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A Review on Plant-Pathogen Interactions

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ABSTRACT: Plants evolved nucleotide-binding domains (NLRs) that recognized effectors of pathogens, which resulted in a second layer of immune effector-triggered immunity (ETI). Biotrophic pathogens manipulate host physiological activities to obtain nutrients from living host cells and tissues, and hemibiotrophic pathogens secrete effectors that suppress host immunity and re-program host physiology to favor pathogen colonization. Plants have membrane-lined pores called plasmodesmata, which connect adjacent cells and facilitate symplast communication. Pathogenic microorganisms disrupt the actin cytoskeleton in plant cells and create hydrophobic spaces between pathogen-host plants to grow in the air. Plant PRR recognizes degraded fragments of bacteria and plant cell walls as PAMPs or DAMPs to trigger immunity. Pathogens use various effectors to suppress PAMP-triggered immunity, including protecting the mycelium from degradation by plant chitinases. Plants secrete antimicrobial proteins and compounds to fight infection by pathogens, but effector proteins secreted by pathogens degrades these compounds. Autophagy is an essential part of plant immunity to different pathogens. The black cob pathogen *Sporisorium reilianum* and the pathogen may act differently, suggesting that the Tin2 of *U. maydis* may be newly functionalized. *Phytophthora sojae* alters protein localization in the host plant cytosol to produce functional abnormalities and pathogenic effects. Microbial manipulation of the host may be achieved by directly targeting ER stress regulators, restricting defense-related vesicle transport as a virulence factor, and inhibiting the interaction between NPR1 and TGA transcription factors, reducing PR gene expression. Some pathogens inhibit the host's RNA silencing process to promote infection, and others neutralize or inhibit ROS production. Plants detect pathogens using their NLR and PRR, and kill cells with their effectors. Effectors are essential elements of plant-pathogen interactions. Although many effectors have been identified and characterized, there are likely still numerous unknown effectors lurking beneath the surface, waiting to be discovered.

Keywords: Effectors, Plasmodesmata, symplast, Autophagy and hydrophobic spaces.

INTRODUCTION

The plants use pattern recognition receptors (PRRs) to recognize pathogen-associated molecular patterns (PAMP) and stimulate pattern-triggered immunity (PTI) defense responses, which in turn limit the growth of pathogens. Plants evolved nucleotide-binding domains (NLRs) that recognized the effectors of pathogens and thus resulted in a second layer of immune effector-triggered immunity (ETI), which in turn, resulted in the evolution of pathogens that inhibited the first layer of immunity. It has been demonstrated that the effectors of pathogens are important pathogenic factors when it comes to infesting plants (Bigeard *et al.,* 2015; Yu *et al.,* 2017; Ghost *et al.,* 2019; Koeck *et al.,* 2011). These effectors have been shown to have multiple effects on a variety of targets, suppressing plant immunity, manipulating plant physiology, and being recognized by host defense mechanisms, thereby making it easier for pathogens to invade, colonize, and expand. The term effector, in its narrow sense, refers to the proteins that pathogens secrete into the extracellular and intracellular spaces of host plants (Duplessis *et al.,* 2011; Saitoh *et al.,* 2012; Gan *et al.,* 2013; Giraldo *et al.,* 2013), which are capable of triggering plant immunity when the effectors are released. As a consequence, the term effectors have been defined broadly, *i.e.,* "proteins and small molecules that alter host cell structure and function, thus facilitating the colonization of pathogens" (Horbach *et al.,* 2011). It is now well known that type III effectors have been extensively studied in bacteria (Mooney *et al.,* 2021; Schreiber *et al.,* 2021), LysM effectors in fungi (Hu *et al.,* 2021), and RxLR effectors in oomycetes (Anderson *et al.,* 2015; Chetsergon *et al.,* 2021), as well as several details about the biological functions that they play, making it possible to gradually gain insights into the mechanism of action of effectors. The purpose of this review is to discuss the role of effectors during infestation in different biological processes, highlighting the recent progress in studying plant-pathogen interactions and phytopathogenic bacteria, oomycete, and fungal effectors, as well as

looking forward to the future challenges facing effector research as a whole.

PLANT-PATHOGEN INTERACTION MODELS

In 1942, Flor proposed the "gene-for-gene" hypothesis between pathogen-free genes and resistance genes to plant diseases. The understanding of plant immunity mechanisms has been further developed. In plant pathology, the zigzag model is widely accepted and recognized as the central dogma (Flor *et al.,* 1971). Plant innate immunity is essentially a combination of two key components: pathogen-associated molecular pattern-primed immunity (PTI) and effector-primed immunity (ETI). It is the PTI response on the cell surface that serves as the first line of defense when pathogen-derived molecules (PAMP) are recognized by pattern recognition receptors (PRRs), mainly receptor-like kinases (RLK) and receptor-like proteins (RLPs) on the cell surface. As a result of pattern recognition, calcium influx, callose deposition, reactive oxygen species (ROS), activation of miRNA pathways, activation of MAPK cascades, and induction of the expression of several defense-related genes, like disease-process-related protein (RP), are usually associated with the reaction (Jones *et al.,* 2006; de Jonge *et al.,* 2011; Navarro *et al.,* 2008). In plants, this process initiates a second line of plant immune defense, ETI, in which plant NLRs, encoded by R genes, recognize and destroy pathogens' effectors, leading to ETI. The immune signaling pathway PTI is not an independent pathway of the immune system, according to recent studies. The PTI signaling pathway is integral to ETI when pathogens are present in plants. ETI activation enhances the PTI signaling pathway during pathogen infection. However, the activation of ETI alone is not sufficient to completely activate plant resistance and ETI likely functions by co-opting the PTI anti-pathogen mechanisms directly (Ngou *et al.,* 2021; Yuan *et al.,* 2020; Ngou *et al.,* 2020).

MODES OF PLANT-PATHOGEN INTERACTIONS

Infestation strategies of plant pathogens depend on their nutritional modes (Lo Presti *et al.,* 2015; Laluk *et al.,* 2010). To survive and complete their life cycle, biotrophic pathogens must manipulate host physiological activities to obtain nutrients from living host cells and tissues. To colonize living cells, they secrete effectors to suppress host immunity while minimizing damage to host cells (Lo Presti *et al.,* 2015; Lowe *et al.,* 2012). *Bipolaris sorokiniana, Verticillium dahlia*, and *Magnaporthe oryzae* are hemibiotrophic pathogens that infest the host by secreting different effectors at these specific spatial and temporal levels, which is a combination of both. The effectors secreted by pathogens are transferred to the host cell, where they interfere with various biological functions. Upon entry into the cell, the intracellular effectors suppress host immunity, re-program host physiology, and favor pathogen colonization (Tariqjaveed *et al.,* 2021). The following sections synthesize well-researched effectors under each mechanism.

A. Physical Barriers

(i) Host Plant Stomatal Defenses. In plants, numerous pathogens enter through their stomata, but guard cells, which are active immune sensing cells, can close those stomata, preventing pathogen entry, as soon as they detect microorganism characteristics (Zhang *et al.,* 2022). The bacterium *Pseudomonas syringae* produces a series of effectors that manipulate the stomata, which in turn increases pathogen entry. The phytohormone salicylic acid (SA), a phytohormone that is essential for stomatal closure, is antagonistic to the hormone jasmonic acid (JA), which is antagonistic to salicylic acid (SA) (Melotto *et al.,* 2006). There is a strong correlation between these results and the JA pathway, which is a common target for effectors that regulate stomatal defense, which suggests that successful pathogen colonization requires overcoming stomatal defenses. *P. syringae* effectors HopM1 and AvrE1, on the other hand, are targeted by the ABA signaling pathway. As a result of this, ABA accumulation in guard cells is increased, resulting in stomatal closure and promoting water-soaking lesions, which in turn leads to stomatal closure (Melotto *et al.,* 2017).

B. Plant Cell Wall Degradation

Most plant pathogens, particularly those lacking specific penetrating structures, secrete cell wall-degrading enzymes (CWDEs) to disrupt and colonize host cells, such as glycoside hydrolases, glycosyltransferases, and pectin lyases (Kubicek *et al.,* 2014; Gibson *et al.,* 2011). In necrotrophic pathogens, CWDEs are positively associated with virulence. *Botrytis cinerea, V. dahliae* and *Mycosphaerella graminicola* all exhibit this mechanism. In some cases, CWDEs target the waxy cuticle of the cell wall, protecting plants against biotic and abiotic stresses (Xue *et al.,* 2017; Ziv *et al.,* 2018). Several CWDEs degrade polysaccharides and cellulose in the cell wall, releasing oligosaccharides that stimulate plant immunity (Nguyen *et al.,* 2011; Van Vu *et al.,* 2012). In rice blast infections, these oligosaccharides are recognized by the OsCERK1 and OsCEBiP immune complexes as DAMPs (danger-associated molecular patterns).

C. Plasmodesmata–Callose Regulation

Plants have membrane-lined pores called plasmodesmata (PD), which connect adjacent cells and facilitate symplast communication. PD is critical to the successful pathogen colonization of plants. Through interactions with the callose synthases CalS1, CalS2, and CalS3, the RxLR3 effector of *Phytophthora brassicas* (Tomczynska *et al.,* 2020) inhibits callose accumulation in PD. *Arabidopsis* expresses HopO1-1, which increases molecular flux distance between adjacent plant cells based on PD. Also, HopO1-1 interacts with and destabilizes plant PD-localized proteins PDLP7 and possibly PDLP5, whereas mutant plants lacking PDLP7 or PDLP5 exhibit significant increases in bacterial proliferation (Li *et al.,* 2021; Liu *et al.,* 2020), suggesting that PDLP7 and PDLP5 play a significant role in plant immunity to bacteria.

D. Host Plant Cytoskeleton Destruction

A plant cell's cytoskeleton undergoes rapid changes when it comes in contact with pathogenic microorganisms, transporting cargo used to execute defenses locally (Schmidt *et al.,* 2007). By disrupting cytoskeleton formation, some effectors manipulate host metabolic and physiological processes. By binding to actin and disrupting the actin cytoskeleton, HopW1 inhibits endocytosis and protein transport to vesicles in *P. syringae* type III effectors. In an interesting twist, *Xanthomonas oleifera* T3E XopR also undergoes liquid-liquid phase separation (LLPS) by hijacking the Arabidopsis actin cytoskeleton's intrinsically disordered region IDR-mediated interactions. As XopR enters the host cell during infection, it forms macromolecular complexes with actin-binding proteins in the cell cortex, disrupting the actin cytoskeleton and altering multiple steps of actin assembly (Sun *et al.,* 2021).

F. Conditions Favorable to Infestation

Making a Hydrophobic Space. Some effectors promote infestations by constructing hydrophobic spaces between pathogen-host plants. Pathogens and their effectors often need to escape the water environment to grow in the air (Zhang *et al.,* 2022). It may contribute to the attachment of mycelium or spores to hydrophobic surfaces, interactions with the environment, host defense and other processes that contribute to spore dispersal and aerial growth of mycelium during escape from aqueous environments by providing a hydrophobic protein coating (Bayry *et al.,* 2012;Wosten *et al.,* 2001). It is highly induced at the start of infection and acts as a sensor for attachment to hydrophobic plant surfaces. Furthermore, the extracellular matrix protein EMP1 of rice blast fungus (Ahn *et al.,* 2004) is similar to hydrophobin in function. *M. oryzae* appressorium formation and pathogenicity were significantly reduced by EMP1 knockout mutants, but no effects were observed on mycelium growth or sporulation. This suggests that EMP1 plays a vital role in forming appressoriums.

Extracellular Alkalinization. In the presence of low pH, pathogenic fungi thrive (Penalva *et al.,* 2014), whereas in the presence of high pH, pathogenic fungi thrive (Fernandes *et al.,* 2017). Plants infected by fungal infections usually have an elevated pH in the surrounding host tissues, and this extracellular alkalinization is thought to contribute to fungal pathogenesis (Alkan *et al.,* 2013; Vylkova *et al.,* 2017). A wide variety of fungi contain rapid alkalinization factor (Ralf) homologs, which can increase extracellular pH and promote invasive fungal growth by stimulating the phosphorylation of mitochondrial-activated protein kinases. Root-infecting fungus *F. oxysporum* produces alkalinization and causes plant disease by using a functional homolog of the plant regulatory peptide Ralf, a peptide hormone capable of increasing the pH of surrounding fruit tissue by more than two units, resulting in an increase in pH within the apoplastic environment, which promotes fungal colonization (Masachis *et al.,* 2016; Thynne *et al.,* 2016).

Masking effect

Inhibition of PTI. The plant PRR recognizes degraded fragments of bacteria and plant cell walls as PAMPs or DAMPs to trigger immunity (Sanchez *et al.,* 2015). To suppress PTI, pathogens secrete various effectors. A PAMP called chitin, recognized by LysM receptors (Miya *et al.,* 2007), activates PTI. We use chitin as an example to demonstrate several ways to suppress the effects of PAMP-triggered immunity. As far as chitin is concerned, pathogens use a variety of methods: (i) protecting the mycelium from degradation by plant chitinases, (ii) inhibiting LysM receptor recognition, (iii) isolating and masking chitin oligosaccharides, (iv) targeting chitinases for degradation, and (v) modifying and transforming cell wall components (Sanchez *et al.,* 2015; Kombrink *et al.,* 2017;Volk *et al.,* 2019). An important strategy used by pathogens to inhibit PTI is maintaining cell wall integrity. To achieve this, plants secrete chitin-binding lectins that bind to the chitin layer to block the action of plant chitinases, thereby inhibiting the release of free chitin. As a member of the Cerato-Platanin Protein (CPP) family, VdCP1 exhibits chitin-binding properties (Zhang *et al.,* 2017), which may protect the fungal cell wall from degradation by enzymes. It is hypothesized that Sta1 plays a role in maintaining fungal cell walls by overexpressing fungal mycelium to chitinase and glucanase. In addition to functioning as a stage-specific stealth molecule, Sta1 may prevent the release of fungal cell wall-derived elicitors. A common strategy for pathogens to avoid PTI is to target receptor-like kinases. Through the use of a specific chitin deacetylase (Tanaka *et al.,* 2021; Gong *et al.,* 2020; Gao *et al.,* 2019), the composition of the cell wall is changed. As chitosan is relatively inactive in immunogenicity and a poor substrate for chitinases, it reduces the release of chitin oligomers, triggering defense (Hadwiger *et al.,* 2013; Cord-Landwehr *et al.,* 2016). The broad bean rust fungus *Uromyces fabae* and the maize anthracnose pathogen *Colletotrichum graminicola* have been studied. The surfaces of the infected structures on the plant cuticle exposed chitin by fluorescence microscopy using fluorescently labeled lectin wheat germ agglutinin (WGA). However, the structures formed after invasion of the host surface do not exhibit chitin but rather glycosylated modifications of chitin. The polysaccharide deacetylases VdPDA1 and FovPDA from the invasive xylem fungus *V. dahliae* and *Fusarium oxysporum*. It has recently been reported that *U. maydis* contains seven genes for chitin deacetylase (CDA). These genes encode enzyme-active proteins, which are differentially expressed during colonization. They modify chitin into chitosan to evade host recognition.

Antagonism. Plants secrete antimicrobial proteins and compounds when they detect infection by pathogens (Osvourn *et al.,* 1996; Selitrennikoff *et al.,* 2001). Plants need to defend themselves against various pathogens using structural antimicrobial compounds, such as saponins and tomatine, among others. Certain effectors secreted by pathogens, acting as detoxifying

enzymes, may degrade these antimicrobial compounds. A saponin detoxifying enzyme produced by *Gaeumannomyces graminis*, which attacks oat roots, effectively infects oat tomatoes, resulting in the steroidal glycol alkaloid tomatine, an antimicrobial compound that is resistant to fungal pathogens. Moreover, *U. maydis* secretes the virulence-promoting repeat effector Rsp3 (Ma *et al.,* 2018) and modified self-protection mechanisms decorated on the mycelium to resist attack by antimicrobial compounds. In fungal hyphae, Rsp3 is highly expressed during plant colonization.

Physiological Activities of the Host

Plant Gene Transcription. The effector protein in the host nucleus acts as a transcription factors to reprogram the host defense pathways. Pepper blotch bacteria inject AvrBs3 into plants as an effector protein. It interacts with soybean transcription factor GmSPL121 to suppress plant immunity and is injected by the bacterium *Xanthomonas campestris* pv. *campestris.* Through interaction with the C-terminal EAR module, the oomycete HaRxL21 mimics the recruitment of the transcriptional co-repressor Topless (TPL) to the transcriptional repressor site of the host plant, thereby suppressing plant immunity and increasing host susceptibility to necrotrophic and biotrophic pathogens (Harvey *et al.,* 2020). A wheat stripe rust effector protein *Puccinia striiformis* Pst_A23 also targets post-transcriptional modifications. In plants, variable splice site-specific precursor RNA motifs suppress host immune responses and promote pathogenicity (Tang *et al.,* 2022) by binding to Pst_A23 effector proteins.

2.4.2 Host Plant RNA. *Blumeria graminis* secretes ribonuclease-like effectors, which are proteins with ribonuclease (RNase-type) folding (Ralphs). In wheat, transgenic expression of the ribonuclease-like effector CSEP0064/BEC1054 increases susceptibility to infection and prevents RIP from degrading ribosomal RNA, so it keeps living cells as a nutrient source for the fungal pathogen (Pennington *et al.,* 2019). There's a lot of stuff in this process, but BEC1054 might be the key. It interacts with total RNA and produces virulence in wheat by targeting a bunch of host proteins, like glutathione-S-transferase, malate dehydrogenase, Pr5, Pr10, and eEF1γ . The effector Fg12, secreted by *F. graminearum* is also a ribonuclease, and Fg12 degrades total soybean RNA, induces plant death, and promotes pathogen virulence (Yang *et al*., 2021), similar to the ribonuclease VdRTX1 secreted by *V. dahliae*, which translocates to the plant nucleus and kills cells. In addition to causing cell death, Zt6 in the *Septoria* blotch fungus *Zymoseptoria tritici* is not essential for the pathogen's virulence (Kettles *et al.,* 2018).

Plant Cell Degradation. It is well known that autophagy and ubiquitin-proteasomes are essential for maintaining cellular homeostasis and maintaining normal cellular physiological functions. The proteasome and autophagy pathways have been increasingly studied as central hubs for microbial effectors (Cohen- Kaplan *et al.,* 2016; Langin *et al.,* 2020) and the autophagy and protein-ubiquitin systems have become common targets for many effectors, and are essential for plant immunity to different pathogens (Banfield *et al.,* 2015). Plants use autophagy as a ubiquitous intracellular degrading process to resist external stresses and degrade harmful substances. Phytoplasmas grow and differentiate because of it. Autophagy is a crucial part of plant innate immunity that limits pathogen-induced programmed cell death (PCD). Arabidopsis autophagy-associated protein (ATG) is a key component of autophagy (Wang *et al.,* 2015). To regulate autophagy, three of the *P. syringae* effector proteins HrpZ1, HopF3, and AvrPtoB employ several molecular strategies. Together, autophagy is likely to deliver a significant offensive towards the infected cell. Since autophagy is enhanced and inhibited by these effectors, this implies that autophagy may have various functions at distinct stages throughout the infection process. The M. In contrast, the AvrPiz-t effector of *R. oryzae* manipulates plant defense by targeting the ubiquitin-proteasome system of the host (Zhang *et al.,* 2022). As an interesting note, the Tin2 homologs in the black cob pathogen *Sporisorium reilianum* and the pathogen may act differently, stabilizing or inhibiting a variety of protein kinases with different ZmTTK analogs, while only the Tin2 of *U. maydis* is thought to be involved in anthocyanin biosynthesis, suggesting that Tin2 of *U. maydis* may be newly functionalized to promote a pathogenic lifestyle (Tanaka *et al.,* 2019). In wheat, the SNF1-related kinase TaSnRK1α interacts with an orphan protein, TaFROG, whose expression is induced by the fungal toxin deoxynivalenol (DON).

Host Plant Protein. A few effectors also alter protein localization to produce functional abnormalities and pathogenic effects in the host plant cytosol. HopNI is another well-studied effector protein of *P. syringae*, which cleaves an intrinsic protein of photosystem II in tomato cells, reducing water photolysis. In the wheat strain *Puccinia striiformis*, the haustorium-specific effector protein Pst_12806 interacts with the wheat TaISP protein's C-terminus Rieske domain (a putative component of cytochrome b6-f). As a consequence, plant electron transport is reduced, ROS accumulate less, which in turn inhibits genes related to defense. In addition, the nucleophile integrin-like effector SsITL interacts with the calcium-sensitive receptor (CAS) in chloroplasts and interferes with CAS-associated SA-mediated immunity (Zhu *et al*., 2013; Tang *et al.,* 2020). Plant resistance to blast fungi is negatively regulated by the endoplasmic reticulum (ER)-lumen-bound immunoglobulin (BIPS) stabilized by *Phytophthora sojae*. Microbial manipulation of the host may be achieved by directly targeting ER stress regulators (Jing *et al.,* 2016). PevD1, an effector of the soil-borne fungus *V. dahliae*, activates CRY2 indirectly by opposing an asparagine-rich protein. Induces JA and SA-responsive genes and promotes colonization of blast-infested leaves by moving from the nucleus to the nucleoplasm (Boevink *et al.,* 2016). Induces ROS-associated zinc-finger transcription factor TaLOL2 in wheat stripe rust (Qi *et al.,* 2019).

Host Vesicle Transport. Plant hosts and pathogens are directly correlated. It has been suggested that

extracellular vesicles may be involved in the host pathogenesis of the maize black cob disease pathogen *Ustilago maydis* (Kwon *et al.,* 2021). It was found that many EVs contained mRNAs for virulence-related proteins, some of which did not contain the predicted secretory signal. As a result, EVs are likely to act as delivery mechanisms for pathogens and hosts to communicate. One host target for BEC4 is the ATP ribosylation factor-GTPase activating protein (ARF-GAP), which controls eukaryotic membrane transport (Schmidt *et al.,* 2014). BEC4 restricts defense-related vesicle transport as a virulence factor.

Manipulating Plant Downstream Immune Responses

Plant Hormone Signaling. The chloroplast can convert SA from chorismate, the shikimate pathway product (Herrmann *et al.,* 1999; Dempsey *et al.,* 2011) which can cause SARs and ISRs. Similarly, SA can be degraded by effectors, such as Ralstonia solanecearum POP (Jacobs *et al.,* 2013), which encodes a type III secreted effector in the AvrE family. Roots and stems of tomatoes are natural infection sites for *R. solanacearum*, so this inhibits SA defenses. A few of *Sclerotinia sclerotiorum's* effectors can degrade SA. Aside from that, effectors can also influence SA indirectly; for example, the effector non-expression of pathogenesis-related genes 1 (NPR1) is crucial for regulating the SA pathway (Dong *et al.,* 2004; Wang *et al.,* 2006), which is related to tolerance to salt, oxidative stress, and plant immunity (Saijo *et al.,* 2020), as well as regulating PR gene expression. PNPi is the conserved effector in stripe rust *Puccinia striiformis*. That means it can interfere with the interaction between NPR1 and TGA transcription factors and reduce PR gene expression (Wang *et al.,* 2016). Plant innate immunity is subverted by the fungal effector RxLR48, which promotes nuclear NPR1 localization and inhibits proteasome degradation to suppress SA signaling.

In both SA-dependent and SA-independent conditions, JA modulates plant immunity to hemibiotrophic pathogen infections, making it an effective target for effectors. The effector *U. maydis* JA/ET signaling inducible factor 1 (Jsi1) was recently found to interact with several members of the TPL/TPR protein family of plant co-repressors (Darino *et al.,* 2021). Unlike the above-mentioned activation of the JA pathway, *M. oryzae* uses an antibiotic biosynthetic monooxygenase effector, ABM, for converting fungal and host JA to hydroxylated JA, which is secreted during host penetration to bypass defense responses.

RNA Silencing. RNAs were found to indirectly regulate the expression of R genes in apple plants by targeting genes associated with co-expression of R genes (Zhang *et al.,* 2019), suggesting that the role of sRNAs in ETI is likely to be significantly more significant than previously anticipated. Small RNAs are common among plant fungal pathogens (Weiberg *et al.,* 2013; Zhang et al., 2016; Wang *et al.,* 2017; Guo *et al.,* 2019). *Puccinia graminis* f.sp. *tritici* inhibits RNA silencing in plants and hinders plant defense by altering the abundance of small RNAs that act as defense regulators. *Sclerotinia sclerotiorum* is a necrotrophic fungus that produces an array of high abundance sRNAs during infection. *S. sclerotiorum* sRNAs are significantly downregulated in hosts when compared to pre-infection, suggesting they may act as a means of silencing immune components in plants. In the oomycete pathogen *P.sojae* (Qiao *et al.,* 2013; Xiong *et al.,* 2014; Hou *et al.,* 2019; Ye *et al.,* 2016), RNA silencing repressors PsPSR1 and PsPSR2 inhibit RNA silencing in plants by suppressing secondary siRNA biogenesis, which promotes infection, and the ectopic expression of these RNA silencing repressors increases plant sensitivity to viruses. It appears that some eukaryotic pathogens have evolved virulence proteins that inhibit the host's RNA silencing process to promote infection. By using the dicer-like proteins BC-DCL1 and BC-DCL2, *B.cinerea* produces small RNAs that are transported into *Arabidopsis* cells to interfere with RNAi. *Arabidopsis* Argonaute1 (Ago1) binds sRNA effectors and suppresses host immunity.

Reactive Oxygen Species. Plant immune responses are triggered by pathogen-induced ROS (Jwa *et al.,* 2017; Torres *et al.,* 2010). Generally, ROS produced by apoplasts is produced by peroxidases, whereas ROS generated at the plasma membrane are NADPH oxidases, also known as respiratory burst oxidase homologs (RBOHs), triggered by peroxidase-induced oxidative burst amplifiers (Bindschedler *et al.,* 2006). To achieve successful colonization, pathogens have devised a variety of strategies to neutralize or inhibit ROS production. To prevent ROS accumulation during early infection, the rice blast fungus secretes the peroxidase-peroxidase CPXB. In vitro, the nontoxic protein AVR-Pii inhibits ROS burst and NADP-ME activity specifically (Singh *et al.,* 2016; Dangol *et al.,* 2019), which is essential for ROS accumulation in rice. The iron-binding SSP family effector BcIBP in *B.cinerea* prevents Arabidopsis ROS formation by limiting metal accumulation in the cells (Liu *et al.,* 2019). Researchers have shown that *M. oryzae's* effector AVR-Pita interacts with the rice mitochondrial COX assembly protein OsCOX11, a key regulator of reactive oxygen metabolism (Han *et al.,* 2021). By increasing COX activity in mitochondria, AVR-Pita prevents ROS accumulation.

Plant Cell Death. Plants detect pathogens using their NLR and PRR, and they kill cells with their effectors. In rice protoplasts and *N. benthamiana*, transient expression of rice fungus effectors (MoCDIP1 to MoCDIP5) induces cell death, suggesting they function during necrotrophic stages. The protein is however secreted into apoplast after plant infection. Upon entering plant cells, it induces cell death and defense responses. On the other hand, some pathogens promote their invasion by inhibiting the HR response, such as *P. syringae* type III effector HopS2, which has exceptionally strong HR inhibition (Guo *et al.,* 2009). By suppressing cell death, ROS accumulation, and callose deposition induced by PST322, an elicitor protein of PST, PstCFEM1 overexpression suppresses wheat stripe rust. Another group of secondary metabolites secreted by pathogens is known as host-selective toxins (HSTs) derived from protein. An example of how PtrToxA interacts with ToxABP1 in

wheat chloroplasts is the host-selective toxins ToxA and ToxB secreted by *P. tritici-repentis* (Ciuffetti *et al.*, 2010). There have been studies showing that effectors with necrosis and ethylene-inducing peptide domains (NEPs) can kill plant cells, like NEP1-like proteins (NLPs), which trigger light-dependent cell death in *Arabidopsis*, as well as post-translational activation of mitogen-activated protein kinase activity, callose deposition, nitric oxide, and reactive oxygen intermediates (Qutob *et al.*, 2007).

CONCLUSIONS

Effectors are essential elements of plant-pathogen interactions. They exert their pathogenic effects primarily by targeting R proteins in the plant. By understanding the intricate interplay between effectors and R proteins, we can uncover new avenues for developing effective strategies to protect plants from devastating diseases. Although current research on effectors is quite prolific, there is still much to uncover about their mechanisms of action. One notable model in this field is the iceberg model proposed by Thordal-Christensen (Thordal- Christensen *et al.*, 2020). This model suggests that while many effectors have been identified and characterized, there are likely still numerous unknown effectors lurking beneath the surface, waiting to be discovered. The iceberg model serves as a reminder that our current understanding of effectors is only the tip of the iceberg, and there is still much more to explore and unravel in this fascinating area of research. By continuing to investigate effectors and their mechanisms of action, scientists can further expand our knowledge and potentially uncover new therapeutic targets or strategies for combating diseases caused by pathogenic effectors.

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