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REVIEW ARTICLE

Apoplastic immunity in plants: A powerful weapon against plant pathogens

P Sujhee[1*] & Raji P[2]

[1]Department of Plant Pathology, College of Agriculture, Kerala Agricultural University, Vellanikkara 680 656, Thrissur, Kerala, India
[2]Department of Plant Pathology, Regional Agricultural Research Station, Pattambi 679 306, Palakkad, Kerala, India

*Correspondence email - sujheep98@gmail.com

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Abstract

Apoplastic immunity is a formidable first line of defence that plants use against invading pathogens at the extracellular interface. The apoplast, which includes the cell wall matrix, intercellular gaps and xylem vessels, serves as a dynamic battlefield for both host monitoring and pathogen attack. This compartment not only facilitates vital physiological activities like water, mineral and nutrient movement, but it also houses a wide range of defence molecules such as antimicrobial proteins, hydrolytic enzymes, secondary metabolites and reactive oxygen species (ROS). Upon pathogen entrance, cell surface-localised pattern recognition receptors (PRRs) identify conserved pathogen-associated molecular patterns (PAMPs), triggering PAMP-triggered immunity (PTI). This identification swiftly triggers signalling cascades involving calcium influx, mitogen-activated protein kinase (MAPK) pathways, apoplastic oxidative burst and ion fluxes, all of which work together to build cell wall barriers and limit pathogen proliferation. Furthermore, defence-related phytohormones, most notably salicylic acid (SA), jasmonic acid (JA) and ethylene (ET) coordinate local and systemic responses, boosting antimicrobial compound accumulation and defence gene activation.

Keywords: apoplast; disease resistance; pathogen-associated molecular patterns; pathogen-associated molecular pattern- triggered immunity; pattern recognition receptors

Introduction

Plants are constantly attacked by a diverse range of microorganisms. Plants are sessile and do not possess an adaptive immune system. Even so, plants evolved a strong innate immune system capable of avoiding infections from most potential pathogens (1). Plants use their cuticle and cell wall as physical barriers to prevent microbial invasion (2). Fig. 1 implies that the plant apoplast contains a variety of hydrolases, protease inhibitors and antibacterial substances to prevent microorganism infection. Once these barriers have been overcome, plant cell surface receptors are activated to sense the invading microbes via detecting the molecular patterns derived from microbes or plants known as microbe-associated molecular patterns (MAMPs) or damage associated molecular patterns, which ultimately activate pattern triggered immunity (PTI), including reactive oxygen species (ROS) production, ion fluxes, callose depositions, defence-related gene expression and secondary metabolism (3, 4). Pathogenic microorganisms have evolved techniques for dealing with plant immune responses over time. Plant pathogens commonly use secreted effectors to impair plant defences and promote infection. To fulfill virulence role, such effectors act in the plant apoplast (apoplastic effectors) or inside plant cells (cytoplasmic effectors). Cytoplasmic effectors translocate into distinct cell compartments and interfere with several plant physiological processes, including protein synthesis or secretion. ROS production, secondary metabolite synthesis, mitogen-activated protein kinase activation (MAPK) and epigenetic changes (5). The initial interactions between microorganisms and plants take place in the plant apoplast, where there are extracellular plant pathogens as well as the feeding structures of numerous filamentous pathogens. Apoplastic effectors frequently play a determining role in plant-pathogen interactions. Here, we highlight recent research on how apoplastic effectors function during plant-pathogen interactions.

Apoplastic-mediated signalling in plant pathogen interaction

The apoplast, which is an extracellular continuum of cell walls, intercellular spaces and apoplastic fluid that connects plant cells, is the first site of contact between a plant and invading bacteria or other microbes and thus plays an important role in determining whether colonisation, immune recognition, or successful defence will occur (6). The plant cell wall serves as both a structural barrier and a dynamic chemical environment in this extracellular compartment; microbes may attempt to modify the cell-wall matrix to gain access to nutrients, while the plant can reinforce the wall through the deposition of polymers or other modifications to prevent pathogen advancement. Furthermore, the apoplast is not biochemically inert at the time of invasion; instead, plants utilise endogenous protein precursors in the apoplast to generate "danger signals" that activate immunological responses (7). One of these responses is the rapid creation of ROS, as well as pH and ion flux alterations, all of which contribute to a hostile environment for the pathogen. Plants produce apoplastic proteases and other defence-related proteins (such as pathogenesis-related proteins

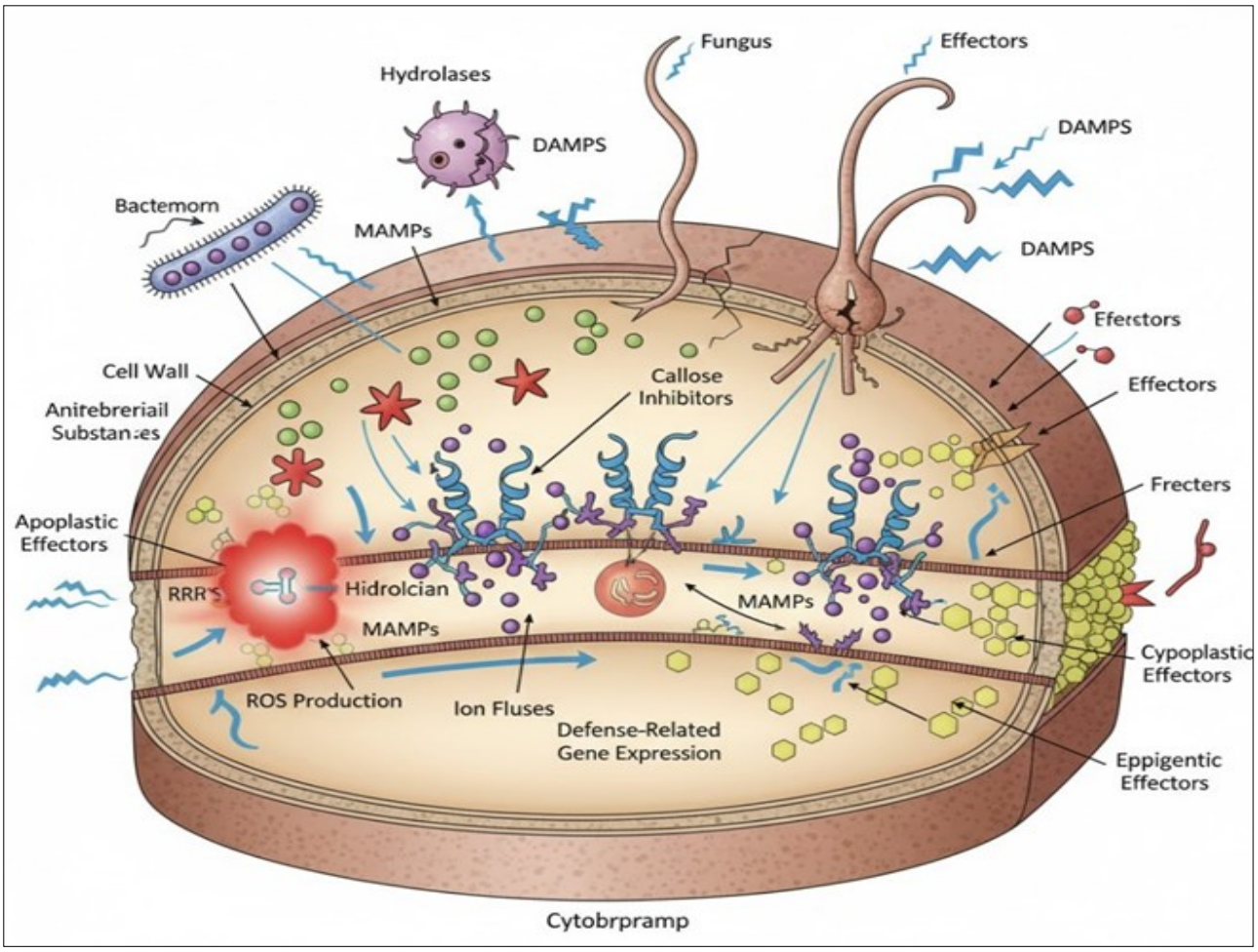

Fig. 1. The apoplast as a site for the interactions of plants with bacteria.

and hydrolases) into the apoplast, which destroy microbial compounds or interfere with pathogen activity. Fig. 2 depicts how bacterial pathogens thwart plant defence by deploying apoplastic effectors or inhibitors that neutralise host proteases, reduce immunological signals and adjust apoplastic conditions, highlighting the apoplast as an active "battlefront" of molecular warfare (8). Thus, the apoplast should be considered not as a passive region, but as a highly dynamic interface where structural, biochemical and molecular conversations or conflicts between plant and bacteria dictate host resistance or susceptibility.

Chemical and physical properties during apoplastic infection

During pathogen invasion the apoplast, that includes the cell wall matrix, intercellular gaps and apoplastic fluid, undergoes significant physical and chemical modification, influencing the outcome of plant-microbe interactions (Fig. 3). Pathogens secrete cell wall degrading enzymes, loosening polysaccharide networks, while the host frequently responds with wall reinforcement via callose or lignin deposition and cross-linking of wall polymers, increasing mechanical resistance and decreasing porosity (9). Chemically, infection leads to rapid changes in the apoplastic

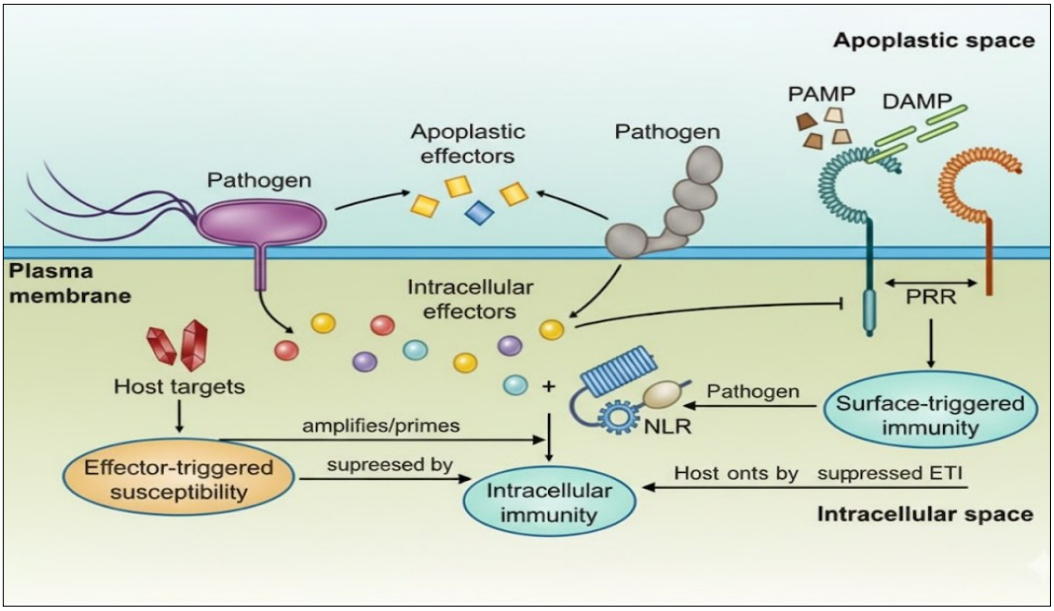

Fig. 2. Apoplastic-mediated signalling in plant pathogen interaction.

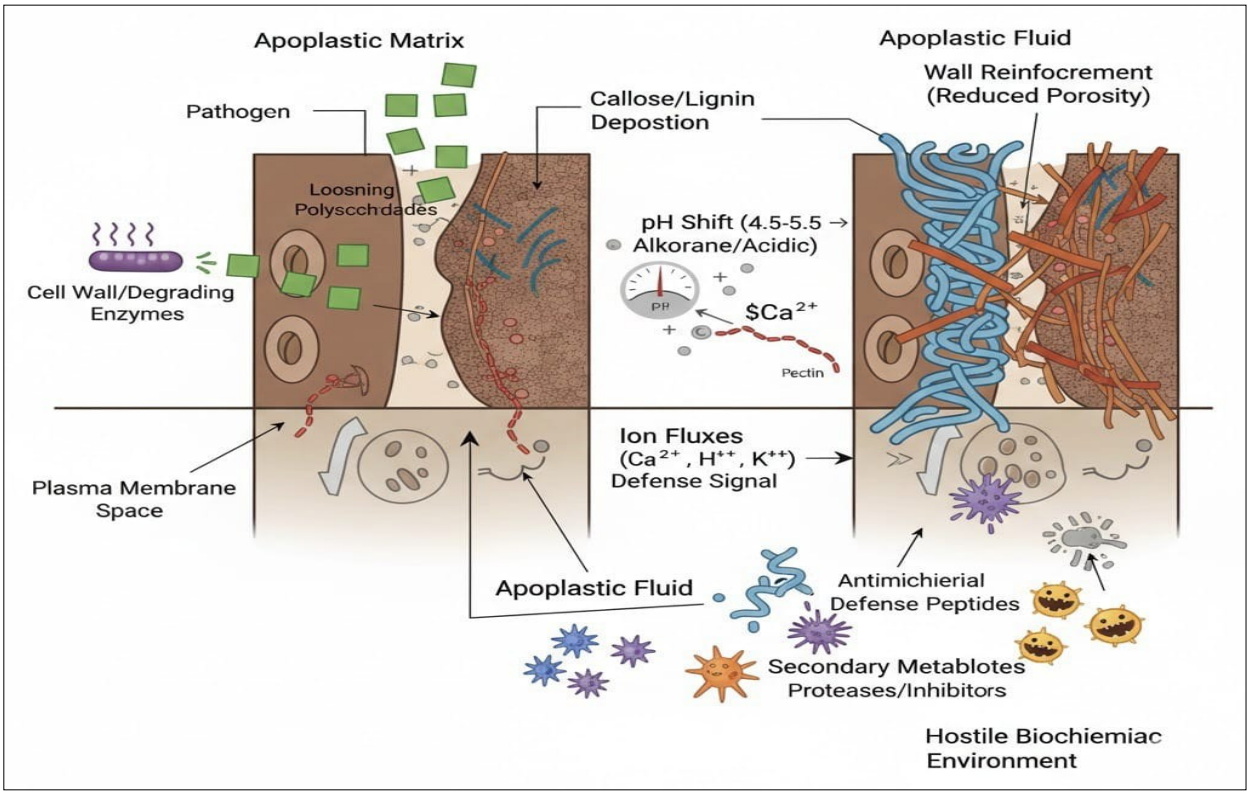

Fig. 3. Chemical and physical properties during apoplastic infection.

environment, causing the pH to shift from 4.5–5.5 to alkaline or acidic, depending on the pathosystem. This affects the activity of cell-wall-modifying enzymes and the binding of ions (e.g., Ca^{2+} to pectin), affecting wall stability and signalling. Microbial recognition is often accompanied by ion fluxes, such as Ca^{2+} release from wall-associated pectins into the apoplastic fluid or cytosol, which act as an early defensive signal (10). In addition, the content of apoplastic fluid changes rapidly: antimicrobial proteins, proteases, defence peptides, secondary metabolites and other secretome components accumulate in the apoplast, creating a hostile biochemical environment for the pathogen. During infection, the apoplast is no longer a static compartment, but rather a dynamically remodelled, highly reactive physical and chemical milieu that mediates both plant defence and pathogen assault.

Recognition mechanisms and apoplastic immune receptors

Apoplastic immunity recognition is the key sensory interface by which plants detect invading microorganisms. This surveillance is carried out by plasma membrane-localised pattern-recognition receptors (PRRs), which constantly monitor the apoplastic environment for 2 types of signals: MAMPs and damage-associated molecular patterns (DAMPs). As shown in Fig. 4, well-characterised LRR-RLKs such as flagellin-sensitive 2 (FLS2), EF-Tu receptor (EFR) and lipooligosaccharide-specific reduced elicitation (LORE) are high-affinity sensors for bacterial flagellin (flg22), elongation factor Tu (elf18) and medium-chain 3-hydroxy fatty acids, respectively (11). The lysin motif (LysM)-type receptors, such as chitin elicitor receptor kinase 1 (CERK1), lysin motif receptor-like kinase 4/5 (LYK4/5) and chitin elicitor binding protein (CEBiP), recognise

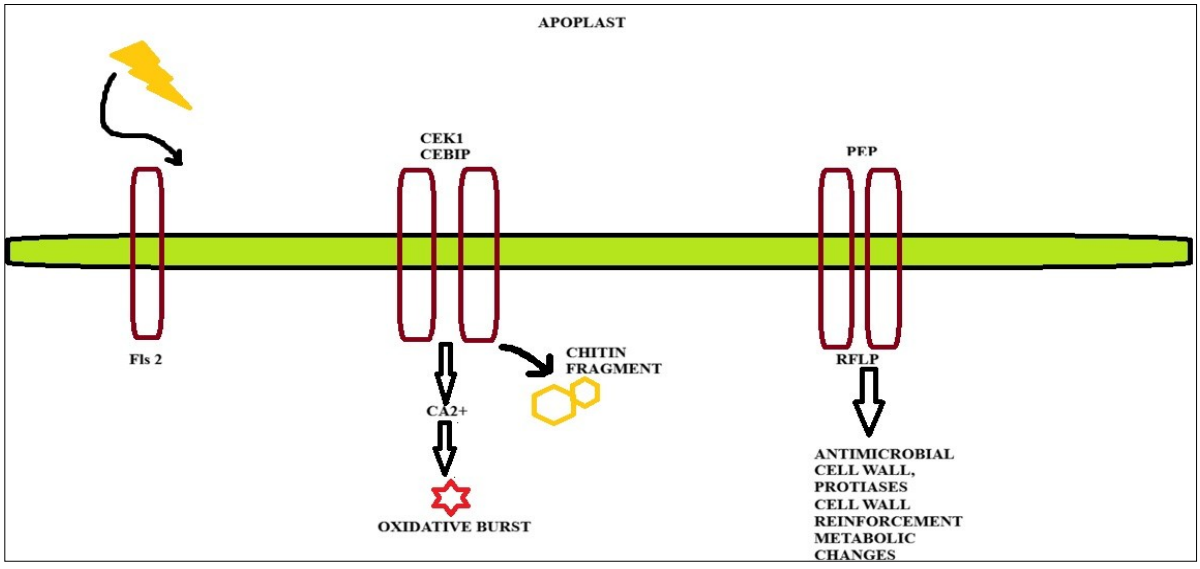

Fig. 4. Plasma membrane pattern-recognition receptors detect conserved microbial molecules in the apoplast and activate defence responses. Examples include FLS2 recognising flagellin, chitin recognising CERK1/CEBIP system and apoplastic Pep1 binding to receptors. Ligand binding triggers early signalling events at the apoplast membrane interface, including calcium influx, an oxidative burst and mitogen-activated protein kinase cascade, leading to pathogen-associated molecular pattern-triggered immunity.

carbohydrate-based signatures like fungal chitin, β-glucans and bacterial peptidoglycan, allowing for broad-spectrum detection across diverse microbial taxa (12). Beyond detecting exogenous infections, the apoplastic environment is also examined for endogenous "danger" signals emitted during tissue injury. These DAMPs, which comprise oligogalacturonides produced by polygalacturonase activity and cello triose obtained from cellulose breakdown, are detected by wall-associated kinase 1 (WAK1) receptors (10). The plant elicitor peptide receptor 1/2 (PEPR1/2) and MDIS1-interacting receptor-like kinase 2 (MIK2) receptors also recognise immune-enhancing endogenous peptides such as plant elicitor peptide ½ (Pep1/2), PAMP-induced peptide 1 (PIP1) and serine-rich endogenous (SCOOP) peptides (13, 14). The recognition of these compounds triggers powerful feed-forward loops that strengthen PTI. The transition from ligand perception to intracellular signalling is a tightly coordinated spatial and temporal process. Ligand binding stimulates the rapid formation of higher-order receptor-co-receptor complexes. Members of the somatic embryogenesis receptor-like kinase (SERK) family, including BRI1-associated receptor kinase 1 (BAK1)/SERK3 and SERK4, as well as suppressor of BIR1-1 (SOBIR1), play critical roles in PRR activation and signal amplification (15). Recent research reveals that these PRR complexes do not function in isolation, but rather actively rearrange into specialised plasma membrane nanodomains. The precise lipid content, membrane organisation and local pH of these nanodomains are now known to be important drivers of ligand binding effectiveness, receptor clustering and overall immune signal resilience (16). Activation of these apoplastic immune complexes initiates a series of early physiological responses at the membrane contact. These include respiratory burst oxidase homolog (RBOH)-mediated oxidative bursts, the activation of Ca^{2+} channels and fast apoplastic alkalinisation. Downstream, activation of receptor-such as cytoplasmic kinases (RLCKs) such BIK1 and PBL1 activates conserved MAPK pathways, resulting in enormous transcriptional reprogramming. This genetic shift causes antimicrobial proteins (such as PR1), proteases and peroxidases to be secreted into the apoplast to strengthen the cell wall (4). Finally, apoplastic proteasomes and subtilases modify the extracellular immunological environment by controlling ligand availability and converting peptide precursors into active signalling molecules (17). Together, these numerous detection and amplification systems construct the apoplast as a dynamic immunological arena that integrates several inputs to organise a multilayered response against invasion.

Antimicrobial substances in the apoplast

To prevent pathogen colonisation during the early stages of infection, the plant apoplast serves as a chemically dynamic defence arena loaded with a variety of antimicrobial chemicals. Reactive oxygen species (ROS) like H_2O_2, O_2^- and reactive nitrogen species (RNS) like NO, which are produced quickly by apoplastic peroxidases and plasma-membrane NADPH oxidases (RBOHD/F), make up a significant portion of this arsenal. These molecules cause direct toxicity, cause cell wall crosslinking and work in concert with phytohormone pathways to inhibit microbial growth (8, 18).

Apoplastic enzymes and proteins

The apoplast is a dynamic molecular battlefield in which plants use a sophisticated arsenal of enzymes and proteins to combat invading pathogens (19). This extracellular compartment is more than just a structural region; it is also the site of active, multidimensional immune surveillance. The production of ROS by oxidative enzymes such as class III peroxidases and oxalate oxidases is central to this defence; these ROS have direct antimicrobial effects and catalyse the lignification and cross-linking of cell wall polymers to strengthen the physical barrier (20). The production of hydrolytic pathogenesis-related (PR) proteins, such as chitinases (PR-3/PR-4) and beta-1,3-glucanases (PR-2), enzymatically breaks down fungal cell wall elements, adding to structural strengthening. The resultant oligosaccharide fragments act as DAMPs, enhancing immunological signalling (21–23). Recent proteomic and molecular breakthroughs have shed more light on the apoplast's complexity, revealing that it also contains a rich array of phyto cytokines and extracellular vesicles (exosomes), which allow for rapid cell-to-cell communication during infection (24, 25). Plants also use polygalacturonase-inhibiting proteins (PGIPs) to counteract pathogen-derived pectin-degrading enzymes, reducing tissue maceration and encouraging the buildup of elicitor-active oligogalacturonides (26). The apoplastic environment is also mediated by a complex proteolytic network in which proteases, including papain-like cysteine proteases (PLCPs) and their inhibitors, influence defence protein maturation and counter pathogen virulence factors (27). Furthermore, a few tiny cysteine-rich antimicrobial proteins, including defensins, lipid transfer proteins and thaumatin-like proteins (PR-5), damage microbial membrane integrity and contribute to both local and systemic resistance (28, 29). These coordinated enzymatic and proteinaceous components combine direct toxicity, cell wall reinforcement and strong immunological signals to provide a formidable first line defence.

Methods of pathogen suppression of apoplastic immunity

For colonisation, phytopathogens have developed a sophisticated arsenal of virulence methods aimed at bypassing or actively dismantling apoplastic immunity, which is the host's principal extracellular defence barrier. A key component of this suppression is the secretion of a variety of effectors into the apoplast that counteract PTI by interfering with immune-related enzymes and signalling molecules (23). Many pathogens use a suite of secreted antioxidants-including catalases, peroxidases and superoxide dismutases to detoxify ROS, preventing cell wall reinforcement and direct antimicrobial damage (30). Pathogens also undermine host surveillance by creating specialised protease inhibitors and effectors that target PRR complexes; this inhibition halts the proteolytic activation of immunological signals and prevents defence cascades from being amplified (31). While pathogens use an arsenal of cell wall-degrading enzymes (CWDEs) such as polygalacturonases and cellulases, they have also evolved mechanisms to avoid host-encoded inhibitors such as PGIPs (32). Pathogens aggressively reprogram the apoplastic environment into a nutrient-rich niche by modulating the flux of carbohydrates, amino acids and necessary metal ions, which is frequently accomplished by effector-mediated modulation of host transporters (33). Collectively, these strategies highlight the apoplast as a highly dynamic and disputed interface where pathogen virulence factors and host defence proteins are constantly co-evolving.

Pathogen-associated molecular patterns-triggered immunity (PAMPTI) through apoplastic receptors

Pathogen-associated molecular patterns-triggered immunity (PAMPTI) occurs at the plant cell surface when conserved PAMPs in the apoplast are detected by plasma membrane-localised pattern recognition receptors. Fig. 5 depicts the leucine-rich repeat receptor kinase FLS2 which recognises bacterial flagellin (flg22), whereas the EFR perceives elongation factor Tu (EF-Tu/elf18); both receptors rapidly recruit the co-receptor BAK1, resulting in receptor complex activation (34). Chitin fragments released into the apoplast are detected by CEBiP and the receptor kinase CERK1, whereas fungal xylanases are sensed by LeEIX receptors, demonstrating the diversity of apoplastic PRRs across pathogen classes (35). Ligand perception activates calcium-dependent protein kinases (CDPKs) and calcineurin B-Like proteins- CBL-interacting protein kinases (CBL-CIPK) signalling modules, resulting in phosphorylation of downstream targets such as the NADPH oxidase RBOHD and a rapid apoplastic ROS burst (36). In parallel, PRR activation induces a MAPK cascade involving MAPKKKs, MKK4/5 and mitogen-activated protein kinase 3/6 (MPK3/MPK6), which transduces extracellular PAMP perception into nuclear transcriptional reprogramming. These signalling pathways converge on the activation of defence-related genes and phytohormone signalling networks, reinforcing cell wall defences and establishing broad-spectrum resistance. Collectively, the image depicts PTI as a tightly coordinated apoplast-to-nucleus signalling process in which surface PRRs translate extracellular pathogen cues into robust intracellular immune responses. Also, PRR activation activates a MAPK cascade comprising MAPKKKs, MKK4/5 and MPK3/MPK6, which converts extracellular PAMP sensing into nucleus transcriptional reprogramming (37, 38). These signalling pathways converge on the activation of defence-related genes and phytohormone signalling networks, which reinforce cell wall defences and develop broad-spectrum resistance. Overall, the image displays PTI as a closely regulated apoplast-to-nucleus signalling mechanism in which surface PRRs transform external pathogen stimuli into powerful intracellular immune responses.

Effector-triggered immunity (ETI) through apoplastic receptors

Effector-triggered immunity is a second and more powerful layer of plant immunological response that is activated when pathogen-secreted effectors disrupt host cellular processes. Pathogens release MAMPs into the apoplast, which are initially detected by plasma-membrane-localised PRRs, resulting in PTI. To overcome PTI, effective pathogens release effectors that target PRRs or downstream apoplastic and intracellular signalling components, reducing PTI outputs (39). These effectors are then identified either directly or indirectly by intracellular nucleotide-binding leucine-rich repeat receptors (NLRs), as illustrated in the image, which detect effector activity rather than the effector itself (40). Activation of NLRs triggers ETI-associated signalling modules that overlap with PTI pathways, such as Ca_2^+ influx, ROS burst, kinase cascade activation, hormone signalling and transcriptional reprogramming. However, ETI responses are numerically greater and qualitatively unique, frequently resulting in a localised hypersensitive response (HR), which is defined by programmed cell death at the infection site and successfully limits pathogen dissemination (41). Fig. 6 depicts the integration and mutual potentiation of PTI and ETI outputs, underlining that ETI does not work independently, but rather enhances PRR-mediated defences to impart long-term disease resistance. Thus, effector-triggered immunity is an important surveillance mechanism that monitors pathogen virulence strategies and strengthens plant resistance via coordinated apoplastic and intracellular immune signalling networks.

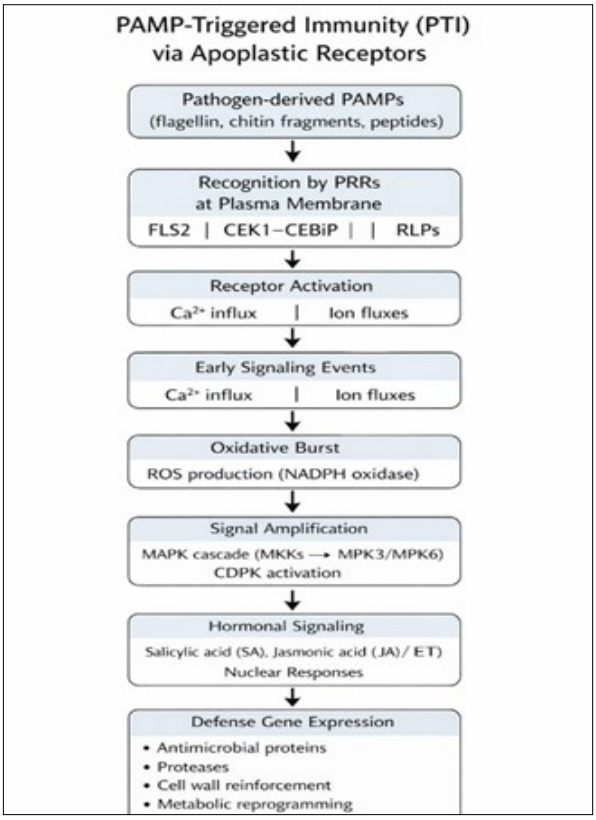

Fig. 5. Pathogen-associated molecular pattern-triggered immunity through apoplastic receptors.

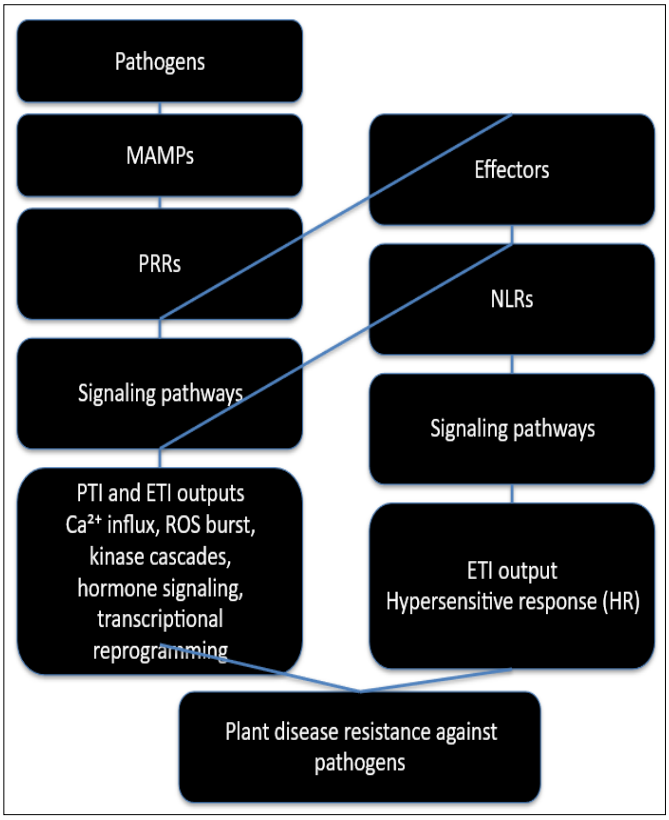

Fig. 6. Effector-triggered immunity through apoplastic receptors.

Damage-associated molecular pattern-triggered immunity through apoplastic receptors

Damage-associated molecular pattern-triggered immunity is a crucial component of apoplastic immune monitoring that is activated in response to host tissue damage caused by mechanical injury, insect herbivory, or pathogen invasion, as shown in the image. Endogenous DAMPs, such as oligogalacturonides, extracellular ATP, plant elicitor peptides (Peps) and cell wall fragments, are released into the apoplast after membrane disruption or cell wall degradation and are detected by plasma membrane-localised PRRs and co-receptors (42). Ligand binding causes receptor complex building and phosphorylation processes that attract RLCKs, which serve as immediate downstream signalling hubs. When PRR-RLCK complexes are activated, they cause early defence signals such as Ca^{+} influx through ion channels, membrane depolarisation with K^{+} efflux and H^{+} fluxes and activation of NADPH oxidases like RBOHD. Fig. 7 exhibits the results in a strong apoplastic ROS burst (43). These early signals activate calcium-dependent protein kinases (CDPKs) and MAPK cascades, which phosphorylate transcription factors and reprogramme nuclear gene expression. The downstream immune outputs such as callose deposition at the cell wall, secretion of extracellular defence molecules (EDNs) and accumulation of defence-related phytohormones such as salicylic acid, jasmonic acid and ethylene, all of which enhance basal resistance (44). Thus, DAMP-triggered immunity is a self-generated danger signalling system that combines apoplastic sensing with intracellular signalling networks to enhance plant defence during tissue injury and pathogen assault.

Apoplast symplast interaction during infection

The interplay between the apoplast and symplast is at the forefront of the plant-pathogen arms race, serving not as a static barrier but as a sophisticated, bidirectional communication centre. To convert extracellular pathogen detection into intracellular defence signals during plant-pathogen interactions, efficient immune responses necessitate continual and closely controlled crosstalk between these compartments. Many pathogens first deploy effectors in the apoplast, from which some penetrate the plasma membrane by endocytosis, membrane translocation patterns, or host-assisted uptake mechanisms to reach cytoplasmic or nuclear destinations, where they depress host immunity or rewire cellular processes. Fig. 8 shows that the spatial challenge is met by an integrated surveillance system that combines intracellular ETI via NLR proteins with immune signalling initiated at the cell surface via PRRs. This demonstrates that PTI and ETI are not distinct pathways, but rather an interconnected signalling continuum that spans the plasma membrane (44). According to recent research, full ETI activation requires PTI-associated signalling components like MAPK cascades, calcium-dependent protein kinases and transcriptional regulators; conversely, ETI significantly increases PTI outputs, such as the production of ROS and the expression of defence genes. This bidirectional information flow is further mediated by membrane-localised transporters, such as SWEET sugar transporters, amino acid transporters, aquaporins and ABC transporters, which govern the movement of nutrients, metabolites and signalling molecules. Pathogens frequently target these transport mechanisms to boost virulence by hijacking nutrient efflux, whereas the host regulates them to limit pathogen access or promote "nutritional immunity." Furthermore, the interchange of extracellular vesicles (EVs) containing antimicrobial proteins and short RNAs adds another layer to the interface, allowing for cross-kingdom RNA interference. Rapid redox and Ca^{2+}signalling provides a direct link between extracellular perception and intracellular responses, as apoplastic ROS produced by NADPH oxidases can affect plasma membrane channels or diffuse into the cytosol, while Ca^{2+} influx activates kinases and transcription factors required for immune reprogramming (14). When considered together, these mechanisms demonstrate how the apoplast-symplast interface functions as a dynamic communication platform, integrating cellular defence, signal amplification and pathogen detection to provide robust resistance to invasive infections.

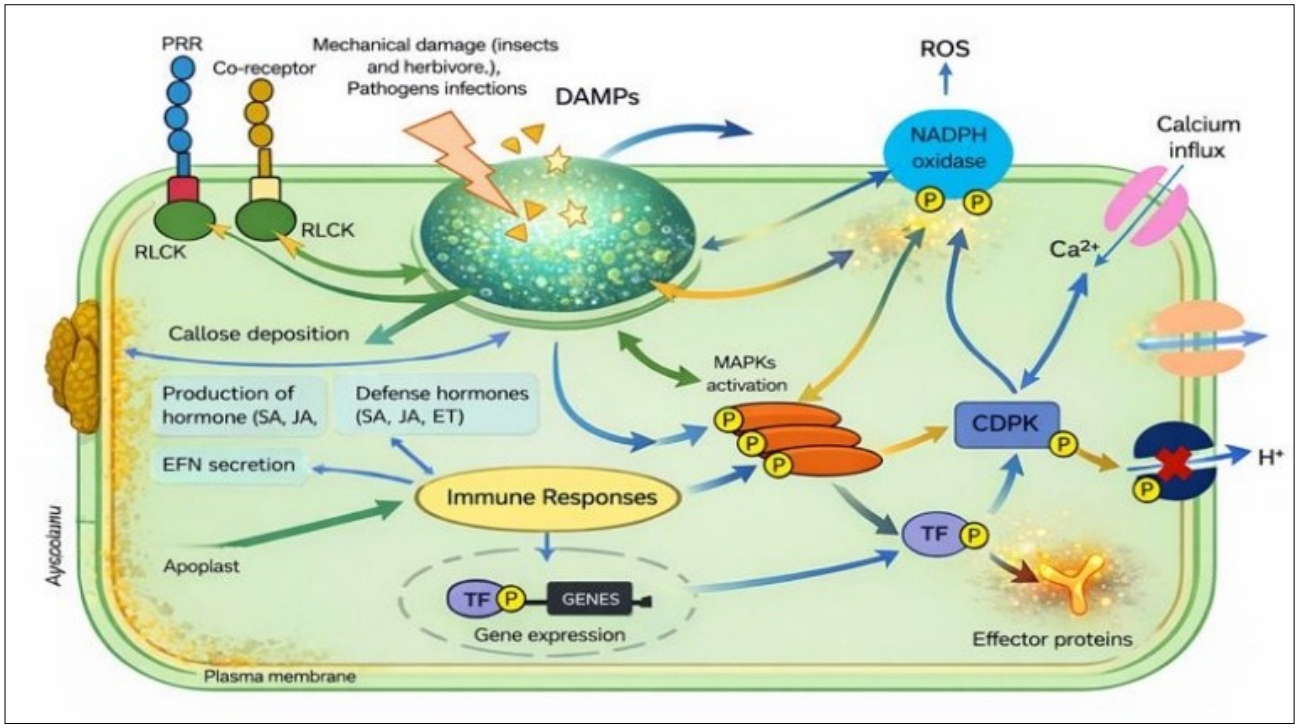

Fig. 7. Damage-associated molecular pattern triggered immunity through apoplastic receptors.

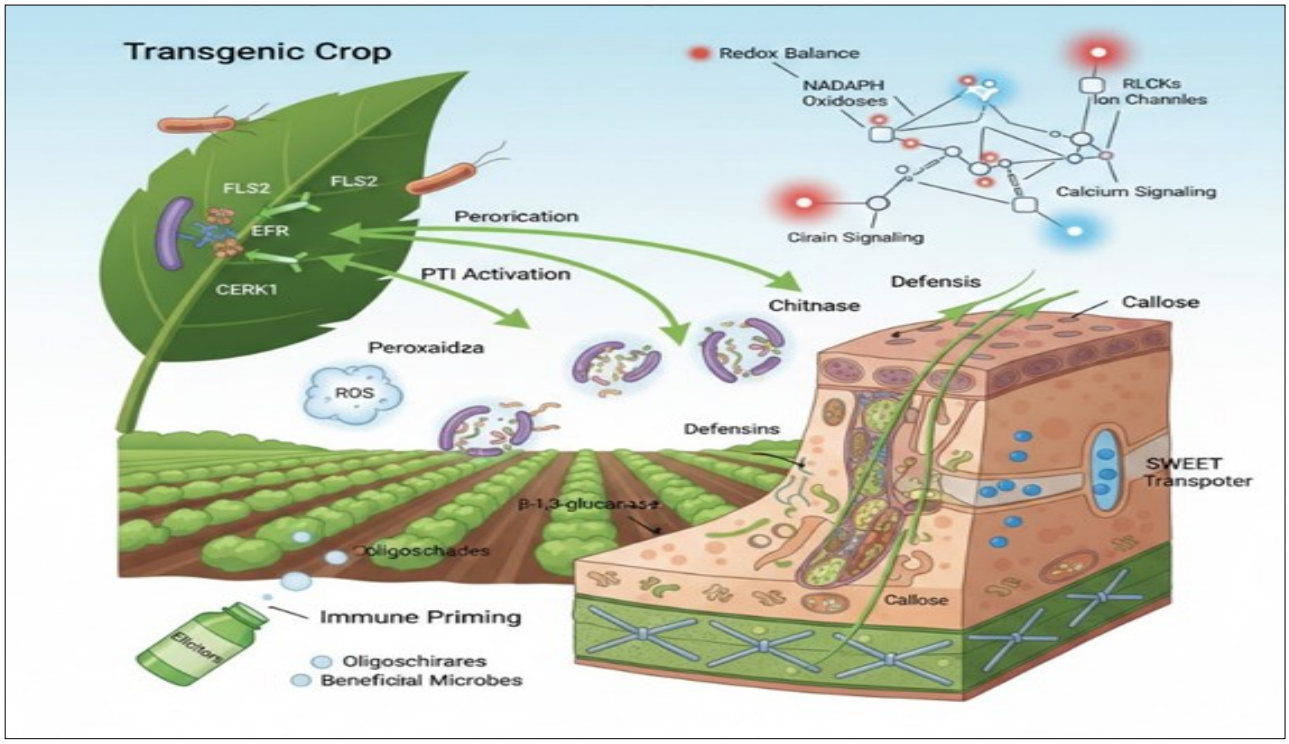

Fig. 8. Apoplastic immunity in crop protection.

Application of apoplastic immunity in crop protetction

Apoplastic immunity can be used to improve crop resistance against a wide range of diseases by increasing the plant's first line of defence at the extracellular interface. One major application involves the enhancement of PTI through the identification and deployment of effective PRRs, where transgenic or genome-edited crops expressing broad-spectrum PRRs such as FLS2, EFR, or CERK1 have shown improved resistance to bacterial and fungal pathogens by enabling early pathogen detection in the apoplast. Manipulation of apoplastic defence enzymes and proteins, such as peroxidases, chitinases, β-1,3-glucanases, defensins and polygalacturonase-inhibiting proteins, has been used to enhance antimicrobial activity and cell wall integrity, limiting pathogen entry and colonisation (45). Furthermore, priming apoplastic immunity with elicitors such as PAMPs, DAMPs, oligosaccharides and beneficial microbes improves basal defence responses, including ROS production and callose deposition, resulting in long-lasting and environmentally friendly disease resistance in crops. Breeding and biotechnology advances have made it possible to fine-tune apoplastic redox balance and calcium signalling, for example, by modulating NADPH oxidases, receptor-like cytoplasmic kinases and ion channels, to achieve rapid and controlled defence activation without compromising growth (45). Furthermore, pathogen-targeting techniques that control apoplastic nutrient pools, such as the inhibition of pathogen-induced SWEET sugar transporters, offer a promising avenue for limiting pathogen feeding and lowering disease susceptibility. The strategic application of apoplastic immunity via receptor engineering, defence protein enhancement, immune priming and precise signalling modulation provides a strong framework for developing long-term, broad-spectrum crop protection strategies that are appropriate for modern agricultural challenges.

Conclusion

Apoplastic immunity is a sophisticated and critical layer of plant defence that serves as a dynamic extracellular warfare for crops and pathogens. Researchers are unlocking new pathways for sustainable agriculture by transforming the apoplast from a passive physical barrier to an active immune hub that incorporates high-sensitivity PRRs, ROS signalling and strategic cell wall remodelling. This system organises a comprehensive response that includes antimicrobial proteins like as chitinases and defensins, as well as nutrition transporter regulation, to starve microbial invader. While major knowledge gaps exist about the spatial-temporal coordination of these components and the precise strategies pathogens employ to avoid them, the integration of modern multi-omics and functional genomics can fill these gaps. Finally, leveraging apoplastic biology through receptor engineering, immune priming and targeted breeding provides a solid foundation for developing resilient, broad-spectrum crop varieties that can withstand the growing pressures of global biotic stress while maintaining optimal growth and yield.

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Authors' contributions

PS collected and reviewed the literature, organised the content and drafted the manuscript. RP conceptualised the review, guided the structure and scientific content, critically revised the manuscript and supervised the overall work. Both authors read and approved the final version of the manuscript.

Compliance with ethical standards

Conflict of interest: Authors do not have any conflict of interest to declare.

Ethical issues: None

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors used ChatGPT to draw the images. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

References

1. Jones JD, Dangl JL. The plant immune system. Nature. 2006;444 (7117):323–29. https://doi.org/10.1038/nature05286
2. Hückelhoven R. Cell wall-associated mechanisms of disease resistance and susceptibility. Annu Rev Phytopathol. 2007;45(1):101–27. https://doi.org/10.1146/annurev.phyto.45.062806.094325
3. Boller T, Felix G. A renaissance of elicitors: perception of microbe-associated molecular patterns and danger signals by pattern-recognition receptors. Annu Rev Plant Biol. 2009;60:37–406. https://doi.org/10.1146/annurev.arplant.57.032905.105346
4. Whisson SC, Boevink PC, Wang S, Birch PR. The cell biology of late blight disease. Curr Opin Microbiol. 2016;34:127–35. https://doi.org/10.1016/j.mib.2016.09.002
5. Dora S, Terrett OM, Sánchez-Rodríguez C. Plant-microbe interactions in the apoplast: communication at the plant cell wall. Plant Cell. 2022;34(5):1532–50. https://doi.org/10.1093/plcell/koac040
6. Ali S, Tyagi A, Bae H. ROS interplay between plant growth and stress biology: challenges and future perspectives. Plant Physiol Biochem. 2023;203:108032. https://doi.org/10.1016/j.plaphy.2023.108032
7. Chen XR, Wang Y, Kale SD, Fang Y, Srivastava V. Apoplastic effectors-what roles do they play in plant-pathogen interactions? Front Microbiol. 2023;14:1149771. https://doi.org/10.3389/fmicb.2023.1149771
8. Li YB, Han LB, Wang HY, Zhang J, Sun ST, Feng DQ, et al. The thioredoxin GbNRX1 plays a crucial role in homeostasis of apoplastic reactive oxygen species in response to Verticillium dahliae infection in cotton. Plant Physiol. 2016;170(4):2392–406. https://doi.org/10.1104/pp.15.01930
9. Boudsocq M, Willmann MR, McCormack M, Lee H, Shan L, He P, et al. Differential innate immune signalling via Ca2+ sensor protein kinases. Nature. 2010;464(7287):418–22. https://doi.org/10.1038/nature08794
10. Albert I, Zhang L, Bemm H, Nürnberger T. Structure-function analysis of immune receptor AtRLP23 with its ligand nlp20 and coreceptors AtSOBIR1 and AtBAK1. Mol Plant Microbe Interact. 2019;32(8):1038–46. https://doi.org/10.1094/MPMI-09-18-0263-R
11. Karimi HZ, Chen KE, Karinshak M, Gu X, Sello JK, Vierstra RD. Proteasomes accumulate in the plant apoplast where they participate in microbe-associated molecular pattern (MAMP)-triggered pathogen defence. Nat Commun. 2025;16(1):1634. https://doi.org/10.1038/s41467-025-56594-3
12. Ferrari S, Savatin DV, Sicilia F, Gramegna G, Cervone F, De Lorenzo G. Oligogalacturonides: plant damage-associated molecular patterns and regulators of growth and development. Front Plant Sci. 2013;4:49. https://doi.org/10.3389/fpls.2013.00049
13. Del Corpo D, Coculo D, Greco M, De Lorenzo G, Lionetti V. Pull the fuzes: processing protein precursors to generate apoplastic danger signals for triggering plant immunity. Plant Commun. 2024;5 (8):100931. https://doi.org/10.1016/j.xplc.2024.100931
14. Yuan M, Jiang Z, Bi G, Nomura K, Liu M, Wang Y, et al. Pattern-recognition receptors are required for NLR-mediated plant immunity. Nature. 2021;592(7852):105–09. https://doi.org/10.1038/s41586-021-03316-6
15. Ngou BPM, Ahn HK, Ding P, Jones JD. Mutual potentiation of plant immunity by cell-surface and intracellular receptors. Nature. 2021;592 (7852):110–15. https://doi.org/10.1038/s41586-021-03315-7
16. Doehlemann G, Hemetsberger C. Apoplastic immunity and its suppression by filamentous plant pathogens. New Phytol. 2013;198 (4):1001–16.
17. Kalunke RM, Tundo S, Benedetti M, Cervone F, De Lorenzo G, D'Ovidio R. An update on polygalacturonase-inhibiting protein (PGIP), a leucine -rich repeat protein that protects crop plants against pathogens. Front Plant Sci. 2015;6:146. https://doi.org/10.3389/fpls.2015.00146
18. Almagro L, Gómez Ros LV, Belchi-Navarro S, Bru R, Ros Barceló A, Pedreño MA. Class III peroxidases in plant defence reactions. J Exp Bot. 2009;60(2):377–90. https://doi.org/10.1093/jxb/ern277
19. Daudi A, Cheng Z, O'Brien JA, Mammarella N, Khan S, Ausubel FM, et al. The apoplastic oxidative burst peroxidase in Arabidopsis is a major component of pattern triggered immunity. Plant Cell. 2012;24(1):275–87. https://doi.org/10.1105/tpc.111.093039
20. Van Der Hoorn RA, Kamoun S. From guard to decoy: a new model for perception of plant pathogen effectors. Plant Cell. 2008;20(8):2009–17. https://doi.org/10.1105/tpc.108.060194
21. Balasubramanian V, Vashisht D, Cletus J, Sakthivel N. Plant β-1,3-glucanases: their biological functions and transgenic expression against phytopathogenic fungi. Biotechnol Lett. 2012;34(11):1983–90. https://doi.org/10.1007/s10529-012-1012-6
22. Grover A. Plant chitinases: genetic diversity and physiological roles. Crit Rev Plant Sci. 2012;31(1):57–73. https://doi.org/10.1080/07352689.2011.616043
23. Di Matteo A, Bonivento D, Tsernoglou D, Federici L, Cervone F. Polygalacturonase-inhibiting protein (PGIP) in plant defence: a structural view. Phytochemistry. 2006;67(6):528–33. https://doi.org/10.1016/j.phytochem.2005.12.025
24. García-Olmedo F, Molina A, Alamillo JM, Rodríguez-Palenzuéla P. Plant defence peptides. Pept Sci. 1998;47(6):479–91. https://doi.org/10.1002/(SICI)1097-0282(1998)47:6<479::AID-BIP6>3.0.CO;2-K
25. Liu JJ, Sturrock R, Ekramoddoullah AK. The superfamily of thaumatin-like proteins: its origin, evolution and expression towards biological function. Plant Cell Rep. 2010;29(5):419–36. https://doi.org/10.1007/s00299-010-0826-8
26. Finkina EI, Melnikova DN, Bogdanov IV, Ovchinnikova TV. Plant pathogenesis-related proteins PR-10 and PR-14 as components of innate immunity system and ubiquitous allergens. Curr Med Chem. 2017;24 (17):1772–87. https://doi.org/10.2174/0929867323666161026154111
27. Cook DE, Mesarich CH, Thomma BP. Understanding plant immunity as a surveillance system to detect invasion. Annu Rev Phytopathol. 2015;53(1):541–63. https://doi.org/10.1146/annurev-phyto-080614-120114
28. Aguirre J, Ríos-Momberg M, Hewitt D, Hansberg W. Reactive oxygen species and development in microbial eukaryotes. Trends Microbiol. 2005;13(3):111–18. https://doi.org/10.1016/j.tim.2005.01.007
29. Heller J, Tudzynski P. Reactive oxygen species in phytopathogenic fungi: signalling, development and disease. Annu Rev Phytopathol. 2011;49(1):369–90. https://doi.org/10.1146/annurev-phyto-072910-095355
30. Chen LQ, Hou BH, Lalonde S, Takanaga H, Hartung ML, Qu XQ, et al. Sugar transporters for intercellular exchange and nutrition of pathogens. Nature. 2010;468(7323):527–32. https://doi.org/10.1038/nature09606
31. Fatima U, Senthil-Kumar M. Plant and pathogen nutrient acquisition strategies. Front Plant Sci. 2015;6:750. https://doi.org/10.3389/fpls.2015.00750

32. Chinchilla D, Zipfel C, Robatzek S, Kemmerling B, Nürnberger T, Jones JD, et al. A flagellin-induced complex of the receptor FLS2 and BAK1 initiates plant defence. Nature. 2007;448(7152):497–500. https://doi.org/10.1038/nature05999

33. Zipfel C, Oldroyd GE. Plant signalling in symbiosis and immunity. Nature. 2017;543(7645):328–36. https://doi.org/10.1038/nature22009

34. Miya A, Albert P, Shinya T, Desaki Y, Ichimura K, Shirasu K, et al. CERK1, a LysM receptor kinase, is essential for chitin elicitor signalling in Arabidopsis. Proc Natl Acad Sci U S A. 2007;104(49):19613–18. https://doi.org/10.1073/pnas.0705147104

35. Kadota Y, Sklenar J, Derbyshire P, Stransfeld L, Asai S, Ntoukakis V, et al. Direct regulation of the NADPH oxidase RBOHD by the PRR-associated kinase BIK1 during plant immunity. Mol Cell. 2014;54(1):43–55. https://doi.org/10.1016/j.molcel.2014.02.021

36. Asai T, Tena G, Plotnikova J, Willmann MR, Chiu WL, Gomez-Gomez L, et al. MAP kinase signalling cascade in Arabidopsis innate immunity. Nature. 2002;415(6875):977–83. https://doi.org/10.1038/415977a

37. Meng X, Zhang S. MAPK cascades in plant disease resistance signalling. Annu Rev Phytopathol. 2013;51:245–66. https://doi.org/10.1146/annurev-phyto-082712-102314

38. Dodds PN, Rathjen JP. Plant immunity: towards an integrated view of plant-pathogen interactions. Nat Rev Genet. 2010;11(8):539–48. https://doi.org/10.1038/nrg2812

39. Tsuda K, Katagiri F. Comparing signalling mechanisms engaged in pattern-triggered and effector-triggered immunity. Curr Opin Plant Biol. 2010;13(4):459–65. https://doi.org/10.1016/j.pbi.2010.04.006

40. Mur LA, Kenton P, Lloyd AJ, Ougham H, Prats E. The hypersensitive response; the centenary is upon us but how much do we know? J Exp Bot. 2008;59(3):501–20. https://doi.org/10.1093/jxb/erm239

41. Gust AA, Pruitt R, Nürnberger T. Sensing danger: key to activating plant immunity. Trends Plant Sci. 2017;22(9):779–91. https://doi.org/10.1016/j.tplants.2017.07.005

42. Couto D, Zipfel C. Regulation of pattern recognition receptor signalling in plants. Nat Rev Immunol. 2016;16(9):537–52. https://doi.org/10.1038/nri.2016.77

43. Kamoun S. A catalogue of the effector secretome of plant pathogenic oomycetes. Annu Rev Phytopathol. 2006;44(1):41–60. https://doi.org/10.1146/annurev.phyto.44.070505.143436

44. Torres MA, Jones JD, Dangl JL. Reactive oxygen species signalling in response to pathogens. Plant Physiol. 2006;141(2):373–78. https://doi.org/10.1104/pp.106.079467

45. Gilroy S, Suzuki N, Miller G, Choi WG, Toyota M, Devireddy AR, et al. A tidal wave of signals: calcium and ROS at the forefront of rapid systemic signalling. Trends Plant Sci. 2014;19(10):623–30. https://doi.org/10.1016/j.tplants.2014.06.013

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