm1-induced HR is suppressed if plants are pretreated with PAMPs [34]. These findings suggest that PTI can regulate ETI either positively or negatively, with negative regulation potentially serving to limit the resource-consuming ETI when PTI alone is sufficient.

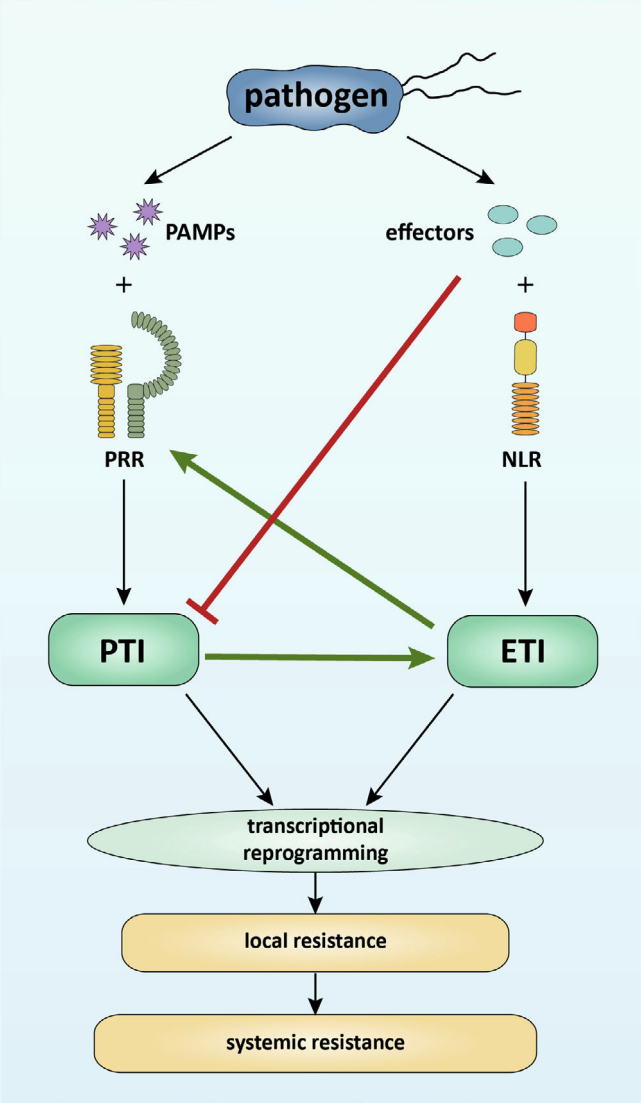

Fig. 2. Schematic representation of interconnections between PTI and ETI underlying plant innate defense responses. Upon pathogen attack, PAMPs are recognized by cell-surface PRRs, leading to the PTI activation. At the same time, pathogen-derived effectors produced to suppress PTI are recognized by intracellular NLRs, resulting in the ETI activation. PTI enhances ETI responses, whose components may remain weak in the absence of PTI. Conversely, ETI reinforces PTI by increasing the accumulation of key signaling components and amplifying downstream signaling. This positive regulatory feedback loop, in which ETI acts as an amplifier of PTI responses, leads to a large-scale transcriptional reprogramming and establishment of both local and systemic resistance.

A large amount of data also suggests that PTI can be regulated by ETI. Indeed, activation of NLRs rapidly increases both transcript and protein levels of key PTI components, including components of receptor complexes and downstream signaling factors. The activation of NLRs such as RPM1, RPS2, RPS5, RPS4, and RPP4 promotes accumulation of BAK1, SOBIR1, BIK1/PBL, RBOHD, MPK3, and MPK6 independently of PTI induction [29, 31, 32]. Moreover, activation of the PRR RLP23 requires ETI signaling components such as RNL ADR1, as well as EDS1 and PAD4 [35, 36].

As a result, ETI activation strengthens PTI responses (Fig. 2). For instance, ETI induced by recognition of the effector AvrRps4 by the receptors RRS1 and RPS4 enhances flg22-triggered ROS production and cell death [32]. Thus, ETI triggered by different effectors amplifies and reinforces PTI responses induced by PAMPs.

THE ROLE OF SIGNALING MOLECULES IN PTI–ETI INTERACTIONS

Signaling pathways underlying PTI and ETI share common hubs, including MAPK cascades, ROS, Ca^{2+} signaling, and phytohormone pathways, which are involved in the interaction between PTI and ETI [4, 9, 37].

MAPK. PTI is characterized by rapid activation of MAPK cascades following ligand recognition by receptors, whereas ETI induces a slower but stronger and more sustained MAPK activation [38]. While the components of MAPK cascades directly phosphorylated by RLCK kinases have been well characterized in PTI signaling, the mechanisms by which NLR signaling activates MAPK cascades remain unclear. Two major MAPK cascades are involved in the positive regulation of PTI. The first is initiated by MAP kinase kinase kinases (MAPKKKs), such as MAP3K1 (also known as MEKK1), leading to the activation of MAPK4 and MAPK11. The second cascade involves MAP3K3, MAP3K5, and YODA kinase, which activate MAPK3 and MAPK6 [39]. In addition to positive regulation, there is also negative regulation of PTI mediated by the MEKK1–MKK1/2–MPK4 cascade [40]. MAPK cascades can be activated either by transmembrane PRRs or by RLCKs, particularly those belonging to subfamilies VII and XII [9, 41]. Identifying specific factors responsible for MAPK activation during ETI remains challenging, as ETI amplifies PTI-derived signals and the two pathways act synergistically [9]. It has been shown that TNL receptors such as RRS1/RPS4 and RPP4 are unable to trigger MAPK activation in transgenic *Arabidopsis* expressing AvrRps4 or AvrRpp4 in the absence of PRR signaling. This suggests that TNL-associated MAPK phosphorylation is

mediated through the PTI pathway. In contrast, MAPK activation via CNL receptors, such as RPS2, RPS5, and RPM1 in *Arabidopsis*, appears to occur independently of PRR signaling [31, 32].

ROS. Both PTI- and ETI-mediated immune responses lead to increased intracellular ROS levels. ROS are typically generated as byproducts of aerobic metabolism, but they also have essential signaling functions in plant immunity. ROS production during immune responses is primarily mediated by NADPH oxidases (respiratory burst oxygen homologs, RBOHs) [9]. For example, during flg22-induced PTI, activation of the PRR-BAK1 complex leads to activation of RLCKs BIK1 and PBL1 (PBS1-like 1), which then phosphorylate RBOHD, resulting in a rapid ROS burst. Additional regulators, such as the MAP4K protein SIK1, further enhance this process by transphosphorylating BIK1, thereby stabilizing it and synergistically promoting RBOHD activation and ROS production [42]. Calcium signaling also plays a critical role in regulating RBOHD activity: calcium-dependent protein kinases (CPKs) phosphorylate RBOHD in response to changes in cytosolic Ca^{2+} levels [9]. Thus, ROS integrate of multiple signaling pathways, including calcium and MAPK signaling, and represent one of the key links between PRR- and NLR-mediated immune responses (Fig. 1).

In the context of ETI, ROS production is more intense and prolonged than in PTI and is associated with the induction of programmed cell death (PCD) [43]. In addition to post-translational modifications of RBOH proteins, ETI involves transcriptional activation of RBOH genes. ETI is further characterized by sustained MAPK activation and enhanced Ca^{2+} influx, which together establish a positive feedback loop that regulates ROS production. Therefore, during the ETI, ROS burst is sustained and amplified by various positive feedback loops. Recent studies have shown that the ETI-associated ROS burst requires PRR-mediated signaling and that this signaling is essential for reaching peak phosphorylation levels of RBOHD during ETI [31, 32]. These findings suggest that ROS accumulation during ETI is largely driven by PTI signaling through PRR/coreceptor complexes.

Calcium. Ca^{2+} ions serve as universal second messengers in eukaryotic cells. Upon pathogen recognition, the cytosolic Ca^{2+} concentration in plant rapidly increases, triggering multiple defense responses. PTI components, such as RLCKs (e.g., BIK1 and PBL1), regulate activation of plasma membrane Ca^{2+} channels, including cyclic nucleotide-gated channels (CNGCs), hyperosmolality-gated calcium-permeable channel (OSCAs), and glutamate receptor-like channels (GLRs) [9, 44]. Upon attack and subsequent infection by a pathogen, two peaks in the Ca^{2+} signal are typically observed that differ in duration and amplitude: a transient peak associated with PTI and a more sustained peak associated with ETI [9]. The sustained Ca^{2+} peak during ETI is mediated by NLR resistosomes, which integrate into the plasma membrane and function as Ca^{2+}-permeable channels, facilitating a robust influx of calcium into the cytosol. Interestingly, integration of resistosomes into the plasma membrane can compromise membrane integrity, potentially generating DAMPs that further stimulate PTI responses [45]. Apart from resistosomes, classical Ca^{2+} channels also appear to contribute to ETI responses. For example, *Arabidopsis* CNGC2 and CNGC4 play an important role in AvrRpt2/RPS2-mediated HR, whereas CNGC11 and CNGC12 contribute to ETI responses against *Hyaloperonospora parasitica* [41]. As described above, Ca^{2+} signaling is also closely linked to MAPK cascades and ROS production via activation of NADPH oxidases, ensuring synergetic regulation of these signaling networks [9].

Phytohormones. Phytohormonal signaling is an important component of plant innate immunity. Phytohormones are endogenous signaling molecules that regulate plant growth, development, and stress responses at very low concentrations [46]. Phytohormones are traditionally divided into growth-related hormones (auxins, gibberellins, cytokinins, brassinosteroids, and strigolactones) and stress-related hormones, including SA, JA, ET, and abscisic acid (ABA). However, recent studies indicate that the boundaries between these categories are being increasingly blurred, as growth hormones influence certain aspects of stress-dependent metabolism, whereas stress hormones participate in the regulation of some physiological processes [47]. A substantial body of evidence has been accumulated on the role of phytohormonal signaling in plant defense responses [48, 49]. Due to the broad scope of this topic, which cannot be fully covered in this review, only one aspect of phytohormonal signaling is discussed below, namely the role of phytohormones in the interaction between PTI and ETI.

SA plays a key role in phytohormonal signaling that occurs upon activation of innate immunity mechanisms [50], contributing to both local and systemic defense responses (see below) and mediating crosstalk between PTI and ETI. Activation of both PTI and ETI leads to increased SA levels in plants [51]. This increase is mediated by TFs such as SARD1 (systemic acquired resistance deficient 1) and CBP60g (calmodulin-binding protein 60g), which activate promoters of genes encoding proteins involved in SA biosynthesis, including ICS1 (isochorismate synthase 1), EDS5 (enhanced disease susceptibility 5), and PBS3 (avrPphB susceptible 3) [52, 53]. Members of the NPR (non-expressor of pathogenesis-related genes) family act as intracellular SA receptors; NPR1 functions

as a positive regulator of SA signaling, whereas NPR3 and NPR4 act as negative regulators [54, 55]. NPR1, NPR3, and NPR4 interact with TFs of the TGA (TGACG-binding) family, collectively regulating the transcription of SA-responsive genes involved in plant defense [53]. In addition to activating defense gene expression, SA plays an important role in regulating the PTI/ETI interactions. Treatment with exogenous SA increases the levels of receptors and signaling components involved in both PTI and ETI [56-58], whereas activation of both pathways is strongly impaired in *npr1*- and *npr4*-mutant plants [59]. Thus, SA establishes a positive feedback loop that amplifies immune responses and synchronizes the production of PTI- and ETI-associated proteins, thereby coordinating the activation of these two signaling pathways.

Along with the positive regulation, phytohormones can also negatively regulate PTI and ETI, for example, through antagonistic interactions between the SA and JA signaling pathways. SA signaling is typically activated in response to biotrophic pathogens, whereas JA and ET signaling pathways are associated with responses to necrotrophic pathogens [60]. SA signaling induces TFs that repress transcription of JA-responsive genes, while coronatine, a functional JA analog, induces NAC TFs that suppress the ICS1 gene promoter activity, thereby reducing SA levels [61]. Thus, activation of JA-dependent responses to necrotrophic pathogens can suppress PTI and ETI pathways, which require SA signaling for full activation.

REGULATION OF TRANSCRIPTION

Signaling cascades initiated by PTI and ETI lead to extensive reprogramming of the plant transcriptional landscape, thereby activating defense mechanisms. This reprogramming is coordinated in the nucleus through multiple regulatory mechanisms, including the activity of diverse TFs, chromatin remodeling complexes, and post-translational modifications of proteins [37, 62]. Notably, due to the close interconnection between PTI and ETI pathways, most transcriptional changes described for these responses largely overlap [63].

Key TF families involved in plant immune responses include WRKY, AP2/ERF, NAC, bZIP, bHLH, and CAMTA (calmodulin-binding transcription activator) [37, 62, 64]. The activity of these TFs is often regulated via post-translational modifications mediated by upstream immune signaling components. For example, WRKY33, WRKY28, CBP60g, CAMTA3, MYB51, and ORA59 (octadecanoid-responsive arabidopsis 59) are directly phosphorylated by both CPKs activated upon Ca^{2+} influx and MAPK cascades [37, 62]. Studies of WRKY33 demonstrate that different signaling pathways modulate its activity through distinct mechanisms: phosphorylation by CPKs enhances its DNA-binding activity, whereas phosphorylation via MAPK pathway increases its transactivation activity. Furthermore, SUMOylation of WRKY33 stabilizes its interaction with MAPKs, forming a sustained positive feedback loop [62, 65]. ORA59, a central regulator of the JA/ET signaling, alters its DNA-binding preferences depending on the phosphorylation status, thereby switching between activation of different groups of genes. In contrast, SA-dependent ubiquitination targets ORA59 for proteasomal degradation [62]. Thus, post-translational modifications enable flexible reconfiguration of transcriptional networks in response to changes in hormonal environment.

Activation of TFs following ligand recognition can occur not only through canonical signaling cascades but also via direct interaction of TFs with ETI receptors [37]. For instance, the barley CNL receptor MLA10 modulates the activity of WRKY and MYB TFs, thereby directly controlling defense gene expression in the nucleus. Similar mechanisms have been described for the *A. thaliana* nuclear TNL receptor pair RPS4/RRS1, which forms a complex with WRKY TFs and functions as a nuclear regulator of gene expression [37]. Another well-characterized TNL receptor encoded by the *N* resistance gene in some *Nicotiana* species interacts with SPL6 (squamosa promoter-binding protein-like 6) in the presence of the tobacco mosaic virus effector p50 [66]. The TNL receptor SNC1 (suppressor of NPR1-1, constitutive 1) has been shown to oligomerize in the nucleus, with its nuclear localization being essential for activation of its defense function [67]. Subsequent studies demonstrated that SNC1 interacts with nuclear co-repressors of the TOPLESS family and acts as an amplifier of immune signaling. This function depends on both its oligomerization and NADase activity, suggesting that SNC1 may function in the nucleus in a resistosome-like form (Fig. 1) [68].

Upon immune response, activated signaling components can induce post-translational modifications not only in TFs but also in RNA polymerase II (PolII). For example, MPK3/6, activated during PTI and ETI, phosphorylate CDKC1 and CDKC2 (cyclin-dependant kinase complex), which in turn phosphorylate the C-terminal domain (CTD) of PolII, enhancing its transcriptional activity. Conversely, the CTD phosphatase PHOSPHATASE-LIKE 3 (CPL3) can dephosphorylate the CTD, thereby acting as a negative regulator of transcription [62, 69].

The structure of chromatin plays a critical role in the regulation of transcription. Analyses using ATAC-seq, DNA-seq, and MNase-seq have shown that

activation of immune responses leads to remodeling of nucleosome architecture, thereby increasing accessibility of key regulatory DNA regions to TFs. Certain chromatin-remodeling ATPases, such as PKR2 and RAD54, promote immunity, whereas others, such as EDA16 and SWP73A, suppress it, indicating a complex relationship between chromatin remodeling and immune responses [62]. Phosphorylated WRKY33, in cooperation with the SWR1 chromatin remodeling complex, promotes accumulation of histone modifications such as H3K4me3 and facilitates replacement of H2A with H2A.Z in H2A-H2B dimers. These changes support sustained expression of genes involved in phytoalexin-mediated defense responses [70]. Transcriptional regulation can also occur through noncanonical functions of ARGONAUTE1 (AGO1). AGO1 associates with chromatin at specific genomic loci via interactions with specific small RNAs and the SWI/SNF (SWItch/sucrose non-fermentable) chromatin remodeling complex, particularly its subunits SWI3B and SWI3D, thereby promoting recruitment of PolII. These AGO1 functions may be regulated by phytohormones as well as flg22-induced PTI [71].

DNA methylation patterns are also altered during immune responses, and it is also known that changes in methylation status of specific genomic regions are critical for proper immune response. For example, DNA demethylases reduce methylation levels at the regulatory regions of flg22-induced defense genes, facilitating TF binding [72]. Transcription of PTI-induced genes can also be regulated by long noncoding RNAs (lncRNAs), such as ELENA1, whose production is induced by PAMPs including flg22 and elf18 (EF-Tu N-terminal peptide). ELENA1 enhances the expression of SA-responsive defense genes, including PR1 (pathogenesis-related 1) [73, 74].

Nuclear import and export of transcriptional machinery components represent a selective regulatory mechanism controlling the plant immune transcriptome. For instance, the nuclear pore complex component CPR5 (constitutive expressor of PR-5) plays a key role in regulating ETI-induced PCD. ETI signaling induces conformational changes in CPR5 that alter nuclear pore permeability, enabling nuclear import of defense-related proteins such as NPR1 and ABI5, thereby promoting transcriptional reprogramming [62]. Notably, NPR1 is considered a key transducer of ROS signaling into transcriptional reprogramming. Under normal conditions, NPR1 forms cytoplasmic oligomers stabilized by disulfide bonds. These bonds are disrupted upon immune activation and subsequent decrease in redox potential, leading to the formation of NPR1 monomers [62, 75]. According to the classical model, NPR1 monomers translocate to the nucleus and function as transcriptional coactivators together with TGA TFs [75]. However, more recent studies suggest that NPR1 functions as a dimer, interacting with two TGA3 dimers to form an enhanceosome in the nucleus [76]. The above-mentioned NPC protein CPR5 also performs additional regulatory functions during ETI. ETI signaling can disrupt CPR5 interaction with cyclin-dependent kinase inhibitors SIAMESE (SIM) and SIAMESE-RELATED1 (SMR1), leading to the activation of the E2F TF, promoting ETI-associated PCD [62]. Moreover, CPR5 exhibits RNA-binding activity and can associate with various transcripts, including that of AGO1, resulting in alternative splicing [77].

Immune responses can also lead to phosphorylation of splicing factors, which regulate defensive genes. For example, activation of MPK4 induces alternative splicing of pre-mRNAs encoding WRKY TFs, CPK kinases, and splicing regulators themselves [62]. During ETI, alternative splicing serves as an important mechanism for regulating NLR expression, thereby preventing autoimmune responses. In the absence of pathogen effectors, alternatively spliced isoforms of TNL mRNAs are degraded via the nonsense-mediated mRNA decay (NMD) pathway. However, in the presence of effectors, NMD is suppressed, allowing expression of fully functional versions of TNL genes [78]. Alternative splicing also regulates phytohormone signaling. For example, the functioning of JAZ genes, key regulators of JA signaling, is controlled by splicing factors PRP39a and PRP40, which are recruited by the mediator subunit MED25 [79].

PLANT RESISTANCE

The final stage of immune responses is the establishment of resistance to pathogens, which can be either local or systemic.

Local resistance: hypersensitive response and extreme resistance. HR represents a form of local resistance characterized by rapid cell death, which leads to the simultaneous elimination of the invading pathogen [15, 80, 81]. HR develops as a result of ETI; simultaneous co-activation of PTI significantly enhances the strength of HR; in some cases, HR development is impossible in the absence of PTI components [30, 32]. HR is preceded by a series of early events, including an increase in ROS and nitric oxide (NO) levels, as well as changes in cytosolic Ca^{2+} concentration. HR is also typically accompanied by lipid peroxidation, cell wall reinforcement, and transcriptional reprogramming [15].

Weak Ca^{2+}, MAPK, and ROS signals generated during PTI are typically insufficient to trigger HR-induced PCD; rather, their amplification via ETI is required, leading to more intense and prolonged signals that induce PCD. One of the key players

in this process is resistosome, which induces a robust Ca^{2+} influx. Enhanced MAPK signaling and the associated ROS production are also critical for PCD induction. ROS accumulation promotes SA biosynthesis, which in turn further enhances ROS production via two mechanisms: inhibition of the mitochondrial electron transport chain and reduction in the antioxidant enzyme activity. These processes establish a self-amplifying loop of SA and ROS accumulation, ultimately leading to cell death. However, SA can also act as a negative regulator of HR. This effect is thought to be related to its role in SAR, where tight control of local PCD is required for proper systemic response [15, 82, 83]. SA is not the only phytohormone involved in HR regulation. For example, ET can influence ETI-associated PCD. ET signaling components such as ETR1 and EIN2 have been shown to accelerate PCD progression, contributing to the expansion of ETI-induced necrotic lesions [84].

Thus, the HR is a type of immune response in which localized cell death serves as a key mechanism for limiting pathogen spread. This form of response illustrates a plant strategy in which the death of some cells is a price paid for maintaining the integrity of the organism and activating systemic resistance, highlighting the importance of the balance between localized cell death and systemic defense.

Another form of local resistance, described in the context of viral infection, is extreme resistance (ER), in which infected cells do not undergo cell death or phenotypic changes, while pathogen replication and spread are blocked at very early stages [85]. The molecular mechanisms underlying ER remain poorly understood. Like HR, ER is initiated by NLR receptors, and depending on virus load, the same NLR protein may trigger either HR or ER. Notably, both HR and ER can induce SAR, indicating shared features between these immune responses [86]. At this point, it remains unclear whether ER and HR are manifestations of a single type of resistance or fundamentally different types of resistance that are induced sequentially. In the case of potato virus X (PVX) infection, the key gene controlling ER in *Solanum stoloniferum* is *Rx1* (resistance to potato virus X 1). Recent studies indicate that the protein encoded by the *Rx1* locus mediates transcript-specific translational repression of viral RNA encoding the capsid protein, without affecting global cellular translation. In addition, nucleocytoplasmic transport of Rx1 is required for ER establishment [86, 87]. Thus, ER represents a unique form of local plant immunity characterized by asymptomatic yet highly effective suppression of viral infection.

Systemic resistance: SAR and ISR. Biotic stress not only induces the local primary response described above, but also initiates the development of systemic resistance, which is typically divided into systemic acquired resistance (SAR) and induced systemic resistance (ISR) [88, 89].

SAR develops as a consequence of a localized defense response, initially triggered by both PTI and ETI, and involves resistance to a broad spectrum of pathogens, which is observed in other, uninfected parts of the plant distant from the site of infection [90, 91]. SAR typically persists for 2-3 weeks but may last longer and, in some cases, can be transmitted to subsequent generations, a phenomenon referred to as transgenerational resistance [89]. SAR is characterized by elevated expression of defense-related genes, including those that encode PR proteins [90]. For SAR to be established, it is evident that the development of a local defense response must lead to the generation of a signal or signals that can be transported throughout the plant systemically and activate the expression of specific genes in distant parts of the plant [92]. In recent decades, significant efforts have been directed toward identifying the components necessary for generating such a signal and determining the nature of the signal itself.

One of the central regulators of SAR is SA. Partial or complete suppression of SA accumulation leads to attenuation or loss of SAR [90]. Early studies suggested that SA itself does not exhibit systemic mobility but plays a key role in inducing the SAR response, being synthesized de novo in healthy leaves, to which a mobile SAR signal is transmitted from pathogen-infected parts of the plant [93]. However, more recent findings indicate that SA may also be systemically transported, although its mobility alone is insufficient to induce SAR. In this model, SA likely acts cooperatively with other mobile signals [94, 95]. Nowadays it is known that one of the key mobile signals is methyl salicylate (MeSA), a derivative of SA [96]. In plants, SA and MeSA exist in a state of dynamic equilibrium. When SA levels rise in response to infection due to the activation of PTI and ETI, MeSA is synthesized. Conversely, after MeSA is transported systemically, it is hydrolyzed, leading to the accumulation of SA (Fig. 3). These processes are mediated by enzymes such as SA-methyltransferase (SAMT), SA-binding protein 2 (SABP2), and methylesterase (MES) [97, 88]. The requirement of SAMT and MES for SAR has been demonstrated in potato, Arabidopsis, and tobacco plants [88, 98]. Thus, systemic transport of MeSA, its conversion back to SA in distal tissues, and possibly the transport of SA itself and other SAR signals collectively lead to activation of SA-dependent gene expression in uninfected tissues, including genes encoding defense proteins. Notably, due to its volatile nature, MeSA can also mediate plant-to-plant communication, inducing pathogen resistance in neighboring plants [88]. Another class of volatile compounds

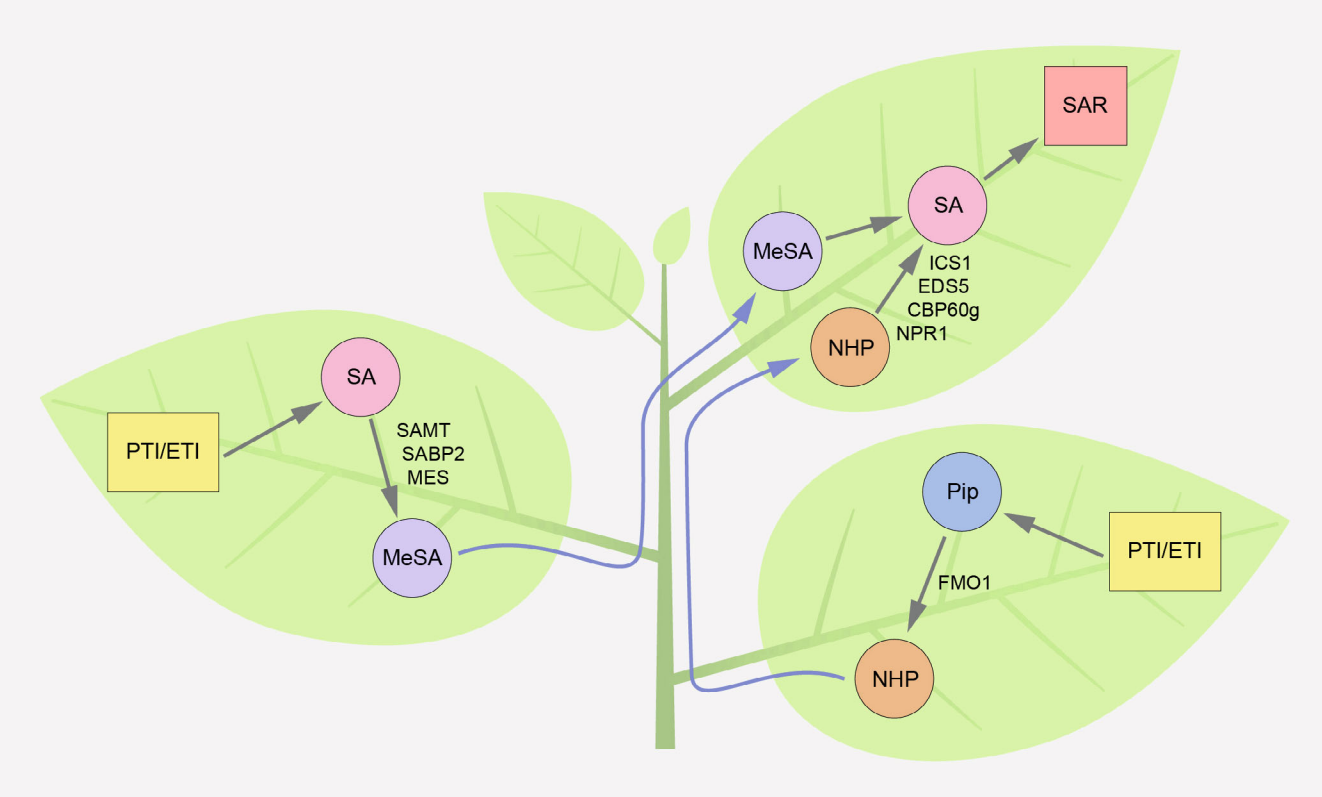

Fig. 3. Two major signaling pathways leading to SAR. Both pathways are activated by PTI and ETI. In the SA/MeSA-dependent pathway (left), pathogen infection of plant tissues triggers PTI/ETI, leading to the local accumulation of SA and subsequent formation of MeSA, which acts as a mobile signal that is transported to the upper leaves and induces SAR. The Pip/NHP-dependent pathway (right) involves the production of Pip upon PTI/ETI activation and its subsequent conversion into NHP, which functions as a mobile SAR signal. In upper leaves, MeSA is converted back to SA, whereas NHP activates expression of genes involved in SA biosynthesis and perception. Thus, signaling through both MeSA and NHP pathways leads to SA accumulation in distal tissues, ultimately triggering SAR. The NHP signal amplification pathway, involving AzA, G3P, NO and ROS, which may also act as systemic SAR signals, is not shown.

that have a similar function in plant-to-plant communication are monoterpenes, such as pinenes, whose biosynthesis is also induced during PTI and ETI [99, 100].

An important component of SAR activation is the non-proteinogenic amino acid Pip (pipecolic acid) [101]. During infection, Pip levels increase significantly in pathogen-infected leaves, and elevated Pip levels are also observed in systemic tissues. Furthermore, exogenous application of Pip results in SAR induction [102]. Pip functions through its biologically active derivative, N-hydroxypipecolic acid (NHP), which is synthesized from Pip by the enzyme FMO1 (flavin-dependent monooxygenase 1). In plants carrying mutations in the FMO1 gene, SAR development is strongly suppressed [101]. Accumulating evidence indicates that NHP is the key signaling molecule in the Pip-dependent SAR activation pathway. NHP is capable of systemic transport throughout the plant, and its exogenous application induces SAR, complements mutations in Pip biosynthesis genes, and activates expression of both these genes and FMO1 gene [91]. Thus, activation of Pip biosynthesis at infection sites leads to local accumulation of NHP and its systemic transport to distal tissues, where NHP promotes further Pip synthesis and its conversion into NHP (Fig. 3).

In addition to NHP, several other molecules have been identified as mobile SAR signals, including azelaic acid (AzA), glycerol-3-phosphate (G3P), NO, and ROS, the synthesis of which is activated by NHP [83, 101]. These components not only mediate long-distance signal transmission but also contribute to signal amplification, which occurs both at infection sites and in systemic leaves during SAR development [91].

Thus, two closely interconnected major signaling pathways mediate SAR: the SA-dependent pathway and the Pip/NHP-dependent pathway [92]. NHP activates transcription of a number of genes in the SA biosynthesis pathway, including *ICS1*, *EDS5*, and *CBP60g*, as well as genes encoding SA receptors such as NPR1 [101]. Thereby, NHP enhances SA signaling in two ways, leading to the activation of defense gene

expression and establishment of SAR in distant parts of the plant. While SA-mediated signaling represents the primary mechanism of Pip action, a minor SA-independent branch of Pip/NHP signaling that also contributes to SAR has been described [103].

In contrast to SAR, which is induced by pathogens, ISR is triggered by non-pathogenic, plant growth-promoting rhizobacteria and fungi. Nevertheless, ISR, like SAR, confers resistance to a broad spectrum of pathogens. For example, in cucumber plants, growth-promoting rhizobacteria have been shown to induce systemic resistance against bacterial, fungal, and viral pathogens, as well as nematodes [104]. ISR is not characterized by a full activation of defense responses, as occurs in SAR, but rather by a physiological state known as priming, in which plants develop a faster and stronger response to subsequent pathogen infection than unprimed plants [105]. Current evidence suggests that ISR initiation resembles that of SAR and involves recognition of microbial molecular patterns by plant receptors. However, in the case of SAR, this recognition leads to a full-scale immune response, whereas ISR results in a moderate activation. For instance, flagellin from the plant growth-promoting bacteria *Burkholderia phytofirmans* activates ISR in grapevine through the FLS2 receptor [106], whose activation in other contexts induces PTI and SAR. Notably, the immune response triggered by *B. phytofirmans* flagellin is weaker and less sustained than that induced by pathogenic flagellin [106]. Thus, activation of functionally identical receptors can lead to SAR during plant interactions with pathogens and to ISR in the case of interactions with bacteria and fungi that are beneficial to plant growth. Several mechanisms may underline this difference. Structural variations in microbial patterns may alter receptor activation, as demonstrated for *B. phytofirmans* flg22, which contains amino acid substitutions compared to flg22 of pathogens and induces weaker immune responses [106]. Additionally, it is known that low-molecular-weight compounds produced by pathogenic bacteria can inhibit PTI [107], and a similar mechanism is suggested to operate in beneficial fungi and bacteria as well [104]. Furthermore, ISR activation and the onset of priming state involve signaling via the JA/ET pathways [108, 109], which may antagonize SA signaling required for full activation of PTI (see above). Together, these observations suggest that beneficial microbes employ multiple strategies to limit the strength of plant immune responses, thereby facilitating mutually beneficial interactions with host plants.

In summary, systemic resistance in plants results from both local and systemic defense responses involving complex interactions between innate immunity pathways and long-distance signaling networks.

CONCLUSIONS

Recent studies demonstrate that ETI operates through components of PTI and amplifies PTI signaling, while PTI, in turn, synergistically enhances ETI signaling. As a result, the crosstalk between PTI and ETI leads to mutual potentiation of these pathways, ensuring effective plant innate immunity and the establishment of resistance to a wide range of pathogens.

Although the mutual dependence of these two pathways has long been recognized, its extent remains unclear. It appears that ETI is more dependent on PTI than *vice versa*. However, PTI alone is generally insufficient to confer robust resistance, which requires signal amplification by ETI. While ETI signaling is ultimately responsible for establishing resistance, it is PTI that can be considered as the main mechanism of defense against pathogens. To suppress PTI, pathogens deploy effector proteins, which in turn trigger ETI. Activation of ETI leads to increased accumulation of key PTI components and enhanced signaling, thereby counteracting effector-mediated suppression of PTI by pathogens. Within this model, ETI can be viewed as a PTI signal amplifier necessary for the development of effective resistance. Further investigation of the interdependence between PTI and ETI, as well as the molecular mechanisms underlying their interactions, will undoubtedly be a main focus of research in the field of plant innate immunity in the coming years.

In addition, further research is needed to better understand the mechanisms underlying the activation of PTI and ETI receptors, as well as other signaling components that mediate signal transduction and interactions required for effective functioning of plant innate immunity. A deeper understanding of the relationship between PTI and ETI will not only contribute to our knowledge of plant immunity in general, but may also facilitate the development of new strategies in crop genetics and breeding to enhance resistance to pathogens.

Abbreviations

ADR1	activated disease resistance 1
AGO	argonaute
AzA	azelaic acid
BAK1	brassinosteroid intensitive 1 (BRI1)-associated kinase
BIK1	botrytis-induced kinase 1
BKK1	BAK-like 1
CAMTA	calmodulin-binding transcription activator
CBP60g	calmodulin-binding protein 60G
CDKC	cyclin-dependent kinase complex
CNGC	cyclic nucleotide-gated ion channel

CNL	coiled-coil-NLR
CPK	calcium-dependent protein kinase
CPR5	constitutive expressor of PR-5
CTD	C-terminal domain
DAMP	damage-associated molecular pattern
ECD	extracellular domain
EDS1	enhanced disease susceptibility 1
ER	extreme resistance
ET	ethylene
ETI	effector-triggered immunity
FLS2	flagellin-sensitive 2
FMO1	flavin-dependent monooxygenase 1
G3P	glycerol 3-phosphate
GLR	glutamate receptor-like channel
HR	hypersensitive response
ICS1	isochorismate synthase 1
ISR	induced systemic resistance
JA	jasmonic acid
JAZ	jasmonate ZIM-domain
lncRNA	long non-coding RNA
LRR	leucine-rich repeat
LysM	lysin motif
MAMP	microbe-associated molecular pattern
MAPK (MPK)	mitogen-activated protein kinase
MeSA	methyl salycilate
NBS	nucleotide binding site
NHP	N-hydroxypipecolic acid
NIK1	NSP-interacting kinase
NLR	NBS-LRR
NMD	nonsense-mediated mRNA decay
NPR	nonexpressor of PR-genes
NRC	NB-LRR protein required for HR-associated cell death
NRG1	N requirement gene 1
NSP	nuclear shuttle protein
ORA59	octadecanoid-responsive arabidopsis 59
OSCA	hyperosmolality-gated calcium-permeable channel
PAD4	phytoalexin-deficient 4
PAMP	pathogen-associated molecular pattern
PBL1	PBS1-like 1
PBS	avrPphB susceptible
PCD	programmed cell death
PES	pre-PTI-mediated ETI suppression
Pip	pipecolic acid
PR	pathogenesis-related
PRR	pattern-recognition receptor
PTI	pattern-triggered immunity
PVX	potato virus X
RBOH	respiratory burst oxygen homologue
RLCK	receptor-like cytoplasmic kinase
RLK	receptor-like kinase
RLP	receptor-like protein
RNL	RPW8-NBS-LRR
ROS	reactive oxygen species
RPL10	ribosomal protein L10
RPS	resistance to *Pseudomonas syringae*
RPW8	resistance to powdery mildew 8
RRS	resistance to *Ralstonia solanacearum*
Rx1	resistance to Potato virus X 1
SA	salicylic acid
SAG101	senescence-associated gene 101
SAR	systemic acquired resistance
SARD1	systemic acquired resistance deficient 1
SERK	somatic embryogenesis receptor kinase
SNC	suppressor of NPR1-1 constitutive 1
SOBIR1	suppressor of BIR1
SPL6	squamosa promoter binding protein-like 6
SWI/SNF	SWItch/Sucrose non-fermentable
TF	transcription factor
TGA	TGACG-binding
TIR	toll-interleukin
TNL	TIR-NLR
VAMP	virus-associated molecular pattern
WAK	wall-associated kinase

Contributions

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Ethics approval and consent to participate

This work does not contain any studies involving human and animal subjects.

Conflict of interest

The authors declare that they have no conflicts of interest.

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