Plant Immune System Defense Against Type III Effectors Intrusion: Mechanisms of Recognition and Response to T3SS-Mediated Pathogenesis

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Abstract. PAMP-triggered immunity (PTI) and effector-triggered immunity (ETI) constitute the first and second lines of defense in the plant immune system. PTI is mediated by pattern recognition receptors (PRRs), which is located on the surface of a cell and typically elicits a relatively weak immune response. The second line of defense, ETI, often results in cell death and is characterized by a more intense and sustained response. Bacterial Type III Effectors (T3Es) can interfere with PRRs on the plant cell surface, thereby suppressing PTI. T3Es are proteins injected into host cells via the Type III Secretion System (T3SS). These effectors inhibit responses of the plant immune system by disrupting the transmission of defense signals during PTI. The T3SS, which structurally resembles a syringe, directly delivers the effectors into the host cells. The T3SS undergoes self-assembly and is subsequently activated under specific conditions, enabling the secretion and localization of T3Es to their target sites within the host cell. To counteract the threat posed by T3Es to plant health and growth, certain plants have evolved mechanisms to resist T3E interference. These plants utilize nucleotide-binding domain, leucine-rich repeat containing receptors (NLRs) and PRRs to recognize T3Es. Following recognition, multiple downstream pathways are activated and signals are released to suppress the spread of T3Es. This review outlines the mechanisms of how pathogenic bacteria inject T3Es into plants and summarizes the currently known strategies employed by the plant immune system to defend against T3Es.

Keywords: Plant immune system, Type III Effectors(T3Es), Type III Secretion System(T3SS), Defense signal

1 Introduction

The plants innate immune system consists of PAMP-triggered immunity (PTI) and effector-triggered immunity (ETI). PTI serves as the first line of defense in plant immunity and is mediated by pattern recognition receptors (PRRs) located on the surface of a cell. PRRs include receptor kinases and receptor-like proteins, which activate downstream signaling pathways upon binding to pathogen - associated molecular patterns

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(PAMPs). Several immune responses then be activated. PTI fight against most non-adaptive pathogens, thus its response is relatively weak and can be suppressed by effectors secreted from pathogens. ETI is the second line of defense, mediated by leucine-rich repeat containing receptors (NLRs) which is an intracellular nucleotide-binding domain. NLRs is able to identify pathogen-secreted effectors. NLRs trigger a more intense and sustained immune response. This would usually cause programmed cell death, thereby restricting pathogen spread[1].

The plant immune system is susceptible. Various bacteria, viruses, and their secreted substances can easily disrupt the plant immune system. Bacterial Type III Effectors (T3Es) can interfere with PRRs on the plant cell surface, thereby suppressing PTI.

The Type III Secretion System (T3SS) is a sophisticated molecular mechanism that enables bacteria to transport T3Es into the host cell cytoplasm. This system is crucial for the pathogenicity of various bacteria, such as Salmonella, by injecting effectors into host cells to suppress immune responses[2-3]. T3Es are a class of proteins injected into host cells by plant pathogenic bacteria via the T3SS. Upon activation, the T3SS recognizes and loads specific T3Es, using its needle-like structure to penetrate the plant cell wall and plasma membrane, directly delivering T3Es into the plant cytoplasm T3Es disrupt plant defense signaling and suppress overall plant immunity[4-6].

The interference of T3Es with the plant immune system poses a significant threat to the healthy growth of various plants, including Arabidopsis thaliana and rice (Oryza sativa). To counteract these threats, some plants have evolved mechanisms to resist T3E interference. This review discusses how pathogenic bacteria exploit the T3SS to disrupt host cells and summarizes current research progress on plant defense mechanisms against the T3SS.

2 Type III Secretion System(T3SS)

2.1 Structure of the T3SS

T3SS is a transmembrane "nano-syringe" capable of directly delivering T3Es into host cells.

The Basal Body is the central component of the T3SS, consisting of several kinds of proteins. For example, proteins SctC, SctD, and SctJ consist in the Basal Body of the Shigella T3SS[7]. The basal body spans both the inner and outer cell membranes of the bacterium, providing mechanical support for the needle structure and effector translocation. It primarily consists of an outer membrane circle, an inner membrane circle, and a neck region. These ring-like structures are connected by the "neck," forming a stable platform that facilitates the assembly of the needle tip complex[8].

The Needle Tip and Translocon is on the end of the structure. Their structure and function vary among different pathogens. The hydrophobic transporter can’t be stalled into the membrane of the host cell if there’s no Needle Tip and Translocon[9].

Chaperones regulate the secretion of T3Es through the T3SS. During effector translocation, chaperones enter the Needle Tip and Translocon from the cytoplasm, helping to maintain T3Es in a partially folded state, which enables them to pass through

T3SS[10]. Additionally, chaperones are involved in the sorting and secretion prioritization of T3Es.

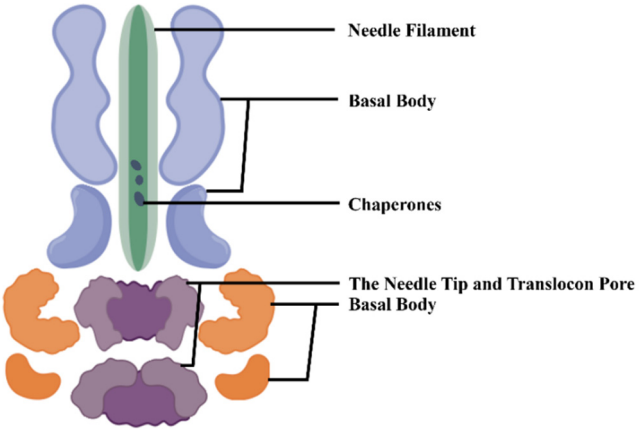

Fig. 1. The Structure of Type III Secretion Systems(Figure 1. is adapted from The Structure and Function of Type III Secretion Systems [Notti RQ et al., 2016, Microbiol Spectr, Volume Page 4])

The figure 1 illustrates a cross-sectional schematic of the T3SS structure(figure 1). The Basal Body, the core structure of the T3SS, consists of multiple proteins that span both the inner and outer membranes of the bacterium. It provides mechanical support for the needle-like structure and effector translocation, forming a stable assembly platform. The Needle Filament, an external extension of the T3SS, is a helical fiber that extends to the host cell surface, creating a channel for effector delivery. The Needle Tip and Translocon, are responsible for inserting hydrophobic transport proteins into the host membrane. Chaperones is crucial for maintaining the folded states of T3Es as they pass through the T3SS, ensuring their smooth translocation through the narrow channel. Additionally, chaperones participate in the sorting and secretion prioritization of T3Es.

2.2 Mechanisms of T3SS in Suppressing Plant Immunity

2.2.1 Assembly and Activation of the T3SS. Before the T3SS is activated, it undergoes a self-assembly process. Proteins are sequentially assembled into structures such as the basal body and needle filament, preparing the system for subsequent activation and effector secretion.

Once the T3SS is fully assembled, it is activated under specific conditions. The activation of the T3SS can be triggered by various factors. For instance, environmental stimuli can induce activation[11]. Additionally, some T3SSs are activated through

contact with host cells. For example, when Salmonella comes into contact with target cells, it stimulates the secretion of proteins such as SipB and SipC. These proteins transmit signals through the needle tip complex to the basal body or secretion apparatus, initiating the secretion process[12]. After the T3SS syringe structure is assembled and activated, transmembrane pore-forming proteins, such as SipB and SipC in Salmonella, utilize the needle complex as a formation platform for pores.

2.2.2 Secretion of T3Es. T3Es typically possess conserved N-terminal secretion signal sequences. Subsequently, T3Es enter the internal channel of the needle tip complex. The needle tip complex recognizes specific receptors or lipid components on the host cell membrane, binds to them, and inserts into the membrane. With the assistance of ATPases and chaperones, T3Es are released into the host cell in a polarized fashion [13].

2.2.3 Interference Mechanisms of T3Es. After entering the host cell, T3Es first target specific intracellular locations. Some effectors can enter the nucleus of the host cell, impacting how the plant genes are expressed directly. Others target specific signaling pathways in the cytoplasm, such as inhibiting programmed cell death (PCD) or activating the jasmonic acid (JA) pathway.

Once localized to their functional sites, T3Es disrupt host defense responses. For example, T3Es can inhibit PCD, as seen with AvrPtoB, which suppresses plant cell death responses. T3Es can also activate the JA pathway while suppressing the salicylic acid (SA) pathway, thereby weakening plant immune responses. Additionally, T3Es can inhibit cell wall remodeling, as demonstrated by AvrPto, which suppresses plant cell wall defense responses[14].

3 Recognition of T3Es by Plants

3.1 Recognition of T3Es by PRRs

PRRs (Pattern Recognition Receptors) are receptors located on the surface of plant cells that recognize PAMPs (Pathogen-Associated Molecular Patterns) or DAMPs (Damage-Associated Molecular Patterns), thereby triggering PTI (PAMP-Triggered Immunity). T3Es (Type III Effectors) can be indirectly recognized by PRRs. For example, certain T3Es modify target proteins within plant cells, leading to changes in their conformation or function, which are subsequently detected by PRRs.

PRRs typically do not directly recognize T3Es but instead trigger immune responses by sensing modifications to plant cellular components caused by T3Es[15]. For instance, AvrPtoB from Pseudomonas syringae suppresses PTI by inhibiting the enzymatic activity of PRRs such as FLS2 and EFR. In response, the R proteins contained in the plants will recognize and counteract the activity of these effectors. In Arabidopsis thaliana, NLRs (Nucleotide-Binding Leucine-Rich Repeat Receptors) such as RPM1 and RPS2 recognize T3Es by monitoring modifications to the RIN4 protein, which is targeted by T3Es[15].

Upon recognizing T3Es, The production of ROS, expression of defense genes and so on are stimulated by Ca2+ influx and elevated NADPH oxidase, RBOHD, encoded by RBOH genes. In this process, PRRs initially undergo ligand-dependent association with co-receptor kinases and intracellular protein kinases to initiate signal transduction[16].

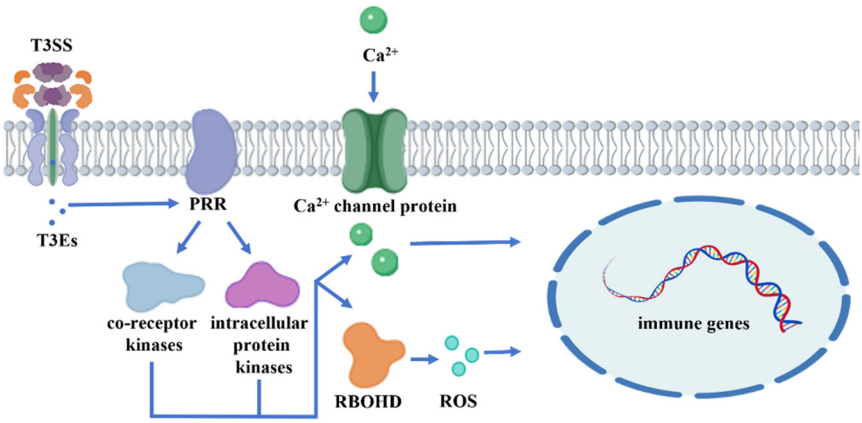

Fig. 2. The Process of How Pattern Recognition Receptors Recognize Type III Effectors and Active PTI

The figure 2 illustrates that immune signaling pathways such as the producing of ROS is activated through the injection of T3Es.

3.2 Recognition of T3Es by NLRs

NLRs (Nucleotide-Binding Leucine-Rich Repeat Receptors) are intracellular immune receptors present in the immune systems of both plants and animals. The second line of defense in the plant immune system, ETI (Effector-Triggered Immunity), is activated when intracellular NLRs specifically recognize pathogen effectors. ETI is generally more intense than PTI and is often accompanied by hypersensitivity response (HR), which involves localized cell death to restrict pathogen spread.

Some NLRs can directly bind to T3Es, triggering immune responses. For example, the NLR RPS2 in Arabidopsis thaliana directly recognizes the effector AvrRpt2 from Pseudomonas syringae, thereby activating an immune response[17].

However, most NLRs do not directly recognize T3Es but instead monitor host cellular proteins modified by T3Es. When T3Es alter these host proteins, NLRs detect the changes and activate downstream immune responses. For instance, the RIN4 protein in Arabidopsis thaliana serves as a guard protein for multiple NLRs. When RIN4 is

modified by Pseudomonas T3Es such as AvrRpm1 or AvrB, the NLR RPM1 is activated[18]. Similarly, when RIN4 is cleaved by AvrRpt2, the NLR RPS2 is activated[19].

NLR proteins contain two distinct N-terminal domain types: coiled-coil(CC) and Toll/interleukin-1 receptor(TIR). Upon recognizing T3Es, NLRs would be activated. TIR-type NLRs localize to and disrupt plasma membrane integrity. This process is preceded by their assembly into pentameric resistosome complexes antibodyosomes. NLRs with NADase-active TIR domains initially form tetrameric resistosomes, which subsequently transduce immune signals downstream through lipase-like proteins including EDS1 and SAG101. Ultimately ETI will be initiated[16].

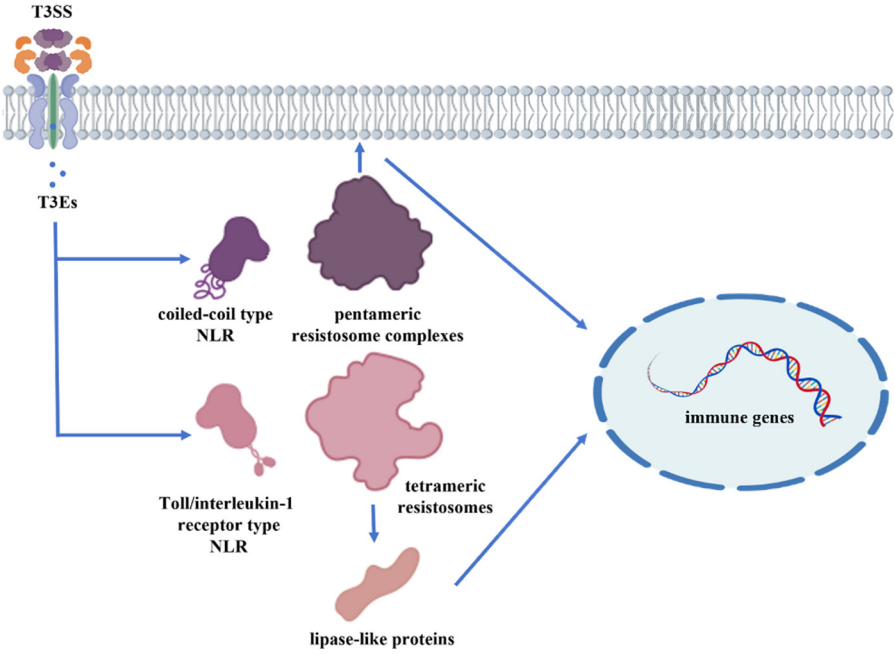

Fig. 3. The Process of How Nucleotide-Binding Leucine-Rich Repeat Receptors Recognize Type III Effectors and Active ETI

The figure3 illustrates how the 2 types of NLRs, coiled-coil type NLR and Toll/interleukin-1 receptor type NLR, activate immune signaling pathways.

4 Downstream Signaling Pathways Activated by Plant Recognition of T3Es

4.1 ROS Burst

Reactive oxygen species (ROS) primarily including O_2^-, H_2O_2, and ·OH. ROS are critical signaling molecules in plant immune responses. Upon recognition of T3Es by PRRs or NLRs, plant cells rapidly produce a large amount of ROS. The ROS burst is mainly driven by NADPH oxidase activation. NADPH oxidases are transmembrane proteins that oxidize NADPH in the cytoplasm to $NADP^+$ while reducing oxygen to O_2^-, which subsequently generates other ROS, such as H_2O_2. After T3Es is recognized by PRRs and NLRs, PRRs would associate with co-receptor kinases and intracellular protein kinases to initiate the expression of NADPH oxidase, RBOHD, and NLRs form antibodyosome to increase the expression of RBOHD. RBOHD ultimately leads to the increasing level of ROS. ROS not only directly kill pathogens but also activate downstream defense responses as a signaling molecules[20].

4.2 Ca^{2+} Signaling Pathway

The generation of Ca^{2+} signals is primarily achieved through calcium channels and the release of intracellular calcium stores. Recognition of T3Es triggers PRRs associating with co-receptor kinases and intracellular protein kinases and NLRs form antibodyosome, which both initiate the Ca^{2+} channel proteins and lead to a rapid increase in intracellular Ca^{2+} concentration. Calcium channels include voltage-gated calcium channels and ligand-gated calcium channels, while intracellular calcium stores mainly consist of the endoplasmic reticulum and vacuoles. The rise in Ca^{2+} signals activates calcium-dependent protein kinases (CDPKs), which subsequently regulate downstream defense responses. In Arabidopsis thaliana, CDPK5 and CDPK6 are activated by Ca^{2+} signals. This will lead to the downstream target proteins phosphorylation[21]. In tobacco, CDPK2 is activated by Ca^{2+} signals following NLR recognition of the tobacco mosaic virus effector p50, thereby regulating localized hypersensitive response (HR) and disease resistance[22].

4.3 MAPK Cascade

The MAPK (Mitogen-Activated Protein Kinase) cascade is a key signaling pathway central to plant immune responses. Upon recognition of T3Es by PRRs, PRRs then PRRs associating with co-receptor kinases and intracellular protein kinases and initiate the MAPK cascade. The MAPK cascade typically involves three tiers of kinases: MAPKKK (MAPK Kinase Kinase), MAPKK (MAPK Kinase), and MAPK. The MAPK cascade, when activated, triggers the upregulation of genes associated with plant defense, including PR1. In Arabidopsis thaliana, when certain T3Es are detected, intracellular MPK3 and MPK6 are phosphorylated following NLR activation, thereby regulating downstream defense responses[23].

4.4 JA and ET Signaling Pathways

Studies have shown that after T3Es invade plants, the JA (Jasmonic Acid) and ET (Ethylene) signaling pathways work synergistically to activate the expression of defense genes. The JA signaling pathway is primarily activated through degrading the repressors of JA signaling, JAZ (Jasmonate ZIM-domain) proteins. When JA accumulates, JAZ proteins are ubiquitinated and degraded by the $SCF^{(coi1)}$ complex, releasing the inhibition of the MYC2 transcription factor and activating JA-dependent defense gene expression. The ET signaling pathway regulates defense gene expression through ET receptors and downstream EIN2/EIN3 transcription factors. Activation of these defense genes includes the induction of NADPH oxidase genes which lead to increased ROS level, and the initiate of the MAPK cascade[24]. For example, in rice (Oryza sativa), the JA and ET signaling pathways will be activated following NLR recognition of rice blast fungus effector AvrPita, thereby enhancing disease resistance[25].

4.5 SA Signaling Pathway

SA (Salicylic Acid) is important for plant immunity. It has the ability to kickstart defense responses against biotrophic and hemibiotrophic pathogens and boost the development of systemic acquired resistance (SAR). SA is primarily synthesized via isochorismate synthase (ICS) and phenylalanine ammonia lyase (PAL). When T3Es are recognized by PRRs and NLRs, PRRs associating with co-receptor kinases and intracellular protein kinases and NLRs form antibodyosome, which both activate PR gene, causing SA synthesis rapidly increased. The buildup of SA triggers the activation of NPR1 (Nonexpressor of Pathogenesis - Related Genes 1), causing it to move into the nucleus and engage with TGA transcription factors to control the expression of defense genes. Upon the accumulation of SA, NPR1 undergoes a structural transformation, shifting from its monomeric state to a multimeric configuration. Subsequently, this multimeric form of NPR1 translocates into the nucleus, where it interacts with TGA transcription factors. This interaction would facilitate the activation of defense-related gene expression. [26]. In rice (Oryza sativa), OsNPR1 is activated by SA signaling following NLR recognition of the Xanthomonas effector AvrXa21, thereby enhancing disease resistance[27].

5 Conclusions

This review outlines the mechanisms by which pathogenic bacteria inject Type III Effectors (T3Es) into plants through the Type III Secretion System (T3SS) and summarizes the currently known strategies employed by plants to defend against T3E invasion. The plant immune system is structured into two distinct layers of defense: PAMP-triggered immunity (PTI) and effector-triggered immunity (ETI). Gram-negative bacteria utilize the T3SS to deliver T3Es into plant cells, thereby interfering with plant immune responses. In response, plants recognize T3Es through pattern recognition receptors (PRRs) and nucleotide-binding leucine-rich repeat receptors (NLRs), activating downstream immune responses such as reactive oxygen species (ROS) bursts, jasmonic

acid (JA) and ethylene (ET) signaling pathways, and other defense mechanisms. These responses trigger transcriptional reprogramming in plant cells, marked by altered expression of defense-related genes or the activation of programmed cell death (PCD). This process limits pathogen spread and enhances plant protection.

This review systematically summarizes the mechanisms by which pathogenic bacteria inject T3Es into plants via the T3SS and the strategies plants employ to counteract T3E invasion through PTI and ETI. The aim is to elucidate the principles underlying plant immune responses to T3E invasion and to provide new insights for the green transformation of agricultural production. T3Es is crucial for suppressing plant immune responses, and most bacterial plant pathogens employ the T3SS to directly inject T3Es into plant cells[28]. T3Es jeopardize the robust growth and optimal yield of economically crucial crops, such as rice (Oryza sativa) and Arabidopsis thaliana. Understanding the mechanisms by which plants counteract T3Es offers a critical theoretical framework for future studies focused on improving plant resistance to T3E-mediated infections. For example, erucamide has been shown to specifically disrupt the assembly of the T3SS in plant pathogens[29]. By employing molecular biology techniques to modulate plant physiological mechanisms, it may be possible to enhance erucamide accumulation in plants, thereby inhibiting T3E-mediated pathogenicity while avoiding the environmental damage caused by traditional chemical pesticides.

Currently, global climate change is accelerating the adaptive evolution of pathogens, and the overuse of traditional pesticides has led to increased pesticide resistance and environmental degradation. Therefore, elucidating the mechanisms by which plants resist T3E invasion may offer an effective pathway to address food security and sustainable agriculture. In this context, molecular biology approaches can be utilized to modulate the plant immune system, enhancing its ability to defend against T3Es.

There remains considerable scope for research into the mechanisms by which plants resist T3Es. For instance, some plant cells can interfere with T3E-mediated suppression of immunity through post-translational modifications. For example, the T3E AvrPtoB from Pseudomonas syringae causes the degradation of the Arabidopsis thaliana lectin RLK LecRK-IX.2 and supress PTI, but the Arabidopsis thaliana lectin RLK LecRK-IX.2 is able to physically interacts with AvrPtoB, phosphorylation AvrPtoB and activating the plant immune response[30]. More broadly, identifying other direct and effective mechanisms by which plants inhibit T3Es could provide robust solutions to the problem of T3E-mediated crop damage. Thus, investigating the mechanisms of plant resistance to T3Es holds profound scientific and practical significance.

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