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Small holes, big impact: Stomata in plant–pathogen–climate epic trifecta

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ABSTRACT

The regulation of stomatal aperture opening and closure represents an evolutionary battle between plants and pathogens, characterized by adaptive strategies that influence both plant resistance and pathogen virulence. The ongoing climate change introduces further complexity, affecting pathogen invasion and host immunity. This review delves into recent advances on our understanding of the mechanisms governing immunity-related stomatal movement and patterning with an emphasis on the regulation of stomatal opening and closure dynamics by pathogen patterns and host phytocytokines. In addition, the review explores how climate changes impact plant–pathogen interactions by modulating stomatal behavior. In light of the pressing challenges associated with food security and the unpredictable nature of climate changes, future research in this field, which includes the investigation of spatiotemporal regulation and engineering of stomatal immunity, emerges as a promising avenue for enhancing crop resilience and contributing to climate control strategies.

Key words: stomatal movement, stomatal patterning, plant resistance, pathogen virulence, apoplastic immunity, and climate change

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INTRODUCTION

More than 400 million years ago, green plants initiated their slow transition from aquatic environments to terrestrial habitats. While transitioning to the land, plant mesophyll cells remain immersed in the extracellular water-filled matrix and are shielded from the atmosphere by the water-proof cuticle layer. Essential inorganic minerals and water are absorbed by the root from the soil and transported through the vascular system to aboveground organs. In addition, plants meet their organic carbon demand by absorbing atmospheric carbon dioxide (CO_2) through specialized microscopic pores on the surface of leaves called stomata, which are surrounded by pairs of guard cells in the epidermis (Lawson and Matthews, 2020). Photosynthesis mediates the assimilation of carbon into organic compounds and the production of oxygen. Concurrently, water within plant tissues is evaporated through stomata due to a vapor pressure difference between plant leaves and the surrounding atmosphere, a phenomenon known as transpiration (Muller et al., 2017; Grossiord et al., 2020).

Plants regulate the stomatal movement, known as the aperture opening and closure, to maintain the trade-off between water conservation and carbon uptake for optimal growth. In ideal environmental conditions, stomatal movement is rhythmically regulated by light. During the day stomata open in response to light for the uptake of CO_2 and photosynthesis, while at night photosynthesis ceases, and stomata are closed to minimize water loss. However, the environment surrounding plants is constantly changing, often with combined variations in light, temperature, soil moisture levels (such as drought and flood), soil salinization and nutrient deficiencies, air humidity, and greenhouse gas concentrations, as well as attacks from pathogenic microbes and herbivorous insects (Zandalinas et al., 2021; Gao et al., 2023). Various phyllosphere microbes exploit stomata as the entrance to colonize in the apoplast, which is nutrient-rich compared with the atmosphere and leaf surface (Arnaud and Hwang, 2015; Melotto et al., 2017). These stresses could exert detrimental effects on plant growth, development, and overall productivity. Thus, plants have evolved layered mechanisms to respond to and adapt to these environmental challenges, and regulation of stomatal closure and opening is one of the key mechanisms (Figure 1).

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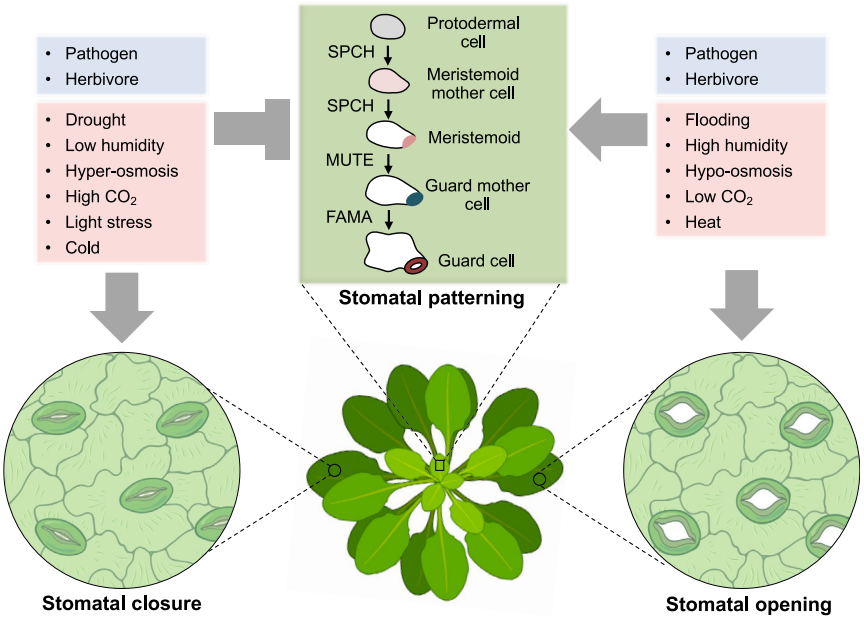

Figure 1. Dynamic regulation of stomatal aperture and patterning upon pathogen or herbivore attacks under changing climate conditions.

Pathogen infections and herbivore infestations exert profound influences on stomatal movement, encompassing closure and opening, with opposing actions during different infection phases. They also remodel stomatal patterning and density, which include the initiation of meristemoid mother cells (MMCs) and subsequent differentiation to guard cells. In addition, climate factors, including humidity, temperature, and greenhouse gas CO_2 induce adaptive changes in stomatal movement dynamics and patterning, enabling plants to cope with changing environmental conditions.

Moreover, stomatal patterning, primarily referring to the density of stomata on the leaf surface, plays a vital role in shaping a plant's capacity to respond to changing environmental conditions (Figure 1). For example, an appropriate stomatal density is pivotal for plants to effectively balance water conservation with efficient photosynthesis. Furthermore, stomatal patterning can influence plant resistance to microbial and insect attacks, as a higher stomatal density may render them more susceptible to certain pathogens or pests (Dutton et al., 2019; Tateda et al., 2019). Over time, plants have evolved mechanisms to optimize their stomatal density in response to environmental cues, thereby enhancing their overall fitness and survival prospects.

Significant strides have been taken in recent years to unravel the intricate molecular mechanisms governing plant stomatal responses to environmental cues, with particular emphasis on their reaction to pathogen infestations. The dynamic modulation of the stomatal aperture during various stages of pathogen invasion closely correlates with a plant's susceptibility or resistance. In addition, research has shed light on the impact of climate alterations on stomatal responses, consequently influencing a plant's ability to fend off pathogens. In this review, we begin by offering an overview of stomatal movement and patterning. We then discuss the most recent advancements in elucidating the signaling pathways that coordinate dynamic stomatal responses to pathogens and herbivorous insects within the context of changing climate conditions. Finally, we explore the potential for augmenting crop disease resistance through the engineering of stomatal immunity.

OVERVIEW OF STOMATAL MOVEMENT AND PATTERNING

Turgor-driven stomatal movement

The opening and closure of stomata are achieved through alterations in the turgor pressure of guard cells, which are orchestrated by the movement of ions across their membranes.

Stomatal opening is initiated by an augmentation in the activity of plasma membrane H^+-ATPase, which induces membrane hyperpolarization, subsequently activating inward-rectifying K^+ (K^+_{in}) channels to facilitate K^+ influx. This influx of K^+, coupled with anion uptake, initiates water absorption and swells of guard cells, resulting in stomatal opening (Saito and Uozumi, 2019; Pantoja, 2021b) (Figure 2). Conversely, during stomatal closure, the suppression of H^+ extrusion, along with the activation of slow (S)- and rapid (R)-type anion channels, leads to depolarization of the guard cell membrane, facilitating the efflux of Cl^-, malate, and NO_3^- anions. Notably, the activation of the S-type anion channel SLOW ANION CHANNEL-ASSOCIATED 1 (SLAC1) and SLAC1 HOMOLOG 3 (SLAH3) in guard cells plays a pivotal role in instigating stomatal closure (Figure 2) (Vahisalu et al., 2008). The membrane depolarization mediated by the active SLAC1 and SLAH3 triggers the activation of GUARD CELL OUTWARD RECTIFYING K^+ (GORK), a voltage-regulated outward-rectifying K^+ (K^+_{out}) channel, resulting in K^+ efflux (Horaruang et al., 2022). The subsequent loss of solutes culminates in water efflux from guard cells through the PLASMA MEMBRANE INTRINSIC PROTEIN 2;1 (PIP2;1), reducing their turgor pressure and constricting the stomatal pore, leading to stomatal closure (Figure 2) (Rodrigues et al., 2017).

At the subcellular level, ion transport systems are present at both the plasma membrane and the endomembrane (Saito and Uozumi, 2019). Significant progress has been achieved in characterizing transport systems at the tonoplast membrane and understanding the interplay between vacuolar- and plasma membrane-resident transport networks (Eisenach and De Angeli, 2017; Cubero-Font and De Angeli, 2021). For instance, it has been proposed that cytosolic malate plays a role in coordinating ion fluxes between the vacuolar and plasma membrane to induce stomatal movement through direct activation of ALUMINIUM-ACTIVATED MALATE TRANSPORTER (ALMT), a family of transporters involved in the transport of malate (Eisenach et al., 2017; Wang et al., 2018a).

Furthermore, the flow of water across the plasma membrane plays an important role in governing turgor-driven stomatal

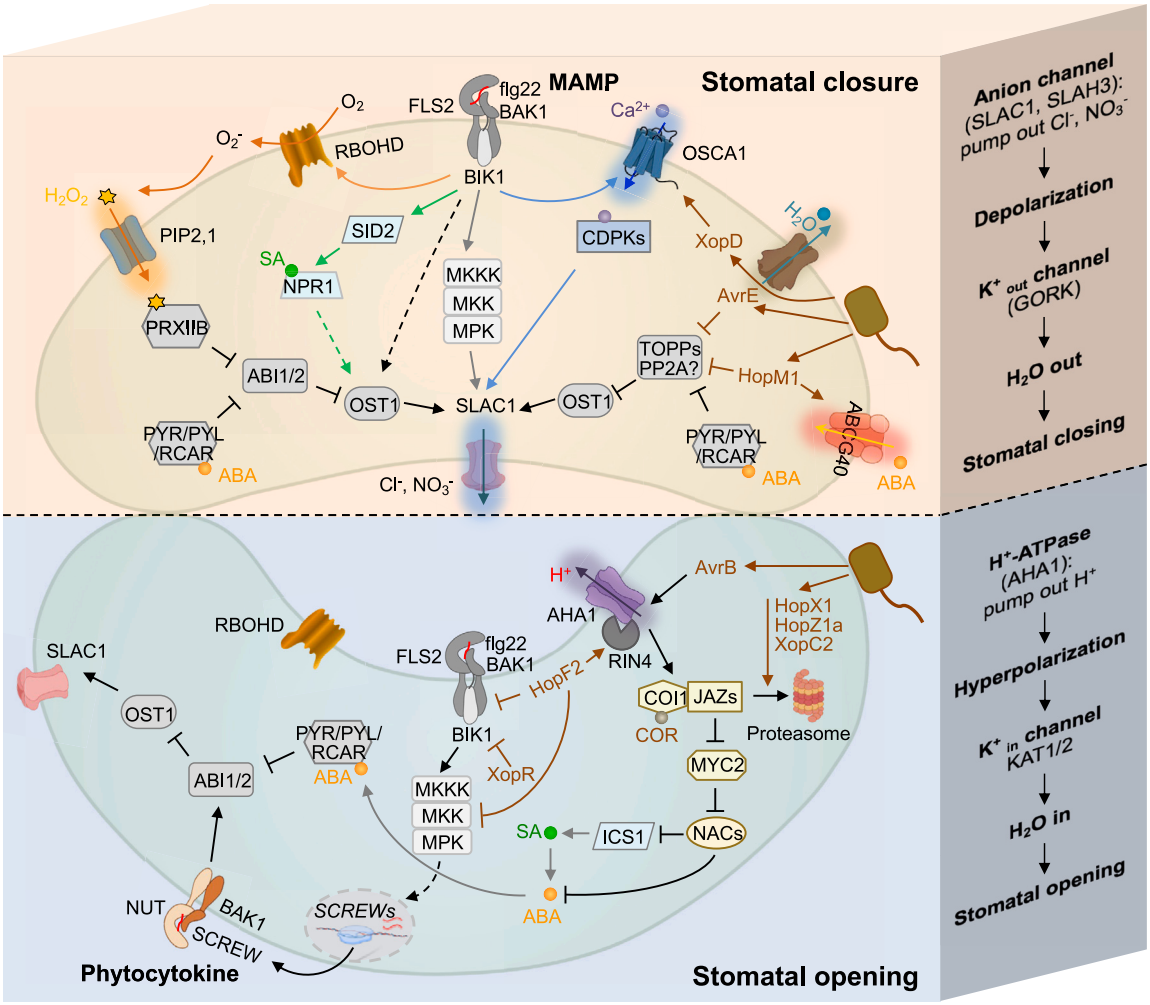

Figure 2. The signaling pathways that regulate stomatal opening and closure during pathogen infections.
The upper guard cell illustrates the signaling pathways involved in stomatal closure in response to infections, with a focus on plasma membrane depolarization and channel activities. MAMP perception (e.g., flg22 perception by FLS2-BAK1) triggers downstream signaling events, including BIK1 phosphorylation, Ca^{2+} influx, ROS burst, and MAPK activation. BIK1 activates NADPH oxidase RBOHD for the apoplast ROS burst. Aquaporin PIP2;1 can facilitate H_2O_2 transport from the apoplast to the cytoplasm, where peroxiredoxin PRXIIB acts as a sensor of H_2O_2, resulting in the oxidation and inactivation of ABI2, leading to the activation of the OST1 kinase and the opening of anion channel SLAC1, ultimately promoting stomatal closure. In addition, BIK1 activates Ca^{2+} channel OSCA1.2, contributing to cytosolic Ca^{2+} increase in promoting stomatal closure. CDPKs sense cytosolic Ca^{2+} increase, subsequently activating SLAC1 to close stomata. MAMP-induced stomatal closure also requires the SID2- and NPR1-mediated SA pathway. Pathogen effectors HopM1 and AvrE hijack ABA signaling components TOPPs, and ABA transporter ABCG40, respectively. Furthermore, XopD increases the *OSCA1.2* expression, further attributing to stomatal closure. In addition, AvrE functions as a water and solute channel to facilitate stomatal closure. The lower guard cell depicts the signaling pathways regulating stomatal opening during infections. MAMP perception induces the expression and production of phytocytokines (e.g., SCREWs perceived by NUT) in guard cells to counteract MAMP- and ABA-induced stomatal closure via enhancing ABI phosphatase activities and subsequently suppressing OST1-mediated activation of SLAC1, leading to stomatal re-opening for apoplastic immunity. Pathogen phytotoxin (e.g., COR perceived by COI1) activates JA signaling for inducing stomatal opening and effectors HopX1, HopZ1a, and XopC2 promote JAZ degradation and JA signaling for stomatal opening. AvrB and HopF2 target RIN4 to activate H^+-ATPase AHA1. XopR targets BIK1 to inhibit RBOHD-mediated ROS production and promotes stomatal opening.

movement (Roelfsema and Hedrich, 2005). Notably, the aquaporin PIP2, crucial for maintaining plant water balance by regulating hydraulic properties of cells and tissues, is implicated in the regulation of stomatal closure (Grondin et al., 2015; Rodrigues et al., 2017). An important inquiry pertains to the role of tonoplast-localized aquaporins in modulating water transport between the tonoplast and plasma membrane, as well as their influence on stomatal movements.

Moreover, the plasticity of plant cell walls and the internal compartments within guard cells is intricately intertwined with turgor pressure and functions as biomechanical drivers of stomatal movement (Keynia et al., 2023; Mirasole et al., 2023). Studies combining genetic and physiological approaches described that the degradation of pectin through PECTATE LYASE LIKE 12 (PLL12) and POLYGALACTURONASE INVOLVED IN EXPANSION 3 (PGX3) is involved in the cell wall-related stomatal movement (Rui et al., 2017; Chen et al., 2021). During the process

of stomatal closure, the vacuole within plant cells undergoes fragmentation into smaller vesicles. In contrast, when stomata are open, the vacuole typically occupies a significant portion of the cell volume as a single compartment. This dynamic behavior of the vacuole plays a critical role in regulating stomatal aperture, water movement, and ion accumulation in plants (Andres et al., 2014; Cao et al., 2022). In a recent study employing a genetically encoded biosensor to monitor the dynamic changes in ionic concentration of Cl^- and pH, along with volumetric alterations in subcellular compartments within living guard cells during fusicoccin-induced stomatal opening, it was revealed that the primary drivers of stomatal opening are the morphological and volume modifications of the vacuole (Mirasole et al., 2023).

Regulation of stomatal movement

The control of stomatal movement is orchestrated by environmental cues that trigger diverse signaling pathways. These include the plant hormones abscisic acid (ABA), salicylic acid (SA), and jasmonic acid (JA), as well as the second messengers, such as reactive oxygen species (ROS) and cytosolic calcium. These signaling networks play a pivotal role in governing the activation and deactivation of ion channels, thereby regulating the opening and closure of stomata (Murata et al., 2015; Hsu et al., 2021).

The hormone ABA is a core regulator of stomatal movement. ABA is synthesized in response to a range of stresses, such as drought, decreased atmospheric humidity, and extreme temperatures, across various tissues in plants. It is then transported through xylem and exported from the vasculature by ATP-binding cassette (ABC) transporter ABCG25, and subsequently imported into guard cells by another ABC transporter ABCG40 (Figure 2) (Zhang et al., 2023). ABA can also be synthesized in guard cells in response to low atmospheric humidity (Bauer et al., 2013). In the absence of ABA, type 2C protein phosphatases (PP2Cs) ABA-INSENSITIVE 1 (ABI1) and ABI2 interact with and inhibit the activity of OPEN STOMATA 1 (OST1)/SNF1-RELATED PROTEIN KINASE 2.6 (SnRK2.6) in guard cells. Binding of ABA to its receptors PYRABACTIN RESISTANCE 1/PYRABACTIN RESISTANCE 1-LIKE/REGULATORY COMPONENT OF ABA RECEPTOR (PYR/PYL/RCAR) induces conformational changes, enabling binding and inhibiting the PP2C activities. Inhibition of PP2Cs leads to the activation of OST1, which, in turn, phosphorylates and activates SLAC1 and SLAH3 ion channels, ultimately leading to stomatal closure (Cutler et al., 2010; Hsu et al., 2021).

SA and JA, two key hormones involved in the regulation of plant resistance to biotic stresses, also play critical roles in the regulation of stomatal movement (Melotto et al., 2017). SA mainly confers plant resistance to biotrophic and hemibiotrophic pathogens. Pathogen invasions elevate SA synthesis primarily through ISOCHORISMATE SYNTHASE 1 (ICS1). The synthesized SA is recognized by its receptor, NONEXPRESSOR OF PATHOGENESIS-RELATED GENES 1 (NPR1) to initiate downstream defense responses (Peng et al., 2021; Jia et al., 2023). The deficiency of genes involved in SA biosynthesis and signaling, including *ICS1* and *NPR1*, compromises pathogen-induced stomatal closure (Melotto et al., 2006; Zeng and He, 2010; Zeng et al., 2011), suggesting that SA is required for stomata-mediated plant resistance to pathogens. Similar to ABA, the exogenous application of SA activates SLAC1 and induces stomatal closure (Khokon et al., 2017; Prodhan et al., 2018). SA-induced stomatal closure remains intact in the *ost1-3* mutant in which ABA-induced stomatal closure is disrupted (Prodhan et al., 2018). In contrast, ABA-induced stomatal closure is compromised in *ics1*, a mutant with significantly impaired SA biosynthesis (Melotto et al., 2006; Zeng and He, 2010). Therefore, it is proposed that SA acts upstream of and integrates with ABA to trigger stomatal closure (Zeng and He, 2010; Prodhan et al., 2018). JA is mainly implicated in plant response to necrotrophic pathogens and herbivores. JA is perceived by the CORONATINE-INSENSITIVE 1 (COI1)-JASMONATE ZIM-DOMAIN (JAZ) coreceptor complex and regulates downstream defense responses through a master transcriptional factor MYC2 (Howe et al., 2018). Contrary to SA and ABA, JA induces stomatal opening through its active analog, JA-isoleucine (JA-Ile) (Melotto et al., 2017). JA likely antagonizes ABA and SA in regulating stomatal movement (Zheng et al., 2012).

ROS is a crucial early signal in the regulation of stomatal closure in response to various stresses. Biotic and abiotic stresses, including drought, high salinity, and pathogens, induce ROS production in multiple cellular compartments (Castro et al., 2021). ROS production in the apoplast is mainly catalyzed by a group of NADPH oxidases, known as RESPIRATORY BURST OXIDASE HOMOLOG (RBOH) proteins. RBOHD and RBOHF are responsible for ABA-induced ROS production in guard cells (Kwak et al., 2003). ABA stimulates ROS production through OST1-mediated phosphorylation of RBOHs to induce stomatal closure (Qi et al., 2018). ABA-induced stomatal closure is impaired in *rbohd/f* mutants, whereas H_2O_2-induced stomatal closure is not affected in *ost1* mutants (Mustilli et al., 2002). These results suggest that ROS acts downstream of OST1 in ABA-induced stomatal closure. Apoplastic ROS can be perceived by plasma membrane-resident receptors and initiates downstream signaling essential for inducing stomatal closure (Escocard de Azevedo Manhaes et al., 2021). The plasma membrane-localized receptor-like kinase GUARD CELL HYDROGEN PEROXIDE-RESISTANT1 (GHR1) mediates H_2O_2-induced SLAC1 activation and stomatal closure (Hua et al., 2012). The receptor-like kinase H_2O_2-INDUCED CA^{2+} INCREASES 1 (HPCA1) is an H_2O_2 sensor mediating H_2O_2-induced activation of Ca^{2+} channels in guard cells and is required for stomatal closure and the propagation of systemic ROS signals (Wu et al., 2020; Fichman et al., 2022). In addition, apoplastic ROS regulates stomatal closure when it is transported into the cytoplasm through aquaporin proteins. For example, the aquaporin PIP1;4 is required for H_2O_2 transportation and ABA-induced stomatal closure in *Arabidopsis* (Rodrigues et al., 2017). In the cytoplasm, H_2O_2 is sensed by intracellular sensors, which transmit H_2O_2 signaling through a redox relay process (Delaunay et al., 2002). It has been reported that the *Arabidopsis* thiol peroxidases as intracellular H_2O_2 sensors regulate stomatal closure by coupling ABA signaling pathway (Miao et al., 2006; Bi et al., 2022).

Diverse environmental stimuli induce Ca^{2+} influx through plasma membrane-resident Ca^{2+}-permeable channels. Several types

of Ca^{2+} channels have been identified. Of these, CYCLIC NUCLEOTIDE-GATED CHANNEL (CNGC) and HYPEROSMOLALITY-GATED CALCIUM-PERMEABLE CHANNEL (OSCA) families have been reported to be involved in the regulation of ABA and pathogen-induced stomatal closure (Tian et al., 2020; Xu et al., 2022). Elevated cytosolic Ca^{2+} attenuates K^+_{in} channels but activates anion channels, leading to massive ion efflux and ultimate stomatal closure (Pantoja, 2021a). Cytosolic Ca^{2+} also binds and activates intracellular Ca^{2+} sensors, including CALCIUM-DEPENDENT PROTEIN KINASEs (CDPKs) and CALCINEURIN B-LIKE (CBL)-CBL-INTERACTING PROTEIN KINASEs (CIPKs), which regulate stomatal movement by activating S-type anion channels or prompting RBOH-mediated ROS production (Qi et al., 2018; Tian et al., 2020).

Stomatal patterning

Stomatal pores, enclosed by a pair of guard cells, are flanked by at least one non-stomatal epidermal cell (Zuch et al., 2022). These structures are initiated from protodermal cells, which undergo asymmetric and symmetric cell divisions in the early stages of leaf development. Meristematic protodermal cells transit to meristemoid mother cells (MMCs), which differentiate into meristemoids, and subsequently give rise to guard mother cells (GMCs) followed by a symmetric division to form a pair of guard cells (Figure 1).

The morphogenesis of stomata is mainly regulated by three basic helix-loop-helix family transcription factors, SPEECHLESS (SPCH), MUTE, and FAMA. SPCH promotes the differentiation of protodermal cells into MMCs and subsequent asymmetric division, MUTE regulates the transition from meristemoid to GMCs, and FAMA controls the final cell division of GMC into a pair of guard cells (Figure 1). These transcriptional factors function downstream of peptide-receptor kinase signaling cascades. EPIDERMAL PATTERNING FACTOR (EPF)1/2 peptides recognized by the complex consisting of ERECTA (ER) receptors and SOMATIC EMBRYOGENESIS RECEPTOR-LIKE KINASE (SERK) and TOO MANY MOUTHS (TMM) coreceptors activate a MITOGEN-ACTIVATED PROTEIN KINASE (MAPK) cascade, which phosphorylates and inhibits SPCH to restrict stomatal development. EPF9, also known as STOMAGEN, antagonizes the EPF1/2-mediated signaling and positively regulates stomatal development (Guo et al., 2021; Zuch et al., 2022).

Stomatal development can be influenced by various environmental factors (Casson and Gray, 2008; Han et al., 2021; Matkowski and Daszkowska-Golec, 2023). Similar to the dynamics of stomatal movement, reshaping stomatal patterning and density enables plants to adjust their transpiration rate, CO_2 uptake, and resistance to pathogens, allowing them to adapt to changing environmental conditions (Figure 1).

DYNAMIC STOMATAL RESPONSES IN PLANT IMMUNITY

Overview of plant immunity

Plant immunity was mainly classified as pattern-triggered immunity (PTI) and effector-triggered immunity (ETI) (Zhang et al., 2020; Ngou et al., 2022). PTI is initiated when plasma membrane-localized pattern recognition receptors (PRRs) perceive microbial molecules known as pathogen/microbe-associated molecular patterns (PAMPs/MAMPs). This perception activates downstream signaling events and ultimately leads to the restriction of pathogen invasions (DeFalco and Zipfel, 2021; Ge et al., 2022). In addition, plants can activate PTI-like responses by perceiving self-derived damage-associated molecular patterns (DAMPs) and extracellular immunomodulatory peptides, known as phytocytokines. The immune signaling pathway, defined as DAMP-triggered immunity (DTI), is proposed to amplify or coordinate with the canonical PTI signaling for a robust immune response (Gust et al., 2017; Hou et al., 2021; Tanaka and Heil, 2021; Rzemieniewski and Stegmann, 2022).

On the other hand, ETI is predominantly activated by NUCLEOTIDE-BINDING DOMIAN LEUCINE-RICH REPEAT (NLR) proteins, which recognize pathogen effector proteins and trigger signaling cascades that often lead to a hypersensitive response (HR), a type of localized programmed cell death limiting pathogens at the site of infection (Cui et al., 2015; Lolle et al., 2020). Recognition of pathogen effector proteins often triggers NLR oligomerization and formation of large protein complexes with other partner proteins, forming resistosomes (Bi and Zhou, 2021; Hu and Chai, 2023; Wang et al., 2023). Resistosomes regulate immune signaling through their activities as Ca^{2+}-permeable influx channels, NADases, or 2′,3′-cAMP/cGMP synthetase activities (Chai et al., 2023; Huang et al., 2023). Local induction of plant PTI and ETI upon pathogen infection often triggers resistance to subsequent pathogen attacks in distal tissues, a phenomenon called systemic acquired resistance (SAR) (Zhang et al., 2020).

PTI and ETI are not mutually exclusive and can synergistically integrate, resulting in an effective defense (Yuan et al., 2021; Ngou et al., 2022). Conversely, the disruption of PTI through the perturbation of PRR coreceptors, such as BRASSINOSTEROID-INSENSITIVE 1 (BRI1)-ASSOCIATED RECEPTOR KINASE 1 (BAK1) and its close homolog SERK4, or a PRR-activated MAPK cascade, can initiate NLR-dependent ETI, leading to cell death and autoimmunity (Zhang et al., 2012; Liu et al., 2020a; Schulze et al., 2022; Yang et al., 2022; Yu et al., 2023). Consequently, PTI and ETI are interdependently regulated, and the immune system is under tight control to maintain a balance of cellular and immune homeostasis.

Initial stomatal closure to restrict pathogen entry

Most plant pathogens can bypass the epidermal barrier and exploit stomatal pores as entry points to invade the leaf apoplast. As a defense mechanism, upon pathogen sensing, stomata are rapidly closed as early as 30 min to restrict subsequent pathogen entry, which is termed stomatal immunity (Melotto et al., 2017). Studies indicated that the initial stomatal closure is induced by the perception of MAMPs, DAMPs, and phytocytokines (Figure 3).

Rapid stomatal closure upon perception of MAMPs

Numerous MAMPs, including bacterial flagellin, lipopolysaccharide, elongation factor-Tu (EF-Tu), fungal chitin, oomycete necrosis, and ethylene-inducing peptide 1 (Nep1)-like proteins (NLPs), as well as yeast elicitor (YEL), have been shown to induce stomatal closure (Figure 3A) (Arnaud and Hwang, 2015; Melotto et al., 2017). Flagellin is a component of bacterial flagella

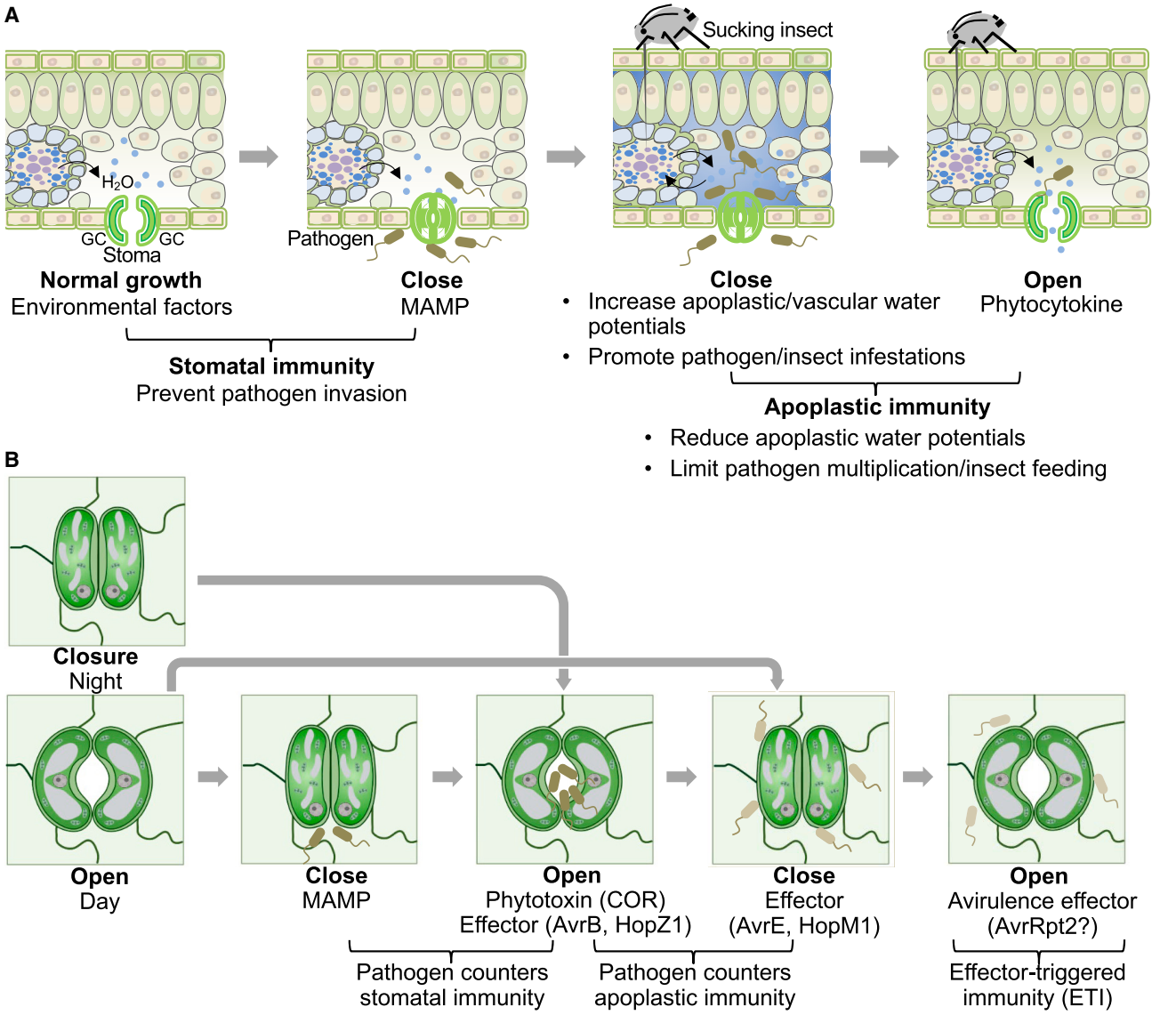

Figure 3. Counter-regulation of stomatal movement during plant–pathogen/insect interactions.

(A) Plants trigger stomatal and apoplast immunity in fending off pathogen infections and sap-sucking insect infestations. In the pre-invasion phase, stomatal guard cells (GCs) sense MAMPs via PRRs in initiating PTI and resulting in stomatal closure. This closure restricts pathogen invasions, referred to as stomatal immunity. Closed stomata increase apoplast/vascular water potentials, creating a conductive environment for pathogen and sap-sucking insect infestations. To counteract this, in the post-invasion phase, PTI induction triggers the expression of phytocytokines, promoting stomatal re-opening. This leads to water loss from the apoplast and limits pathogen proliferation in the apoplast, termed apoplastic immunity. Stomatal opening reduces water potential in vascular tissues, hindering the feeding of sap-sucking insects like aphids.

(B) Pathogens dynamically counter-regulate stomatal movement. In response to MAMP-induced stomatal immunity, pathogens deploy phytotoxins (e.g., COR) and effector proteins (e.g., AvrB and HopZ1) to reopen stomata, allowing entry into the apoplast for colonization. This action suppresses MAMP-induced stomatal immunity. Some pathogen effector proteins and phytotoxins may induce stomatal opening to promote invasion through stomata in the dark. In the post-invasion phase, pathogens utilize additional effector proteins, including HopM1 and AvrE, to induce stomatal closure, creating a favorable aqueous environment in the apoplast. This could also be a mechanism to counteract phytocytokine-mediated apoplastic immunity. Some effectors could be recognized by plant NLR immune receptors and function as avirulence proteins that could potentially suppress effector-triggered stomatal closure to release water from the apoplast.

required for bacterial movement toward stomata. The release of immunogenic fragments from flagellin is necessary for the cognate receptor FLAGELLIN-SENSING 2 (FLS2)-mediated stomatal immunity. In *Arabidopsis*, the apoplastic galactosidase BETA-GALACTOSIDASE 1 (BGAL1) and unknown protease(s) hydrolyze flagellin, releasing immunogenic fragments (Buscaill et al., 2019). Most plants can perceive flg22, a highly conserved 22-amino-acid epitope of flagellin. However, some bacteria can evade plant perception through variations in the flg22 sequence (Sanguankiattichai et al., 2022). In addition to evading FLS2 recognition, some flg22 variants from commensal bacteria can act as antagonists of immunogenic flg22 peptide to suppress FLS2 activation (Colaianni et al., 2021; Parys et al., 2021).

Meanwhile, plants have evolved variants of FLS2 to recognize these polymorphic peptides. As an illustration, soybean (*Glycine max*) possesses *GmFLS2* homologs capable of recognizing the flg22 epitope derived from *Ralstonia solanacearum*, an antigen typically undetected by most other plant species (Wei et al., 2020). Furthermore, the riverbank grape (*Vitis riparia*) has evolved *Vr*FLS2-sensing flg22 from *Agrobacterium tumefaciens*, which is typically unrecognized by the majority of other plants (Furst et al., 2020). Moreover, some *Solanaceous* plants, including tomatoes, potatoes, and peppers, perceive flgII-28, another epitope of flagellin (Hind et al., 2016). In tomatoes, the perception of flg22 and flgII-28 by the cognate receptors FLS2 and FLS3, respectively, triggers stomatal closure (Hind et al., 2016; Roberts et al., 2020). Clearly, bacterial flagellin and its FLS2 receptor take center stage in the ongoing battlefield of plant–microbe interactions with a dual role, serving as tools for bacterial movement toward stomata to bolster virulence, while also acting as triggers that initiate stomatal closure and activate plant defense mechanisms.

The perception of flg22 by FLS2 recruits coreceptors BAK1 and SERK4, and subsequently induces the phosphorylation of the BOTRYTIS-INDUCED KINASE 1 (BIK1) family of RECEPTOR-LIKE CYTOPLASMIC KINASEs (RLCKs) (Yu et al., 2017; DeFalco and Zipfel, 2021). BIK1 phosphorylates the permeable Ca^{2+} channel OSCA1.3 and consequently activates the channel to mediate Ca^{2+} influx, leading to stomatal closure (Thor et al., 2020). The phosphorylation of OSCA1.3 is required for flg22-induced, but not ABA-induced, stomatal closure (Thor et al., 2020), indicating a specific role of the OSCA1.3 channel in stomatal immunity (Figure 2). In addition, BIK1 also phosphorylates and activates CNGC2 and CNGC4 to mediate flg22-induced cytoplasmic Ca^{2+} concentration ($[Ca^{2+}]_{cyt}$) elevation in the seedlings (Tian et al., 2019).

BIK1 also directly phosphorylates the NADPH oxidase RBOHD that mediates the flg22-induced apoplast ROS burst (Kadota et al., 2014; Li et al., 2014). ROS are the essential molecules in triggering stomatal closure (Castro et al., 2021). Consequently, the *bik1* mutant has a defect in MAMP-triggered stomatal immunity (Kadota et al., 2014; Li et al., 2014). As unique Ca^{2+} sensor protein kinases, CDPKs are activated upon flg22 perception and phosphorylate RBOHD for ROS production (Boudsocq et al., 2010; Dubiella et al., 2013; Gao et al., 2013). It remains interesting whether CNGC/OSCA1.3-mediated Ca^{2+} influx and RBOHD-mediated ROS burst function independently or cooperatively in mediating stomatal immunity. In addition, BIK1 phosphorylates EXTRA-LARGE G PROTEIN 2 (XLG2), which further activates RBOHD for ROS burst, contributing to stomatal closure (Liang et al., 2016). Moreover, MAMP-activated MAP kinase cascade, including MPK3/MPK6 and their upstream kinases MKK4/MKK5, induces stomatal closure through the regulation of the metabolism of osmotically active organic acids, such as malate and citrate (Su et al., 2017). Therefore, the regulation of stomatal immunity involves the coordination of multiple PTI components to establish a resilient defense mechanism.

The regulation of ABA on MAMP-induced stomatal closure has been widely studied (Melotto et al., 2017). FLS2 activation does not increase the level of ABA, but a reduced ABA level in ABA-deficient mutants compromises flg22-induced stomatal closure (Melotto et al., 2006; Du et al., 2014). Apparently, the basal level of ABA is essential for MAMP-induced stomatal closure. Notably, flg22 perception can activate SLAC1 and SLAH3 channel activities, and loss of SLAC1/SLAH3 anion channels or OST1 abolishes flg22-induced stomatal closure (Melotto et al., 2006; Guzel Deger et al., 2015) (Figure 2). By contrast, flg22-induced stomatal closure remains intact in the *abi1-1* mutant, in which ABI1 is constitutively active and the ABA-induced activation of SLAC1/SLAH3 is disrupted (Guzel Deger et al., 2015). These results hint that FLS2 and ABA signaling pathways may converge at OST1 to activate SLAC1- and SLAH3-mediated stomatal closure.

Some fungi also penetrate through stomata to invade plant tissues. Chitin, the fungal cell wall component could be degraded by plant chitinase into chitin oligosaccharides, which are perceived by plant lysin M receptor-like kinases, including CHITIN ELICITOR RECEPTOR KINASE 1 (CERK1), as MAMPs to trigger stomatal closure and guard cell death (Ye et al., 2020). RLCK PBS1-LIKE 27 (PBL27) downstream of CERK1 directly phosphorylates and activates the SLAH3 anion channel activities in triggering stomatal closure (Liu et al., 2019). Likely as a counter-defense strategy, some fungi secrete chitin deacetylases to convert chitin to chitosan oligosaccharides (Lopez-Moya et al., 2019). Chitosan oligosaccharides are not perceived by chitin receptors and do not induce stomatal closure (Ye et al., 2020), implying an elegant strategy that fungi use to evade stomatal immunity. Consistently, chitin deacetylases were reported to be required for fungal pathogenicity (Cord-Landwehr et al., 2016; Gao et al., 2019; Xu et al., 2020).

Stomatal closure in response to DAMPs and phytocytokines

Infection by pathogens or the perception of MAMPs leads to the production of DAMPs, which instigate DTI to either amplify or compensate for plant immunity (Hou et al., 2019b; Tanaka and Heil, 2021). DAMPs, including extracellular ATP (eATP), cell wall-derived polysaccharide fragments, and certain phytocytokines, have demonstrated the ability to induce stomatal closure. However, given that DAMP production typically occurs after the initial pathogen entry, the physiological significance of DAMP-induced stomatal closure remains uncertain. Two potential explanations arise: DAMPs may either enhance the stomatal response to MAMPs or serve as a contingency mechanism for stomatal closure if MAMP signaling is compromised by pathogens.

eATP, which is detected by the L-type lectin receptor-like kinase LecRKI.9/DOES NOT RESPOND TO NUCLEOTIDES 1 (DORN1), triggers stomatal opening and closure in a concentration-dependent manner. At higher concentrations, typically falling within the range of 150–250 μM, eATP elicits stomatal closure (Clark et al., 2011), whereas at lower concentrations ranging from 5 to 15 μM eATP prompts stomatal opening (Clark et al., 2011; Hao et al., 2012; Chen et al., 2017). The precise mechanism behind this opposing effect of eATP at different concentrations remains uncertain. Nonetheless, it has been demonstrated that the eATP-induced stomatal closure relies on RBOHD-mediated ROS production, rather than ABA perception and signaling (Chen et al., 2017). A notable

increase in the eATP levels surrounding guard cells has been observed in response to pathogen infection, although the precise concentration of eATP remains unclear (Chen et al., 2017). Interestingly, APY1 and APY2, two apyrases (nucleoside triphosphate diphosphohydrolase, NTPDase) responsible for regulating eATP levels, exhibit robust expression in guard cells (Clark et al., 2011). This observation implies that plants possess the capability to actively modulate stomatal immunity by regulating eATP concentrations. Exploring the fluctuation in eATP levels during pathogen infections could provide valuable insights into the mechanisms underlying variations in stomatal responses to eATP based on its concentration. Moreover, such investigations may shed light on whether the promotion of stomatal opening by lower concentrations of eATP during specific stages of invasion contributes to plant resistance or is manipulated by pathogens to promote their entry.

Certain phytocytokines induce stomatal closure like MAMPs. For instance, the phytocytokine PAMP-INDUCED PEPTIDE 1 (PIP1), perceived by RECEPTOR-LIKE KINASE 7 (RLK7), initiates stomatal closure in an OST1- and SLAC1-dependent manner (Hou et al., 2019a; Shen et al., 2020). However, other phytocytokines might employ different mechanisms with flg22 when eliciting stomatal closure. One such phytocytokine, Peptide elicitor 1 (Pep1), perceived by two closely related receptor kinases PEPR1 and PEPR2, is released from its precursor PROPEP1 via Ca^{2+}-dependent metacaspase cleavages in response to wounding and microbial attack (Hander et al., 2019; Shen et al., 2019). Although Pep1 triggers stomatal closure through S-type anion channels, SLAC1 and SLAH3, unlike flg22, its activation of anion channels and subsequent stomatal closure do not rely on the ABA signaling component OST1 (Zheng et al., 2018). Furthermore, the secreted peptide PLANT NATRIURETIC PEPTIDE A (PNP-A) has been shown to regulate stomatal movement and enhance plant resistance to pathogens (Ficarra et al., 2018). PNP-A is a functional analog of natriuretic peptides regulating cellular water and ion homeostasis in vertebrates. In *Arabidopsis*, PNP-A enhances pathogen-induced stomatal closure, suggesting its role in priming stomatal closure (Ficarra et al., 2018). However, the precise molecular mechanisms governing PNP-A regulation of stomatal closure and its impact on pathogen resistance remain to be determined.

Stomatal reopening to promote apoplast immunity

Pathogens establish themselves within the apoplast once they gain entry into plant tissues through stomata. Research has shown that the presence of an aqueous environment within the leaf apoplast is conducive to pathogen colonization (Figure 3A) (Xin et al., 2016; Aung et al., 2018). However, stomatal closure, triggered after the initial pathogen recognition, serves the dual role of limiting pathogen entry and conserving moisture within the apoplast. This retained apoplastic moisture, in turn, fosters a favorable environment for bacterial colonization. In addition, apoplastic hydration can impact vascular water potential and hydrothode guttation, thereby influencing plant susceptibility to vascular pathogens and the feeding behavior of sucking insects (Erb and Reymond, 2019; Paauw et al., 2023). Hence, the reopening of stomata subsequent to pathogen-induced stomatal closure proves advantageous in sustaining normal physiological processes in plants and enhancing resistance to both pathogens and herbivorous insects (Figure 3A).

Phytocytokines promote stomatal reopening

The stomatal response to flg22 treatment is highly dynamic, typically characterized by an initial closure occurring within 2 h, followed by a subsequent reopening phage at around 3–4 h (Melotto et al., 2006; Liu et al., 2022c), suggesting that plants can employ a mechanism to reopen the closed stomata. A recent study has revealed that the SMALL PHYTOCYTOKINES REGULATING DEFENSE AND WATER LOSS (SCREWs) play a counter-regulatory role against flg22-induced stomata closure, aiming to disrupt the aqueous apoplast, which is conducive to pathogen manipulation (Liu et al., 2022c). SCREW1 and SCREW2, also known as CTNIP1 and CTNIP4, are rapidly induced upon flg22 perception and recognized by plasma membrane-localized receptor kinase PLANT SCREW UNRESPONSIVE RECEPTOR (NUT), also referred to as HAESA-LIKE 3, HSL3, along with the coreceptors BAK1 and SERK4 (Liu et al., 2022c; Rhodes et al., 2022). The SCREW-mediated reopening of closed stomata renders the flg22-induced stomatal closure a transient process (Liu et al., 2022c). Furthermore, SCREWs-NUT can counteract ABA-induced stomatal closure by enhancing the activity of protein phosphatase 2C ABI1 and ABI2 on OST1 and regulate drought stress responses (Liu et al., 2020b, 2022c) (Figure 2).

In line with the aforementioned findings, the rice ABA-deficient mutant *Osaba1* with an elevated stomatal conductance showed enhanced resistance to *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) (Zhang et al., 2019). This supports the notion that the opening stomata contribute to post-invasive resistance against bacterial pathogens. These studies broaden the concept of stomatal immunity, which goes beyond the function of closing stomata to prevent pathogen invasion. It encompasses the concept of reopening stomata to disrupt the formation of an aqueous apoplast, consequently restricting the proliferation of pathogenic bacteria and bolstering plant immunity (Figure 3). Apart from establishing an aqueous living space, manipulating nutrient release through effector proteins also contributes to pathogen proliferation in the apoplast (Gentzel et al., 2022). Whether SCREWs or other phytocytokines can modulate plant nutrient dynamics in response to pathogens remains a subject for future research.

In addition to invading through stomata and colonizing the apoplast, certain pathogens like some *Xanthomonas* and *Pseudomonas* strains can establish themselves within vascular tissues through hydathodes, which are pores located at the tips of vascular endings along the leaf margin and are responsible for plant guttation (Cerutti et al., 2019; Paauw et al., 2023). Hydathode and vascular immunity have also been shown to protect plants from pathogen colonization. Similar to stomata, hydathode pores are responsive to ABA and light (Cerutti et al., 2017). Some phytocytokines, such as PIP1 and SCREWs, are significantly expressed in vasculature and hydathodes (Hou et al., 2014; Liu et al., 2022c). Furthermore, the water potential within a plant's vascular system is precisely regulated through the coordinated action of stomata and

hydathodes (Bellenot et al., 2022). It will be interesting to determine whether peptide-receptor signaling is involved in the regulation of plant hydathode and vascular immunity against certain pathogens.

NLRs regulate stomatal opening

The occurrence of plant NLR-mediated ETI is often accompanied by HR, a unique process characterized by the rapid death and wilting of plant tissues. It has been proposed that the wilting of plant tissues during ETI might be linked to accelerated water loss through open stomata, in combination with disruptions in sap flow through vascular tissues to the infection sites (Freeman and Beattie, 2009).

While direct evidence supporting the activation of NLRs in triggering stomatal opening is currently lacking, some interesting observations have emerged. For instance, the *P. syringae* effector AvrRpt2, recognized by NLR RESISTANT TO *P. SYRINGAE* 2 (RPS2), has been shown to suppress *Pst* DC3000-induced apoplast water soaking, an early symptom of infected leaves with an increased water level in the apoplast (Xin et al., 2016; Hu et al., 2022; Roussin-Leveillee et al., 2022). In addition, the chemical compound DFPM ([5-(3,4-dichlorophenyl)furan-2-yl]-piperidine-1-ylmethanethione) has been known to inhibit ABA-induced stomatal closure. Notably, certain key ETI signaling components, including lipase-like proteins ENHANCED DISEASE SUSCEPTIBILITY 1 (EDS1) and PHYTOALEXIN DEFICIENT 4 (PAD4), as well as protein folding chaperone components such as SUPPRESSOR of G2 ALLELE of SKP1 b (SGT1b) and REQUIRED FOR MLA12 RESISTANCE 1 (RAR1), are essential for DFPM's ability to modulate ABA-regulated stomatal movement. This regulation encompasses both the induction of stomatal closure and the restraint of stomatal opening (Kim et al., 2011). These findings suggest a potential antagonistic relationship between NLR-induced stomatal opening and MAMP-induced stomatal closure. Similar to SCREW-NUT, NLR-induced stomatal opening likely serves as a defense mechanism to counteract the creation of an aqueous living space in the apoplast by virulent pathogens (Figure 3A and 3B) (Xin et al., 2016).

The regulation of stomatal movement by NLRs might involve complex mechanisms. The *snc1-1* mutant, which carries a constitutively active NLR SUPPRESSOR OF NPR1-1, CONSTITUTIVE 1 (SNC1), has been observed to inhibit ABA-triggered stomatal closure and enhance apoplastic defense against *Pst* DC3000 (Kim et al., 2011; Yan et al., 2019). SNC1 belongs to the Toll/Interleukin-1 Receptor (TIR)-type NLR (TNL) protein family. TIR domains possess NADase enzymatic activity, which is crucial for immune activation (Essuman et al., 2022). Emerging evidence suggests a connection between cytosolic NAD levels and ABA biosynthesis and signaling (Feitosa-Araujo et al., 2022). This connection may help explain the altered stomatal immunity observed in the *snc1-1* mutant. In addition, the *snc1-1* mutant also exhibits a significantly increased level of the defense hormone SA (Zhang et al., 2003). SA is shown to have an antagonistic effect with ABA in plant defense (Robert-Seilaniantz et al., 2011). It remains an open question whether SNC1's suppression of ABA-regulated stomatal closure functions through an antagonistic crosstalk between ABA and SA signaling pathways.

MANIPULATION OF STOMATAL DYNAMICS BY PATHOGEN TOXINS AND EFFECTORS

The successful invasion and colonization of the plant apoplast by pathogens involve sophisticated strategies, including the production of specific toxins and effector proteins that manipulate plant immunity (Wang et al., 2022). Some toxins and effectors can exert profound effects on stomatal behavior at various stages of infection, leading to dynamic changes in stomatal responses (Figure 3B). In the pre-invasion stage, certain toxins and effectors can suppress MAMP-induced stomata closure, allowing pathogens to overcome stomatal immunity and maintain access to the open stomata. In addition, some toxins and effectors can promote stomatal opening, creating a favorable environment for their entry into the plant. In the post-invasion stage, certain effectors can induce stomatal closure, potentially establishing a water-soaked apoplast, which provides a conducive environment for pathogen multiplication and spread (Figure 3B). The temporal-spatial secretion of pathogen effectors, partly assisted by chaperone proteins, and toxins during different infection stages may enable the dynamic regulations of plant stomatal movement while suppressing the plant immunity.

Pathogen toxins suppress MAMP-induced stomatal closure and promote stomatal opening

The polyketide phytotoxin coronatine (COR), produced by certain pathovars of *P. syringe*, plays a significant role in countering MAMP-induced stomatal closure in plants (Melotto et al., 2006). The COR biosynthesis gene is upregulated when *Pst* DC3000 is in contact with the leaf surface prior to its penetration into the leaves, suggesting that COR works in the pre-invasive phase (Panchal et al., 2016a). COR structurally mimics JA-Ile to interact with the COI1-JAZ coreceptor complex, resembling how JA-Ile operates in plants. Consequently, COR hijacks the plant's JA signaling pathway through the JA master regulator MYC2 and downstream transcriptional factor NACs, attributed to the suppression of SA biosynthesis and SA-mediated stomatal closure (Zheng et al., 2012). SA biosynthesis is essential and acts upstream of ABA to trigger stomatal closure (Figure 2) (Zeng and He, 2010). Correspondingly, COR also represses ABA-triggered stomatal closure through COI1 and NACs (Zheng et al., 2012). COR has also been suggested to promote stomatal opening through a pathway involving RPM1-INTERACTING 4 (RIN4), a protein that interacts with NLR proteins. This pathway may be related to changes in the plasma membrane's hyperpolarization, which can influence stomatal behavior (Melotto et al., 2017) (Figure 2).

The bacterial pathogen *X. campestris* pv. *campestris* (*Xcc*) manipulates stomatal closure in *Arabidopsis* by secreting a small molecule controlled by the *rpf/diffusible signal factor* (*DSF*) gene cluster. Similar to COR, this unknown molecule inhibits both MAMP- and ABA-induced stomatal closure, likely by targeting a component in the MPK3-mediated signaling pathway (Gudesblat et al., 2009).

Some toxins could directly induce stomatal opening to promote pathogenicity. For instance, syringolin A, a nonribosomal peptide, produced by certain stains of *P. syringae* can promote

stomatal opening, effectively counteracting stomatal immunity in beans and *Arabidopsis* (Schellenberg et al., 2010). Syringolin A functions as a proteasome inhibitor, blocking the proteasome-mediated turnover of NPR1 and thereby also suppressing SA signaling and MAMP-induced stomatal closure (Schellenberg et al., 2010; Misas-Villamil et al., 2013). Similarly, fusicoccin, a fungal phytotoxin produced by *Fusicoccum amygdale*, induces stomatal opening by activating H^+-ATPases via 14-3-3 proteins (Camoni et al., 2019).

Pathogen effectors disrupt MAMP-induced stomatal closure and promote stomatal opening

Some effector proteins secreted from pathogenic bacteria, such as *Pseudomonas* and *Xanthomonas* species, could disrupt stomatal immunity to facilitate pathogen entry (Figure 3B). For instance, *P. syringae* effector, HopF2, suppresses MAMP-induced stomatal closure. HopF2 possesses ADP-ribosyltransferase activity and can ADP-ribosylate MKK5 and RIN4 (Wang et al., 2010; Wilton et al., 2010). However, its ability to disrupt stomatal immunity is independent of its ADP-ribosyltransferase activity (Hurley et al., 2014). XopR, an effector from *Xcc*, suppresses MAMP-triggered stomatal closure by associating with BIK1 and inhibiting RBOHD-mediated ROS production in *Arabidopsis* (Wang et al., 2016). XopS from *X. campestris* pv. *vesicatoria* (*Xcv*) interferes with bacteria or MAMP-triggered stomatal closure in pepper by binding and stabilizing transcription factor WRKY40, which acts as a repressor in attenuating the induction of SA-responsive genes but enhances JA-mediated responses (Bascom, 2022). AvrRxo1 from *X. oryzae* pv. *oryzicola* (*Xoc*) impairs *Xoc*-induced stomatal closure by promoting the degradation of PYRIDOXAL PHOSPHATE SYNTHASE 1 (PDX1) in rice, a metabolic enzyme that catalyzes the synthesis of vitamin B6, and consequently interferes with ABA biosynthesis (Liu et al., 2022a). Hence, effectors display versatile modes of action in suppressing MAMP-induced stomatal closure.

Bacterial effectors could also directly promote stomatal opening, facilitating pathogen entry and infection. AvrB from *P. syringae* induces stomatal opening and enhances bacterial virulence by elevating the *Arabidopsis* plasma membrane H^+-ATPase (AHA1) activity through promoting RIN4-INTERACTING RECEPTOR-LIKE PROTEIN KINASE (RIPK)-mediated RIN4 phosphorylation (Lee et al., 2015). It was proposed that the phosphorylated RIN4 might regulate an unknown kinase activity to phosphorylate and activate AHA1 (Liu et al., 2011; Lee et al., 2015). AHA1 activation by AvrB promotes the COI1-JAZ interaction and JAZ degradation, leading to the activation of the JA signaling and consequent stomatal opening (Zhou et al., 2015b). HopX1 and HopZ1a from *P. syringae* with cysteine protease and acetyltransferase activities, respectively, can degrade JAZ proteins, leading to the activation of JA signaling and stomatal opening in *Arabidopsis* (Jiang et al., 2013; Gimenez-Ibanez et al., 2014). XopC2 from *Xoc* phosphorylates *Oryza sativa* SKP1-LIKE 1 (OSK1), an adaptor protein of the Skp1-Cullin-F-box ubiquitin ligase complex, to promote JAZ degradation, leading to the JA signaling activation and consequent stomatal opening (Wang et al., 2021). XopAP from *Xoc*, a putative lipase, induces stomatal opening by inhibiting vacuolar H^+-pyrophosphatase (V-PPase) through competitive binding to PtdIns(3,5)P2 (Liu et al., 2022b).

In summary, bacterial effectors have evolved diverse mechanisms to manipulate stomatal behavior, either by suppressing closure or actively inducing stomatal opening, ultimately facilitating pathogen entry and enhancing infection, showcasing the remarkable adaptability of pathogens in subverting plant defenses.

Pathogen effectors close stomata to establish an aqueous apoplastic habitat

Creating an aqueous living space by closing stomata is a crucial strategy employed by pathogen effectors to enable aggressive colonization within the plant phyllosphere (Xin et al., 2016; Aung et al., 2018). Pathogen effectors primarily manipulate plant ABA signaling to induce stomatal closure (Figure 2). HopM1 and AvrE from *P. syringae* induce stomatal closure through elevating ABA accumulation or/and signaling (Figure 2) (Hu et al., 2022; Roussin-Leveillee et al., 2022). HopM1 induces the expression of the guard-cell-specific ABA transporter ABCG40 (Roussin-Leveillee et al., 2022), leading to elevated ABA accumulation in guard cells, promoting stomatal closure (Roussin-Leveillee et al., 2022). It was shown previously that HopM1 induces apoplast water soaking through the degradation of HOPM1 INTERACTOR 7 (MIN7), an ADP ribosylation factor-guanine nucleotide exchange factor involved in vesicle trafficking (Xin et al., 2016). Notably, MIN7 degradation by HopM1 seems to be not related to the stomatal closure and ABA accumulation in guard cells (Roussin-Leveillee et al., 2022). This raises complex and multifaceted scenarios of additional factors being involved beyond MIN7 leading to the induction of water soaking in the apoplast.

AvrE, on the other hand, activates ABA signaling by targeting TYPE ONE PRTEIN PHOSPHATASEs (TOPPs) to de-repress OST1, resulting in ABA signaling activation (Figure 2) (Hu et al., 2022). WtsE, an AvrE-family effector from maize bacterial pathogen *Pantoea stewartia*, also targets the regulatory subunits of PHOSPHATASE 2A (PP2A) heterotrimeric enzyme complexes, key negative regulators in ABA signaling (Jin et al., 2016). Therefore, targeting PP2A could also be related to the activation of ABA signaling and the induction of stomatal closure by AvrE. Interestingly, WtsE induces apoplastic hydration and nutrient release from maize cells (Gentzel et al., 2022), potentially promoting increased water flow into the apoplast as water follows nutrient gradients. It was recently reported that AvrE-family effectors function as water/solute-permeable channels, through which cytoplasmic water/solute/nutrient flows to the apoplast to induce apoplastic hydration (Nomura et al., 2023) (Figure 2). Water/solute/nutrient outflow contributes to the reduction of turgor in guard cells and consequently promotes stomatal closure. It will be interesting to investigate whether the channel activity of AvrE is required for its regulation of stomatal closure.

HopAM1 from *P. syringae* promotes ABA-mediated stomatal closure in water-stressed plants (Goel et al., 2008). HopAM1 possesses a noncanonical TIR domain, which exhibits an enzymatic activity of hydrolyzing NAD^+ to produce nicotinamide and a cADPR variant (v2-cADPR) in plants (Eastman et al., 2022; Manik et al., 2022). The specific biochemical mechanism linking HopAM1-mediated NAD^+ metabolism, v2-cADPR production,

and plant sensitivity to ABA remains to be fully elucidated. In addition, XopD, a nuclear-targeted effector from *Xcc*, accelerates ABA-mediated stomatal closure by activating the expression of ABA-responsive genes and Ca^{2+}-permeable channel *OSCA1.1* (You et al., 2023). Apparently, pathogen effectors exploit various components in ABA-signaling-mediated stomatal closure to create a humid habitat that favors their growth (Figures 2 and 3).

MODULATION OF STOMATAL PATTERNING AND DEVELOPMENT BY PATHOGENS AND EFFECTORS

Pathogen infections modulate stomatal patterning and development

Stomata play a crucial role as potential entry points for pathogens, including fungi and bacteria, into plant tissues. Investigating the connection between a plant's resistance to foliar pathogens and stomatal density has unveiled intriguing insights into plant–pathogen interactions. Research on various plant species consistently demonstrates a strong correlation between stomatal density on leaves and a plant's vulnerability to pathogen infections. For instance, studies have revealed that leaves of *Gentiana trifolia* with lower stomatal density tend to exhibit slower rates of fungal *Septoria gentianae* infection. Conversely, leaves with higher stomatal density are more prone to increased incidence of infections (Tateda et al., 2019). *Arabidopsis* transgenic plants overexpressing EPF9/STOMAGEN with increased stomatal density are more susceptible, whereas those overexpressing EPF2 or EPFL7 with decreased stomatal density show enhanced resistance to *Pst* DC3000 by dip-inoculation (Dutton et al., 2019). Apparently, the relationship between stomatal density and the susceptibility of a plant to pathogen invasion is complex but generally exhibits a positive correlation.

Plants have evolved mechanisms to modulate stomatal density as a defense strategy. A study reported that infection with *Pst* DC3000 in *Arabidopsis* can lead to a reduction in stomatal density in newly developing leaves (Dutton et al., 2019). This suggests that plants can limit further pathogen invasion by systemically suppressing stomatal density, and a systemic signal transmits information from the infected tissue to emerging leaves to adjust stomatal development accordingly. This systemic response requires the perception of MAMP flg22 and the biosynthesis of SA in the infected mature leaves (Dutton et al., 2019). Interestingly, signals associated with SAR, such as L-pipecolic acid and azelaic acid, and lipid transfer protein DEFECTIVE IN INDUCED RESISTANCE 1 (DIR1), which is required for the generation and/or translocation of mobile SAR signal, do not appear to be critical for the systemic response regulating stomatal density (Dutton et al., 2019). This suggests that the systemic regulation of stomatal development as a defense against pathogens is independent of classical SAR. Recent findings have highlighted that the sugar levels in preexisting leaves play a pivotal role in shaping systemic stomatal development in newly emerging leaves (Bao et al., 2023). Notably, pathogens engage in competition with plants for access to sugars during the infection process (Cox et al., 2017; Chen et al., 2023). It is plausible that *Pst* DC3000 may influence systemic stomatal development by impacting local sugar status. Nevertheless, these findings highlight the sophisticated ways in which plants can regulate stomatal density as part of their defense mechanisms against pathogens.

The regulation of stomatal patterning is primarily governed by specific transcriptional processes during the early stages of leaf epidermal cell development. However, it is perplexing that transcript levels of genes typically associated with stomatal development, such as EPFs, SPEECHLESS, and MUTE, do not appear to be different in the systemic leaves (Dutton et al., 2019). This observation raises the possibility that post-transcriptional, translational, or post-translational regulations may play a role in governing this process. Different developmental stages of stomata in the same leaf and from various tissues may also exert additional layers of complexities for the role of stomata in plant–microbe interaction. Understanding how these regulations contribute to the dynamic regulation of stomata in response to pathogen challenges could shed light on previously uncharted aspects of plant defense strategies.

Pathogen effectors regulate stomatal patterning

The ectopic expression of *P. syringae* effector AvrPto and AvrPtoB in plants has been associated with the induction of stomatal clustering in *Arabidopsis* (Meng et al., 2015). This suggests that these effectors may play a role in promoting pathogenicity by modulating stomatal density and patterning. AvrPto and AvrPtoB target the BAK1/SERK coreceptors and disrupt ligand-induced receptor–coreceptor interaction (Shan et al., 2008; Zhou et al., 2014; Meng et al., 2015). BAK1/SERK4 are shared coreceptors of multiple receptor kinases, including ER family receptor kinases regulating stomatal patterning (Ma et al., 2016). Similarly, these effectors may block the ER-BAK1/SERK complex formation, leading to impaired ER signaling and uncontrolled stomatal patterning. The physiological significance of the stomatal clustering induced by AvrPto and AvrPtoB remains unclear. It could potentially be a side effect of their targeting BAK1/SERK4 coreceptors, or it might be a deliberate manipulation of stomatal density aimed at promoting virulence. Further research with some advanced techniques, such as optogenetics, that enables the control of the specific signaling sector, is needed to determine the exact role and importance of this phenomenon in plant–pathogen interactions.

STOMATAL DYNAMICS IN RESPONSE TO HERBIVOROUS INSECTS

The stomatal response to herbivores is a complex process with various mechanisms associated with the unique feeding behaviors of different insects. Some piercing-sucking herbivores, like phytophagous mites, spruce aphids, and lace bugs, feed on leaf mesophyll by inserting their stylets into plant tissues through stomata, which leads to excessive water loss from plants. Plants have developed intricate defense mechanisms against herbivorous insects while balancing their needs for gas exchange and water conservation (Lin et al., 2022). Stomatal closure, while a defense against herbivores, can increase water potential within the plant vascular system, potentially benefiting sap-sucking insects (Figure 3A). In addition, some herbivores can induce stomatal closure to facilitate their continuous feeding (Erb and Reymond, 2019). Similar to the response to pathogens, the

stomatal response to herbivores is highly dynamic and involves complex mechanisms.

Plants close stomata in response to herbivore-caused wounding

Wounding caused by herbivore feeding in leaves constitutes a major factor inducing stomatal closure. Stomatal closure helps reduce water loss, limit herbivore access to mesophyll tissues, and defend against potential pathogen entry through wound sites. The increase in JA levels and the activation of JA signaling in guard cells are critical components of the stomatal response to wounding. It is noteworthy that JA and its derivatives JA-Ile and methyl JA (MeJA) may play distinct roles in the regulation of stomatal movement under different stress conditions, likely due to their interplays with other factors, such as ABA levels and signaling (Melotto et al., 2017). Upon perception by the COI1 receptor complex, MeJA triggers Ca^{2+} signaling, which activates K^+ efflux channel GORK through CIPK5, forming calcium sensor-kinase complexes (Forster et al., 2019). Notably, wounding-induced stomatal closure occurs not only in wounded leaves but also in unwounded distal leaves (Forster et al., 2019). This is consistent with the observation that plants can sense local wounding events and trigger long-distance calcium signaling, leading to the systemic accumulation of JA (Toyota et al., 2018). The increase in JA levels, calcium signaling, and the activation of channel proteins such as GORK play key roles in mediating stomatal closure. In addition, the capacity of plants to exhibit systemic responses to local wounding signal underscores their remarkable communication and defense mechanisms evolved to safeguard against herbivore attacks.

Stomatal response upon sensing HAMPs

The recognition of HERBIVORE-ASSOCIATED MOLECULAR PATTERNs (HAMPs) by plant receptors is an intriguing aspect of plant defense mechanisms against herbivores. HAMPs include specific molecular patterns found in the oral secretions of herbivorous insects. These compounds can serve as signals that plants recognize as an indicator of herbivore attack. For instance, inceptin, a peptide fragment derived from a plant chloroplastic ATP synthase γ-subunit (cATPC) found in oral secretions from *Spodoptera frugiperda*, is perceived by a plant receptor called the INCEPTIN RECEPTOR (Schmelz et al., 2006; Steinbrenner et al., 2020). HAMP perception triggers typical defense signaling, including plasma membrane depolarization, $[Ca^{2+}]_{cyt}$ elevation, and ROS production, which can potentially induce stomatal closure (Acevedo et al., 2015). While the focus has been on early defense signaling, some HAMPs likely have the ability to induce stomatal closure, which is a crucial component of plant defense against herbivores, especially those that use stomata as entry points for feeding. Further investigations are needed to uncover the specific mechanisms involved in HAMP perception by guard cells and their role in stomatal regulation.

Stomatal closure upon sensing DAMPs induced by herbivores

Chewing herbivores physically damage plant tissues, leading to the release of cell wall fragments and intracellular components into extracellular spaces. Some of these components, such as oligogalacturonides, eATP, and cytosolic peptides, are recognized as DAMPs and have been reported to trigger stomatal closure (Hou et al., 2019b; Tanaka and Heil, 2021). Phytocytokines, such as systemin in tomatoes and Pep1 orthologs in various plant species, are induced upon herbivore feeding and wounding and trigger JA-dependent defense responses and stomatal closure (Yamaguchi et al., 2006, 2010; Wang et al., 2018b). Nevertheless, when considering chewing as the primary feeding behavior of these herbivores, the biological significance of stomatal closure in plant defense against chewing insects remains unclear.

Phloem-feeding insects, such as aphids, are sensitive to the water status within plant tissues, and water-limited conditions can enhance plant resistance to aphid infestations (Erb and Reymond, 2019). Stomatal movement plays a direct role in controlling transpiration rate, which in turn influences the water potential in the vascular system. When stomata are open, transpiration increases, leading to a reduction in water potential within the leaf vascular system. Recent research has proposed that plants may bolster their resistance to aphid infestations by regulating stomatal opening and reducing vascular water potential, a process mediated by SCREW-NUT signaling (Figure 3A) (Liu et al., 2022c). Aphid feeding, but not wounding, significantly induces the expression of *SCREW* genes in *Arabidopsis*. Overexpression of SCREWs enhances resistance to aphids, while deletion of the NUT receptor weakens this resistance (Liu et al., 2022c). The connection between increased resistance to aphids and the water loss induced by stomatal opening suggests that plants may employ this mechanism to deter aphid infestations.

Herbivores induce stomatal closure to prevent HIPV release

Stomata also mediate the release of herbivore-induced plant volatiles (HIPVs), which are widely recognized as an important defensive strategy employed by plants against herbivory (Erb and Reymond, 2019). HIPVs serve as signals to protect plants from insect herbivores by priming defenses in systemic and neighboring plant tissues or by attracting natural enemies of the herbivores (Dicke and Baldwin, 2010). Likely as a counter-defense strategy, caterpillar *Helicoverpa zea* secretes salivary enzyme glucose oxidase (GOX), which induces stomatal closure in tomato and soybean leaves, thereby constraining HIPV-mediated plant defenses (Lin et al., 2021). GOX catalyzes the oxidation of glucose, producing H_2O_2, a key signaling molecule involved in stomatal closure. Cytoplasmic glucose concentrations are closely linked to stomatal opening (Flutsch et al., 2020). Therefore, GOX may promote stomatal closure or prevent stomatal opening by generating H_2O_2 or depleting glucose.

In conclusion, herbivores have evolved various strategies to manipulate plant stomata, which can impact both the plant defense and the herbivore feeding success. This complex interplay between plants and herbivores highlights the dynamic nature of plant–herbivore interactions and the sophisticated mechanisms that have evolved on both sides in response to these interactions.

STOMATAL RESPONSES TO INFECTIONS UNDER CHANGING CLIMATE

Climate variables, such as atmospheric humidity, temperature, greenhouse gases, and light, exert a significant impact on the

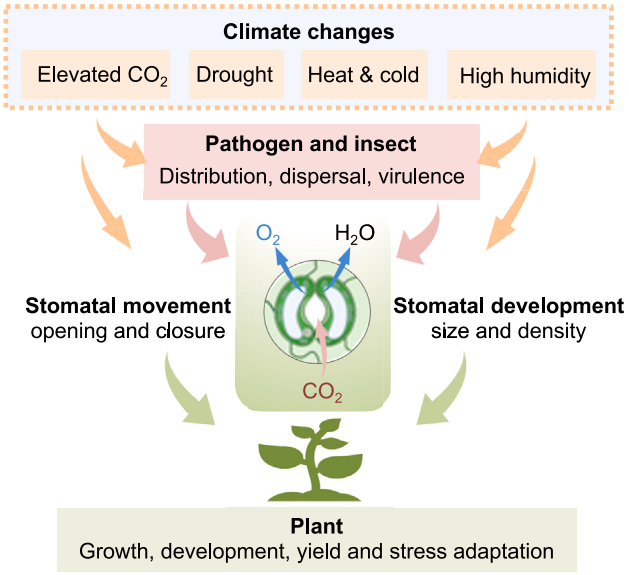

Figure 4. Climate-induced stomatal responses in plant–pathogen interactions and their impact on plant growth, development, and stress adaptation.

Climate factors, such as atmospheric CO_2 levels, drought, and temperature, directly affect the distribution, dispersal, and virulence of pathogens and insects, as well as plant stomatal movement and development. Furthermore, the dynamic responses of pathogens and insects to changing climate conditions add further layers of complexity to this intricate interplay. Consequently, these layered interactions significantly influence plant growth, development, yield, and stress adaptation.

dynamics of plant–pathogen interactions, as well as stomatal movement (Figure 4). Alterations in climate conditions can either enhance or weaken plant resistance to pathogens, thereby either deceasing or amplifying the likelihood of plant diseases emerging (Cheng et al., 2019; Singh et al., 2023). Stomatal responses to pathogens, serving as the forefront of plant defense against pathogens, are profoundly affected by these climate factors. Conversely, plants have the ability to influence the climate by regulating stomatal movement, thereby influencing the infection and dispersion of pathogens.

Stomatal response to pathogen infections under high humidity

Elevated humidity stands as a pivotal environmental factor that significantly contributes to the proliferation of plant diseases. High humidity can compromise the stomatal immunity during the pathogen pre-invasion stage. Notably, this heightened humidity compromises the ability of *Arabidopsis* and common bean (*Phaseolus vulgaris*) to close stomata in response to *P. syringae*, facilitating a greater infiltration of pathogens into leaf tissues via stomata and leading to severe infections (Panchal et al., 2016b). The compromised stomatal closure under high humidity conditions is accompanied by notable changes in guard cell hormone signaling. Specifically, high humidity activates the JA signaling while downregulating the SA signaling in guard cells (Panchal et al., 2016b). *P. syringae*-induced stomatal closure relies on SA but is antagonized by JA-Ile (Melotto et al., 2006; Zeng et al., 2011). Therefore, it was proposed that high humidity compromises stomatal immunity by activating JA signaling, while repressing SA signaling. Furthermore, high humidity also promotes the catabolism of ABA in guard cells (Okamoto et al., 2009), which may further contribute to the compromised *P. syringae*-induced stomatal closure. The foodborne pathogenic bacteria *Escherichia coli* O157:H7 can induce stomatal closure through the central ABA signaling component OST1. However, high humidity does not interfere with stomatal closure induced by *Escherichia coli* O157:H7 (Roy et al., 2013). This implies that high humidity-caused reduction of ABA levels in guard cells might not be sufficient to suppress stomatal closure in response to bacteria.

In addition, high humidity also promotes the plant susceptibility to pathogens during the post-invasion stages. As aforementioned, AvrE and HopM1 promote stomatal closure, thereby contributing to the establishment of an aqueous apoplast (Figure 3B). Importantly, the full virulence potential of AvrE and HopM1 relies on the presence of high humidity (Xin et al., 2016). Consequently, it can be inferred that high humidity may collaborate with pathogen effectors to further promote plant susceptibility by facilitating the establishment of aqueous apoplasts within plants during the post-invasion stages.

Furthermore, NLR activation in guard cells has the counterintuitive effect of inhibiting *Pst* DC3000-induced stomatal closure and increasing leaf water potential. This NLR activation effect might represent a stomata-related resistance mechanism in the post-invasion phase (Figure 3B) (Yan et al., 2019). This phenomenon becomes particularly relevant when considering that tissue water loss through stomata has been postulated to contribute to HR during ETI (Beattie, 2011). In addition, high humidity can disrupt NLR-mediated autoimmunity and ETI-associated HR in an SA accumulation-dependent manner (Yoshioka et al., 2001; Freeman and Beattie, 2009; Mosher et al., 2010). This raises the intriguing possibility that high humidity might suppress SA biosynthesis and/or signaling, and, in turn, promote the plant susceptibility to pathogens by influencing the stomatal response.

Stomatal response to pathogen infections under elevated temperatures

High temperatures encourage plants to increase their stomatal aperture to facilitate a higher transpiration rate and evaporative cooling of leaves. Plants have specialized proteins to perceive temperature changes and initiate the adaptive response to cope with temperature fluctuations (Ding et al., 2020; Kerbler and Wigge, 2023). Specifically, high temperatures stimulate stomatal opening through the blue light receptors PHOTOTROPIN 1 (PHOT1) and PHOT2, and their downstream kinase, BLUE LIGHT SIGNALING 1 (BLUS1) (Takemiya et al., 2013; Kostaki et al., 2020). High temperatures also affect stomatal patterning by deactivating the red/far-red photoreceptor phytochrome B and stabilizing the transcription factor PHYTOCHROME-INTERACTING FACTOR4 (PIF4) (Kostaki et al., 2020; Kerbler and Wigge, 2023). Low temperatures typically inhibit stomatal opening and induce stomatal closure to maintain leaf temperature in an ABA-dependent manner (Agurla et al., 2018).

Temperature fluctuation is another pivotal environmental factor affecting plant defense against pathogens (Figure 4). Many

plants, including *Arabidopsis*, rice, and tomato, are more susceptible to pathogens when exposed to elevated temperatures for a short time (Huot et al., 2017). The heat-mediated suppression of plant resistance was thought to mainly depend on its suppression of SA accumulation and signaling (Huot et al., 2017; Kim et al., 2022). However, the temperature-regulated SA production does not rely on the thermosensor phytochrome B, but on a liquid–liquid phase separation-driven biomolecular condensate mechanism. High temperature compromises the formation of guanylate-binding protein-like GTPase (GBPL)-activated condensates, which drives the expression of *CALMODULIN BINDING PROTEIN 60-LIKE G* (*CBP60g*) and *SAR-DEFICIENT 1* (*SARD1*), two critical regulators of SA biosynthesis (Kim et al., 2022).

An elevated temperature from 22°C to 28°C delays the *Pst* DC3000-induced stomatal closure in *Arabidopsis* (Yan et al., 2019). Elevated temperatures can potentiate PTI signaling likely as a consequence of co-evolution due to vigorous pathogen multiplication and MAMP production at elevated temperatures (Cheng et al., 2013). Therefore, the delay in pathogen-induced stomatal closure at elevated temperatures may not be due to altered PTI signaling. Instead, it was suggested that it might be attributed by the suppression of SA production as elevated temperatures suppress pathogen-induced SA biosynthesis (Mang et al., 2012; Huot et al., 2017; Kim et al., 2022).

Point mutations in *Arabidopsis* SNC1 and tobacco NLR protein N retain plant disease resistance at an elevated temperature, suggesting that the activity of some NLRs also mediate high-temperature suppression of plant immunity and might be "thermosensors" (Zhu et al., 2010). Expression of active form of *SNC1* (*snc1-1*) in guard cells promotes stomatal opening and inhibits pathogen- and ABA-induced stomatal closure. Notably, elevated temperatures have been observed to alleviate the stomatal closure defects in the *snc1-1* transgenic plants (Yan et al., 2019). Moreover, suppression of ETI and autoimmunity triggered by NLR mis-regulation by elevated temperatures was also fulfilled through the inhibition of the SA biosynthesis (Cheng et al., 2013; Yan et al., 2019; Kim et al., 2022). Therefore, the influence of temperature on NLR-regulated stomatal movement may rely on its suppression of NLR activities and downstream SA production. In addition, mutations in ABA biosynthesis genes can restore the *snc1-1*-mediated suppression of stomatal closure induced by *P. syringae* infections, even under high-temperature conditions (Mang et al., 2012). This observation suggests that ABA plays a pivotal role in the high-temperature-mediated inhibition of stomatal responses and the disease resistance conferred by *snc1-1*. The nuclear localization of SNC1 is crucial for its role in plant immune regulation. Both elevated temperatures and ABA treatment significantly reduce the nuclear accumulation of SNC1 and another two NLR proteins N and RESISTANT TO *P. SYRINGAE* 4 (RPS4) (Zhu et al., 2010; Mang et al., 2012). Conversely, mutations in genes regulating ABA biosynthesis but not signaling enhance the nuclear accumulation of SNC1 and RPS4, even at high temperatures (Mang et al., 2012). This implies that high temperatures may elevate ABA levels but not ABA sensitivity under the condition of pathogen infections, leading to the suppression of NLR-mediated stomatal opening by potentially inhibiting NLR translocation into the nucleus.

Stomatal responses to pathogen infections in varying light conditions

Light serves as a key environmental factor that regulates stomatal movement in plants (Figure 1). Light affects stomatal opening and closing through various photoreceptors and signaling pathways (Shimazaki et al., 2007; Pierik and Ballare, 2022). Under a normal photoperiod, blue light sensed by blue light receptors, PHOT1 and PHOT2, triggers a series of events leading to K^+ influx, turgor pressure increases of guard cells, and ultimately stomatal opening. In addition, red light and far-red light regulate stomatal opening and closure through the phytochrome photoreceptors. Red light is known to promote stomatal opening, while far-red light has the opposite effect, contributing to stomatal closure (Chen et al., 2012; Matthews et al., 2020).

Light is essential for a robust defense response against various pathogens in plants (Roden and Ingle, 2009; Ballare, 2014). Intense or prolonged exposure to light enhances plants' resistance to pathogen infections (Muhlenbock et al., 2008). This is likely due to the fact that light plays a crucial role in SA accumulation and the activation of SA-mediated defense responses during pathogen infections (Sano et al., 2014; Lajeunesse et al., 2023). Constant light treatment suppresses pathogen-induced stomatal closure during the post-invasion stage in response to *Pst* DC3000 infection, leading to increased water loss from the apoplast and preventing pathogen colonization (Lajeunesse et al., 2023). The inhibition of pathogen-induced stomatal closure by constant light treatment is attributed to the potentiation of SA signaling (Lajeunesse et al., 2023). It remains to be determined whether light also regulates the phytocytokine SCREW-mediated stomatal opening in the post-invasion stage. Red light responses are also crucial for the suppression of pathogen-induced stomatal closure, as *Arabidopsis* mutants lacking four PIF homologous genes, known modulators of red light-regulated responses, exhibit closed stomata and water soaking phenotype in constant light upon pathogen inoculation (Lajeunesse et al., 2023).

Stomatal movement follows a circadian rhythm, which is influenced by the plant's internal clock and light regulation. The circadian rhythm plays a significant role in plant defense against pathogens (Zhang et al., 2013; Zhou et al., 2015a). In the natural circadian rhythm, plants exhibit the highest susceptibility at midnight and the greatest resistance in the morning (Bhardwaj et al., 2011). The key circadian regulators, such as CIRCADIAN CLOCK-ASSOCIATED 1 (CCA1), LATE ELONGATED HYPOCOTYL (LHY), and their downstream target GLYCINE-RICH RNA BINDING PROTEIN 7 (GRP7), regulate defense responses, including stomatal closure, which contribute to plant defense against *P. syringae* (Zhang et al., 2013). It is worth noting that there are no established direct connections between the circadian modulation of stomatal movement and plant resistance to pathogens.

Stomatal responses to pathogen infections in elevated greenhouse gas environments

The concentration of greenhouse gases, including CO_2, wields a considerable influence on stomatal behavior. Overall, high CO_2 levels stimulate stomata closure, while low CO_2 concentrations

prompt stomatal opening (Figure 4). The CO_2-stimulated stomatal movement requires potential CO_2 sensors, two β-carbonic anhydrases, βCA1 and βCA4, and downstream signaling components, including a MATE-type transporter RESISTANT TO HIGH CARBON DIOXIDE 1 (RHC1), and a Raf-like protein kinase HIGH LEAF TEMPERATURE 1 (HT1) (Hu et al., 2010; Tian et al., 2015). Under elevated CO_2 concentrations, increased intracellular HCO_3^- levels, catalyzed by βCA, promote the RHC1-HT1 interaction but disrupt the HT1-OST1 interaction, consequently abolishing HT1's inhibition on OST1 and leading to OST1 activation. Active OST1 phosphorylates and activates SLAC1 to promote anion efflux and stomatal closure (Tian et al., 2015). SLAC1 itself was also suggested to be a CO_2/HCO_3^- sensor in guard cells, functioning in high CO_2-induced stomatal closure (Zhang et al., 2018). Elevated CO_2 also triggers stomatal closure by stimulating MPK4/MPK12 interaction with HT1 and suppressing HT1 activities (Hiyama et al., 2017; Takahashi et al., 2022). Under reduced CO_2 concentrations, HT1 phosphorylates and activates two other Raf-like protein kinases, CONVERGENCE OF BLUE LIGHT AND CO_2 1 (CBC1) and CBC2, which redundantly regulate stomatal opening by inhibiting S-type anion channels (Hiyama et al., 2017). Furthermore, a diverse array of plant species demonstrates a reduction in stomatal density as a response to the ongoing increase in atmospheric CO_2 levels. In *Arabidopsis*, the secreted protease CO_2 RESPONSE SECRETED PROTEASE (CRSP) cleaves the pro-peptide EPF2 to mediate the CO_2 elevation-triggered reduction of stomatal density (Engineer et al., 2014).

The influence of CO_2 on plant–pathogen interactions is a growing area of research, especially concerning its effects on stomatal behavior (Figure 4). Elevated CO_2 levels enhance *Arabidopsis* resistance to *Pst* DC3000 following dip inoculation, suggesting that increased CO_2 concentration can strengthen stomatal defense (Zhou et al., 2017). However, research indicates that this enhanced plant resistance under high CO_2 is likely attributed to the reduced stomatal density rather than an increase in MAMP-induced stomatal closure (Xu et al., 2016; Zhou et al., 2017). Surprisingly, low CO_2 conditions could also have a positive impact on plant resistance against pathogens by modulating stomatal behaviors. It has been proposed that low CO_2 conditions impede COR-mediated stomatal reopening by influencing ABA biosynthesis and signaling, although the exact molecular mechanisms remain to be elucidated (Zhou et al., 2017).

ENHANCING CROP DISEASE RESISTANCE THROUGH ENGINEERING STOMATAL IMMUNITY

Pathogen attacks present a substantial threat to global food security, especially under the influence of extreme climate conditions. Stomata play a crucial role in plant's response to both biotic and abiotic stresses, and they often employ shared molecular mechanisms to do so (Arnaud and Hwang, 2015; Melotto et al., 2017). Furthermore, the signaling pathways governing stomatal movements exhibit a high degree of conservation across the plant kingdom (Clark et al., 2022). Thus, the innovative approach of engineering stomatal immunity holds promise to enhance crop protection against diseases and environmental abiotic stresses, offering a potential solution to address the challenges posed by pathogen attacks in agriculture in a changing climate.

Genetic modifications targeting signaling components involved in stomatal movement have been studied to promote stomatal closure and improve plant response to drought and pathogen stresses in both *Arabidopsis* and crop plants. For instance, *Fv*WRKY42 from strawberry *Fragaria vesca*, a member of plant WRKY transcription factors, is induced in response to various biotic and abiotic stresses (Wei et al., 2018). Overexpression of *Fv*WRKY42 in *Arabidopsis* increases plant resistance to fungal powdery mildew and enhances tolerance to osmotic and drought stresses. These plants also exhibit heightened stomatal closure following ABA and drought treatments, along with altered expression of ABA-responsive genes (Wei et al., 2018). It remains unknown how *Fv*WRKY42 mechanistically regulates stomatal closure and whether the altered ABA-responsive genes are among many other signaling processes that *Fv*WRKY42 is involved in. MicroRNAs (miRNA) are essential regulators of plant growth, development, and stress responses. miR167 has been shown to modulate *Arabidopsis* defense to pathogens through regulating stomatal apertures. Overexpression of *miR167* in *Arabidopsis* displayed reduced auxin responses and constitutive small stomatal apertures, thereby impeding pathogen entry and conferring resistance to *P. syringae* infection (Caruana et al., 2020). It would be intriguing to elucidate the specific target of miR167 in this intricate process.

In addition, components regulating stomatal density have been explored to alter the plant adaptation to stress response. Rice transgenic plants with overexpression of *OsEPF1* showed substantially reduced stomatal density and low stomatal conductance, subsequently improving drought tolerance and water conservation (Caine et al., 2019; Mohammed et al., 2019). Notably, an extensive screening of the stomatal traits, including stomatal density and size, across a large collection of conventionally bred rice varieties and *Os*EPF1-overexpressing transgenic plants revealed a complicated relationship between stomatal traits and plant response to drought, salinity, or temperature stresses, and photosynthesis (Caine et al., 2023). These complexities underline the need for a holistic and multifaceted approach to develop effective strategies for enhancing plant stress resilience.

The exogenous applications of small peptide elicitors derived from phytocytokines can bolster plant resistance against bacterial and fungal pathogens. However, it remains uncertain whether the improved disease tolerance is directly linked to the regulation of stomatal movement. As an example, Peps could enhance plant resistance against bacterial and fungal infections as well as herbivore attacks when applied as a pretreatment (Huffaker et al., 2011; Klauser et al., 2015). In *Arabidopsis*, plant mutants lacking Peps receptors PEPR1 and PEPR2 exhibit increased susceptibility to bacterial *Pst* DC3000 by spraying but not infiltration assays, suggesting a role for stomatal immunity in this context (Zheng et al., 2018). Similarly, in chickpeas and tomatoes, chitosan-triggered immunity is associated with the regulation of stomatal closure (Czekus et al., 2020; Narula et al., 2020). In addition, exogenous treatment with glutamate, an important amino acid for all living organisms, induces the expression of MAMP-, DAMP-, and SA-induced genes and conferred

resistance against fungal pathogens in *Arabidopsis*, rice, and tomato without causing discernible growth retardation (Goto et al., 2020). Glutamate-induced resistance against *P. cannabina* pv. *alisalensis* in cabbage occurs only after spraying but not infiltration assays (Sakata et al., 2023). This implies that glutamate-induced resistance might be linked to stomatal immunity limiting bacterial entry. Nevertheless, the modulation of the stomatal aperture by peptides and certain amino acids represents a promising approach to bolster plant resistance against bacterial and fungal pathogens. However, more research is needed to fully elucidate the mechanisms involved and optimize its application for crop protection.

The use of biocontrol agents, including plant growth-promoting Rhizobacteria, to induce plant protection against both biotic and abiotic stresses is an exciting field of research in agriculture. The modulation of stomatal apertures is potentially involved in this process (Bhat et al., 2023). *Bacillus amyloliquefaciens* FZB42 is a well-known plant growth-promoting Rhizobacteria that has been shown to enhance plant resistance against various pathogens, including black shank disease caused by *Phytophthora nicotianae.* FZB42 appears to restrict pathogen infection by promoting stomatal closure, likely through the modulation of ABA and SA signaling pathways (Wu et al., 2018). Understanding the interplay between biocontrol agents, the plant immune response, and stomatal behavior will aid in the development of effective and sustainable methods for disease control in agriculture.

Manipulating stomatal movement to enhance crop disease resistance is undeniably a complex challenge, but it carries significant promise for sustainable agriculture. The future of this endeavor relies on extensive research, including large-scale genomic studies and interdisciplinary collaboration. These efforts will be pivotal in fully harnessing the potential of stomatal regulation as a tool to enhance plant disease resistance while mitigating potential trade-offs with other vital plant functions.

CONCLUDING REMARKS AND FUTURE PERSPECTIVES

Stomata, the natural openings on the surface of plant leaves, are primarily responsible for gas exchange (photosynthesis and respiration) and transpiration (water loss). They play a crucial role in maintaining plant health and function. Meanwhile, stomata are exploited by pathogens as points of entry for infections. Thus, the stomatal movement and density are dynamically regulated by plants, pathogens, and herbivores as defense and counter-defense strategies. In addition, nearly all climate factors, such as temperature, humidity, and CO_2 levels, can significantly impact stomatal behavior, pathogen growth, and plant development. Thus, plant regulation of stomatal movement and development in response to pathogen infection pressures under changing climate is highly complex. Understanding these interactions is essential for developing effective agricultural practices that can combat both pathogen infections and environmental stresses.

Emerging evidence has suggested that stomatal movement is a dynamic process involving a series of responses by both plants and pathogens during different stages of plant–pathogen interactions: pre-invasion and post-invasion phases. In the pre-invasion phase, plants detect MAMPs and DAMPs to close stomata as a defense mechanism in restricting subsequent pathogen entry. However, the closed stomata can have negative consequences, such as limiting photosynthesis and respiration rates, which affects plant health and productivity. In addition, the closed stomata also create a humid environment in the apoplast, which can be conducive to pathogen multiplication. In the post-invasion phase, pathogen infection and MAMP recognition induce the expression and production of phytocytokines in plants, some of which could reopen stomata, disrupting the aqueous environment in the apoplast, unfavorable for pathogen multiplication. On the pathogen side, pathogens have evolved counter-defense strategies to overcome stomatal closure by deploying toxins and effectors, which can induce stomatal opening or suppress MAMP-induced stomatal closure to promote subsequent pathogen entry. Furthermore, some effectors, likely secreted into the plant cells at the later infection stage, can induce stomatal closure to re-establish a humid apoplast environment in promoting pathogen colonization.

The plant hormone ABA-mediated signaling has been shown to be involved in both plant- and pathogen-regulated stomatal movement. ABA can influence stomatal behavior by controlling the activities of various ion channels in guard cells. The immune signaling intercepts with the ABA signaling at multiple steps. Pathogen-derived toxins and effectors interfere with ABA signaling, often by manipulating other plant defense hormone pathways, such as JA and SA signaling. In addition, fluctuation in climate factors, such as temperature, humidity, and CO_2 levels, can perturb stomatal responses to pathogens, thus significantly affecting the incidence and severity of plant diseases.

The intricate interplay between plants, pathogens, and environmental factors adds layers of complexity to the mechanisms governing stomatal behavior. Despite significant progress in our understanding of stomatal movement in the context of plant–pathogen interactions, this area of research remains highly complex and continuously evolving. Numerous avenues for future research will continue to uncover new aspects of this process and fill gaps in stomatal biology in the context of plant–pathogen interactions under changing climate. First, it is instrumental to further identify signaling components and mechanisms involved in stomatal response to pathogens and understand how plants adjust their stomatal behavior when facing with multiple stressors, such as simultaneous pathogen infections and abiotic stressors (e.g., drought, high temperature). Second, connections between stomatal dynamics and spatiotemporal patterns of regulatory factors involved in plant–pathogen interactions are largely lacking. The field will benefit from visualizing and monitoring stomatal behavior throughout the entire process of pathogen infections, from initial recognition to colonization. Third, it is much needed to expand our understanding of how stomata respond to various phyllospheric microbes, including fungi, viruses, and sap-sucking insects, and the potential differences in stomatal responses to beneficial endophytes versus pathogens, and how these responses contribute to plant health. Fourth, it is important to elucidate the evolutionary aspects of stomatal responses to pathogens, considering the role of stomata in plant adaptation to different environmental conditions. Stomata occurred along

with plants concurring the lands and are crucial for all terrestrial plant adaptation to complicated environmental conditions. It is important to reveal the conservation and specificity of stomatal responses to pathogens across diverse plant species, especially in economically important crops. Finally, research in genetic rewiring stomatal behaviors holds potential in the development of crops with increased resistance to pathogens and climate changes.

As technology advances and interdisciplinary research approaches are employed, we can expect to gain deeper insights into the nuanced regulatory mechanisms behind stomatal movement during pathogen interactions. These insights have the potential to lead to innovative strategies for enhancing plant defense, reducing crop losses, and improving agricultural sustainability in the face of emerging challenges such as climate change and evolving pathogens.

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REFERENCES

Acevedo, F.E., Rivera-Vega, L.J., Chung, S.H., Ray, S., and Felton, G.W. (2015). Cues from chewing insects - the intersection of DAMPs, HAMPs, MAMPs and effectors. Curr. Opin. Plant Biol. **26**:80–86.

Agurla, S., Gahir, S., Munemasa, S., Murata, Y., and Raghavendra, A.S. (2018). Mechanism of Stomatal Closure in Plants Exposed to Drought and Cold Stress. Adv. Exp. Med. Biol. **1081**:215–232.

Andrés, Z., Pérez-Hormaeche, J., Leidi, E.O., Schlücking, K., Steinhorst, L., McLachlan, D.H., Schumacher, K., Hetherington, A.M., Kudla, J., Cubero, B., and Pardo, J.M. (2014). Control of vacuolar dynamics and regulation of stomatal aperture by tonoplast potassium uptake. Proc. Natl. Acad. Sci. USA **111**:E1806–E1814.

Arnaud, D., and Hwang, I. (2015). A sophisticated network of signaling pathways regulates stomatal defenses to bacterial pathogens. Mol. Plant **8**:566–581.

Aung, K., Jiang, Y., and He, S.Y. (2018). The role of water in plant-microbe interactions. Plant J. **93**:771–780.

Ballaré, C.L. (2014). Light regulation of plant defense. Annu. Rev. Plant Biol. **65**:335–363.

Bao, Q.X., Mu, X.R., Tong, C., Li, C., Tao, W.Z., Zhao, S.T., Liu, Y.X., Wang, W.N., Wei, Y.T., Yu, F.H., et al. (2023). Sugar status in preexisting leaves determines systemic stomatal development within newly developing leaves. Proc. Natl. Acad. Sci. USA **120**, e2302854120.

Bascom, C. (2022). Pepper pathogens: XopS pumps the brakes on the pepper immune response. Plant Cell **34**:1433–1434.

Bauer, H., Ache, P., Lautner, S., Fromm, J., Hartung, W., Al-Rasheid, K.A.S., Sonnewald, S., Sonnewald, U., Kneitz, S., Lachmann, N., et al. (2013). The stomatal response to reduced relative humidity requires guard cell-autonomous ABA synthesis. Curr. Biol. **23**:53–57.

Beattie, G.A. (2011). Water relations in the interaction of foliar bacterial pathogens with plants. Annu. Rev. Phytopathol. **49**:533–555.

Bellenot, C., Routaboul, J.M., Laufs, P., and Noël, L.D. (2022). Hydathodes. Curr. Biol. **32**:R763–R764.

Bhardwaj, V., Meier, S., Petersen, L.N., Ingle, R.A., and Roden, L.C. (2011). Defence responses of Arabidopsis thaliana to infection by Pseudomonas syringae are regulated by the circadian clock. PLoS One **6**, e26968.

Bhat, M.A., Mishra, A.K., Jan, S., Bhat, M.A., Kamal, M.A., Rahman, S., Shah, A.A., and Jan, A.T. (2023). Plant Growth Promoting Rhizobacteria in Plant Health: A Perspective Study of the Underground Interaction. Plants **12**, 629.

Bi, G., and Zhou, J.M. (2021). Regulation of Cell Death and Signaling by Pore-Forming Resistosomes. Annu. Rev. Phytopathol. **59**:239–263.

Bi, G., Hu, M., Fu, L., Zhang, X., Zuo, J., Li, J., Yang, J., and Zhou, J.M. (2022). The cytosolic thiol peroxidase PRXIIB is an intracellular sensor for H(2)O(2) that regulates plant immunity through a redox relay. Nat. Plants **8**:1160–1175.

Boudsocq, M., Willmann, M.R., McCormack, M., Lee, H., Shan, L., He, P., Bush, J., Cheng, S.H., and Sheen, J. (2010). Differential innate immune signalling via Ca(2+) sensor protein kinases. Nature **464**:418–422.

Buscaill, P., Chandrasekar, B., Sanguankiattichai, N., Kourelis, J., Kaschani, F., Thomas, E.L., Morimoto, K., Kaiser, M., Preston, G.M., Ichinose, Y., and van der Hoorn, R.A.L. (2019). Glycosidase and glycan polymorphism control hydrolytic release of immunogenic flagellin peptides. Science **364**:1126.

Caine, R.S., Harrison, E.L., Sloan, J., Flis, P.M., Fischer, S., Khan, M.S., Nguyen, P.T., Nguyen, L.T., Gray, J.E., and Croft, H. (2023). The influences of stomatal size and density on rice abiotic stress resilience. New Phytol. **237**:2180–2195.

Caine, R.S., Yin, X., Sloan, J., Harrison, E.L., Mohammed, U., Fulton, T., Biswal, A.K., Dionora, J., Chater, C.C., Coe, R.A., et al. (2019). Rice with reduced stomatal density conserves water and has improved drought tolerance under future climate conditions. New Phytol. **221**:371–384.

Camoni, L., Visconti, S., Aducci, P., and Marra, M. (2019). From plant physiology to pharmacology: fusicoccin leaves the leaves. Planta **249**:49–57.

Cao, W., Li, Z., Huang, S., Shi, Y., Zhu, Y., Lai, M.N., Lok, P.L., Wang, X., Cui, Y., and Jiang, L. (2022). Correlation of vacuole morphology with stomatal lineage development by whole-cell electron tomography. Plant Physiol. **188**:2085–2100.

Caruana, J.C., Dhar, N., and Raina, R. (2020). Overexpression of Arabidopsis microRNA167 induces salicylic acid-dependent defense against Pseudomonas syringae through the regulation of its targets ARF6 and ARF8. Plant Direct **4**, e00270.

Casson, S., and Gray, J.E. (2008). Influence of environmental factors on stomatal development. New Phytol. **178**:9–23.

Castro, B., Citterico, M., Kimura, S., Stevens, D.M., Wrzaczek, M., and Coaker, G. (2021). Stress-induced reactive oxygen species compartmentalization, perception and signalling. Nat. Plants **7**:403–412.

Cerutti, A., Jauneau, A., Laufs, P., Leonhardt, N., Schattat, M.H., Berthomé, R., Routaboul, J.M., and Noël, L.D. (2019). Mangroves in the Leaves: Anatomy, Physiology, and Immunity of Epithemal Hydathodes. Annu. Rev. Phytopathol. **57**:91–116.

Cerutti, A., Jauneau, A., Auriac, M.C., Lauber, E., Martinez, Y., Chiarenza, S., Leonhardt, N., Berthomé, R., and Noël, L.D. (2017).

Immunity at Cauliflower Hydathodes Controls Systemic Infection by Xanthomonas campestris pv campestris. Plant Physiol. **174**:700–716.

Chai, J., Song, W., and Parker, J.E. (2023). New Biochemical Principles for NLR Immunity in Plants. Mol. Plant Microbe Interact. **36**:468–475.

Chen, C., Xiao, Y.G., Li, X., and Ni, M. (2012). Light-regulated stomatal aperture in Arabidopsis. Mol. Plant **5**:566–572.

Chen, D., Cao, Y., Li, H., Kim, D., Ahsan, N., Thelen, J., and Stacey, G. (2017). Extracellular ATP elicits DORN1-mediated RBOHD phosphorylation to regulate stomatal aperture. Nat. Commun. **8**:2265.

Chen, J., Sun, M., Xiao, G., Shi, R., Zhao, C., Zhang, Q., Yang, S., and Xuan, Y. (2023). Starving the enemy: how plant and microbe compete for sugar on the border. Front. Plant Sci. **14**, 1230254.

Chen, Y., Li, W., Turner, J.A., and Anderson, C.T. (2021). PECTATE LYASE LIKE12 patterns the guard cell wall to coordinate turgor pressure and wall mechanics for proper stomatal function in Arabidopsis. Plant Cell **33**:3134–3150.

Cheng, C., Gao, X., Feng, B., Sheen, J., Shan, L., and He, P. (2013). Plant immune response to pathogens differs with changing temperatures. Nat. Commun. **4**:2530.

Cheng, Y.T., Zhang, L., and He, S.Y. (2019). Plant-Microbe Interactions Facing Environmental Challenge. Cell Host Microbe **26**:183–192.

Clark, G., Fraley, D., Steinebrunner, I., Cervantes, A., Onyirimba, J., Liu, A., Torres, J., Tang, W., Kim, J., and Roux, S.J. (2011). Extracellular nucleotides and apyrases regulate stomatal aperture in Arabidopsis. Plant Physiol. **156**:1740–1753.

Clark, J.W., Harris, B.J., Hetherington, A.J., Hurtado-Castano, N., Brench, R.A., Casson, S., Williams, T.A., Gray, J.E., and Hetherington, A.M. (2022). The origin and evolution of stomata. Curr. Biol. **32**:R539–R553.

Colaianni, N.R., Parys, K., Lee, H.S., Conway, J.M., Kim, N.H., Edelbacher, N., Mucyn, T.S., Madalinski, M., Law, T.F., Jones, C.D., et al. (2021). A complex immune response to flagellin epitope variation in commensal communities. Cell Host Microbe **29**:635–649.e9.

Cord-Landwehr, S., Melcher, R.L.J., Kolkenbrock, S., and Moerschbacher, B.M. (2016). A chitin deacetylase from the endophytic fungus Pestalotiopsis sp. efficiently inactivates the elicitor activity of chitin oligomers in rice cells. Sci. Rep. **6**, 38018.

Cox, K.L., Meng, F., Wilkins, K.E., Li, F., Wang, P., Booher, N.J., Carpenter, S.C.D., Chen, L.Q., Zheng, H., Gao, X., et al. (2017). TAL effector driven induction of a SWEET gene confers susceptibility to bacterial blight of cotton. Nat. Commun. **8**, 15588.

Cubero-Font, P., and De Angeli, A. (2021). Connecting vacuolar and plasma membrane transport networks. New Phytol. **229**:755–762.

Cui, H., Tsuda, K., and Parker, J.E. (2015). Effector-triggered immunity: from pathogen perception to robust defense. Annu. Rev. Plant Biol. **66**:487–511.

Cutler, S.R., Rodriguez, P.L., Finkelstein, R.R., and Abrams, S.R. (2010). Abscisic acid: emergence of a core signaling network. Annu. Rev. Plant Biol. **61**:651–679.

Czekus, Z., Poor, P., Tari, I., and Ordog, A. (2020). Effects of Light and Daytime on the Regulation of Chitosan-Induced Stomatal Responses and Defence in Tomato Plants. Plants **9**:59.

DeFalco, T.A., and Zipfel, C. (2021). Molecular mechanisms of early plant pattern-triggered immune signaling. Mol. Cell **81**:4346.

Delaunay, A., Pflieger, D., Barrault, M.B., Vinh, J., and Toledano, M.B. (2002). A thiol peroxidase is an H2O2 receptor and redox-transducer in gene activation. Cell **111**:471–481.

Dicke, M., and Baldwin, I.T. (2010). The evolutionary context for herbivore-induced plant volatiles: beyond the 'cry for help'. Trends Plant Sci. **15**:167–175.

Ding, Y., Shi, Y., and Yang, S. (2020). Molecular Regulation of Plant Responses to Environmental Temperatures. Mol. Plant **13**:544–564.

Du, M., Zhai, Q., Deng, L., Li, S., Li, H., Yan, L., Huang, Z., Wang, B., Jiang, H., Huang, T., et al. (2014). Closely related NAC transcription factors of tomato differentially regulate stomatal closure and reopening during pathogen attack. Plant Cell **26**:3167–3184.

Dubiella, U., Seybold, H., Durian, G., Komander, E., Lassig, R., Witte, C.P., Schulze, W.X., and Romeis, T. (2013). Calcium-dependent protein kinase/NADPH oxidase activation circuit is required for rapid defense signal propagation. Proc. Natl. Acad. Sci. USA **110**:8744–8749.

Dutton, C., Hõrak, H., Hepworth, C., Mitchell, A., Ton, J., Hunt, L., and Gray, J.E. (2019). Bacterial infection systemically suppresses stomatal density. Plant Cell Environ. **42**:2411–2421.

Eastman, S., Smith, T., Zaydman, M.A., Kim, P., Martinez, S., Damaraju, N., DiAntonio, A., Milbrandt, J., Clemente, T.E., Alfano, J.R., and Guo, M. (2022). A phytobacterial TIR domain effector manipulates NAD(+) to promote virulence. New Phytol. **233**:890–904.

Eisenach, C., and De Angeli, A. (2017). Ion Transport at the Vacuole during Stomatal Movements. Plant Physiol. **174**:520–530.

Eisenach, C., Baetz, U., Huck, N.V., Zhang, J., De Angeli, A., Beckers, G.J.M., and Martinoia, E. (2017). ABA-Induced Stomatal Closure Involves ALMT4, a Phosphorylation-Dependent Vacuolar Anion Channel of Arabidopsis. Plant Cell **29**:2552–2569.

Engineer, C.B., Ghassemian, M., Anderson, J.C., Peck, S.C., Hu, H., and Schroeder, J.I. (2014). Carbonic anhydrases, EPF2 and a novel protease mediate CO2 control of stomatal development. Nature **513**:246–250.

Erb, M., and Reymond, P. (2019). Molecular Interactions Between Plants and Insect Herbivores. Annu. Rev. Plant Biol. **70**:527–557.

Escocard de Azevedo Manhães, A.M., Ortiz-Morea, F.A., He, P., and Shan, L. (2021). Plant plasma membrane-resident receptors: Surveillance for infections and coordination for growth and development. J. Integr. Plant Biol. **63**:79–101.

Essuman, K., Milbrandt, J., Dangl, J.L., and Nishimura, M.T. (2022). Shared TIR enzymatic functions regulate cell death and immunity across the tree of life. Science **377**:486.

Feitosa-Araujo, E., da Fonseca-Pereira, P., Knorr, L.S., Schwarzländer, M., and Nunes-Nesi, A. (2022). NAD meets ABA: connecting cellular metabolism and hormone signaling. Trends Plant Sci. **27**:16–28.

Ficarra, F.A., Grandellis, C., Garavaglia, B.S., Gottig, N., and Ottado, J. (2018). Bacterial and plant natriuretic peptides improve plant defence responses against pathogens. Mol. Plant Pathol. **19**:801–811.

Fichman, Y., Zandalinas, S.I., Peck, S., Luan, S., and Mittler, R. (2022). HPCA1 is required for systemic reactive oxygen species and calcium cell-to-cell signaling and plant acclimation to stress. Plant Cell **34**:4453–4471.

Flütsch, S., Nigro, A., Conci, F., Fajkus, J., Thalmann, M., Trtílek, M., Panzarová, K., and Santelia, D. (2020). Glucose uptake to guard cells via STP transporters provides carbon sources for stomatal opening and plant growth. EMBO Rep. **21**, e49719.

Forster, S., Schmidt, L.K., Kopic, E., Anschutz, U., Huang, S., Schlucking, K., Koster, P., Waadt, R., Larrieu, A., Batistic, O., et al. (2019). Wounding-Induced Stomatal Closure Requires Jasmonate-Mediated Activation of GORK K(+) Channels by a Ca(2+) Sensor-Kinase CBL1-CIPK5 Complex. Dev. Cell **48**:87–99.e86.

Freeman, B.C., and Beattie, G.A. (2009). Bacterial growth restriction during host resistance to Pseudomonas syringae is associated with leaf water loss and localized cessation of vascular activity in Arabidopsis thaliana. Mol. Plant Microbe Interact. **22**:857–867.

Fürst, U., Zeng, Y., Albert, M., Witte, A.K., Fliegmann, J., and Felix, G. (2020). Perception of Agrobacterium tumefaciens flagellin by FLS2(XL) confers resistance to crown gall disease. Nat. Plants **6**:22–27.

Gao, F., Zhang, B.S., Zhao, J.H., Huang, J.F., Jia, P.S., Wang, S., Zhang, J., Zhou, J.M., and Guo, H.S. (2019). Deacetylation of chitin oligomers increases virulence in soil-borne fungal pathogens. Nat. Plants **5**:1167–1176.

Gao, L., Kantar, M.B., Moxley, D., Ortiz-Barrientos, D., and Rieseberg, L.H. (2023). Crop adaptation to climate change: An evolutionary perspective. Mol. Plant **16**:1518–1546.

Gao, X., Chen, X., Lin, W., Chen, S., Lu, D., Niu, Y., Li, L., Cheng, C., McCormack, M., Sheen, J., et al. (2013). Bifurcation of Arabidopsis NLR Immune Signaling via Ca(2+)-Dependent Protein Kinases. PLoS Pathog. **9**, e1003127.

Ge, D., Yeo, I.C., and Shan, L. (2022). Knowing me, knowing you: Self and non-self recognition in plant immunity. Essays Biochem. **66**:447–458.

Gentzel, I., Giese, L., Ekanayake, G., Mikhail, K., Zhao, W., Cocuron, J.C., Alonso, A.P., and Mackey, D. (2022). Dynamic nutrient acquisition from a hydrated apoplast supports biotrophic proliferation of a bacterial pathogen of maize. Cell Host Microbe **30**:502–517.e4.

Gimenez-Ibanez, S., Boter, M., Fernández-Barbero, G., Chini, A., Rathjen, J.P., and Solano, R. (2014). The bacterial effector HopX1 targets JAZ transcriptional repressors to activate jasmonate signaling and promote infection in Arabidopsis. PLoS Biol. **12**, e1001792.

Goel, A.K., Lundberg, D., Torres, M.A., Matthews, R., Akimoto-Tomiyama, C., Farmer, L., Dangl, J.L., and Grant, S.R. (2008). The Pseudomonas syringae type III effector HopAM1 enhances virulence on water-stressed plants. Mol. Plant Microbe Interact. **21**:361–370.

Goto, Y., Maki, N., Ichihashi, Y., Kitazawa, D., Igarashi, D., Kadota, Y., and Shirasu, K. (2020). Exogenous Treatment with Glutamate Induces Immune Responses in Arabidopsis. Mol. Plant Microbe Interact. **33**:474–487.

Grondin, A., Rodrigues, O., Verdoucq, L., Merlot, S., Leonhardt, N., and Maurel, C. (2015). Aquaporins Contribute to ABA-Triggered Stomatal Closure through OST1-Mediated Phosphorylation. Plant Cell **27**:1945–1954.

Grossiord, C., Buckley, T.N., Cernusak, L.A., Novick, K.A., Poulter, B., Siegwolf, R.T.W., Sperry, J.S., and McDowell, N.G. (2020). Plant responses to rising vapor pressure deficit. New Phytol. **226**:1550–1566.

Gudesblat, G.E., Torres, P.S., and Vojnov, A.A. (2009). Xanthomonas campestris overcomes Arabidopsis stomatal innate immunity through a DSF cell-to-cell signal-regulated virulence factor. Plant Physiol. **149**:1017–1027.

Guo, X., Wang, L., and Dong, J. (2021). Establishing asymmetry: stomatal division and differentiation in plants. New Phytol. **232**:60–67.

Gust, A.A., Pruitt, R., and Nürnberger, T. (2017). Sensing Danger: Key to Activating Plant Immunity. Trends Plant Sci. **22**:779–791.

Guzel Deger, A., Scherzer, S., Nuhkat, M., Kedzierska, J., Kollist, H., Brosché, M., Unyayar, S., Boudsocq, M., Hedrich, R., and Roelfsema, M.R.G. (2015). Guard cell SLAC1-type anion channels mediate flagellin-induced stomatal closure. New Phytol. **208**:162–173.

Han, S.K., Kwak, J.M., and Qi, X. (2021). Stomatal Lineage Control by Developmental Program and Environmental Cues. Front. Plant Sci. **12**, 751852.

Hander, T., Fernandez-Fernandez, A.D., Kumpf, R.P., Willems, P., Schatowitz, H., Rombaut, D., Staes, A., Nolf, J., Pottie, R., Yao, P., et al. (2019). Damage on plants activates Ca(2+)-dependent metacaspases for release of immunomodulatory peptides. Science **363**, eaar7486.

Hao, L.H., Wang, W.X., Chen, C., Wang, Y.F., Liu, T., Li, X., and Shang, Z.L. (2012). Extracellular ATP promotes stomatal opening of Arabidopsis thaliana through heterotrimeric G protein alpha subunit and reactive oxygen species. Mol. Plant **5**:852–864.

Hind, S.R., Strickler, S.R., Boyle, P.C., Dunham, D.M., Bao, Z., O'Doherty, I.M., Baccile, J.A., Hoki, J.S., Viox, E.G., Clarke, C.R., et al. (2016). Tomato receptor FLAGELLIN-SENSING 3 binds flgII-28 and activates the plant immune system. Nat. Plants **2**, 16128.

Hiyama, A., Takemiya, A., Munemasa, S., Okuma, E., Sugiyama, N., Tada, Y., Murata, Y., and Shimazaki, K.I. (2017). Blue light and CO(2) signals converge to regulate light-induced stomatal opening. Nat. Commun. **8**:1284.

Horaruang, W., Klejchová, M., Carroll, W., Silva-Alvim, F.A.L., Waghmare, S., Papanatsiou, M., Amtmann, A., Hills, A., Alvim, J.C., Blatt, M.R., and Zhang, B. (2022). Engineering a K(+) channel 'sensory antenna' enhances stomatal kinetics, water use efficiency and photosynthesis. Nat. Plants **8**:1262–1274.

Hou, S., Shen, H., and Shao, H. (2019a). PAMP-induced peptide 1 cooperates with salicylic acid to regulate stomatal immunity in Arabidopsis thaliana. Plant Signal. Behav. **14**, 1666657.

Hou, S., Liu, D., and He, P. (2021). Phytocytokines function as immunological modulators of plant immunity. Stress Biol. **1**:8.

Hou, S., Liu, Z., Shen, H., and Wu, D. (2019b). Damage-Associated Molecular Pattern-Triggered Immunity in Plants. Front. Plant Sci. **10**:646.

Hou, S., Wang, X., Chen, D., Yang, X., Wang, M., Turrà, D., Di Pietro, A., and Zhang, W. (2014). The secreted peptide PIP1 amplifies immunity through receptor-like kinase 7. PLoS Pathog. **10**, e1004331.

Howe, G.A., Major, I.T., and Koo, A.J. (2018). Modularity in Jasmonate Signaling for Multistress Resilience. Annu. Rev. Plant Biol. **69**:387–415.

Hsu, P.K., Dubeaux, G., Takahashi, Y., and Schroeder, J.I. (2021). Signaling mechanisms in abscisic acid-mediated stomatal closure. Plant J. **105**:307–321.

Hu, H., Boisson-Dernier, A., Israelsson-Nordström, M., Böhmer, M., Xue, S., Ries, A., Godoski, J., Kuhn, J.M., and Schroeder, J.I. (2010). Carbonic anhydrases are upstream regulators of CO2-controlled stomatal movements in guard cells. Nat. Cell Biol. **12**:87–93.

Hu, Y., Ding, Y., Cai, B., Qin, X., Wu, J., Yuan, M., Wan, S., Zhao, Y., and Xin, X.F. (2022). Bacterial effectors manipulate plant abscisic acid signaling for creation of an aqueous apoplast. Cell Host Microbe **30**:518–529.e6.

Hu, Z., and Chai, J. (2023). Assembly and Architecture of NLR Resistosomes and Inflammasomes. Annu. Rev. Biophys. **52**:207–228.

Hua, D., Wang, C., He, J., Liao, H., Duan, Y., Zhu, Z., Guo, Y., Chen, Z., and Gong, Z. (2012). A plasma membrane receptor kinase, GHR1, mediates abscisic acid- and hydrogen peroxide-regulated stomatal movement in Arabidopsis. Plant Cell **24**:2546–2561.

Huang, S., Jia, A., Ma, S., Sun, Y., Chang, X., Han, Z., and Chai, J. (2023). NLR signaling in plants: from resistosomes to second messengers. Trends Biochem. Sci. **48**:776–787.

Huffaker, A., Dafoe, N.J., and Schmelz, E.A. (2011). ZmPep1, an ortholog of Arabidopsis elicitor peptide 1, regulates maize innate immunity and enhances disease resistance. Plant Physiol. **155**:1325–1338.

Huot, B., Castroverde, C.D.M., Velásquez, A.C., Hubbard, E., Pulman, J.A., Yao, J., Childs, K.L., Tsuda, K., Montgomery, B.L., and He, S.Y. (2017). Dual impact of elevated temperature on plant defence and bacterial virulence in Arabidopsis. Nat. Commun. **8**:1808.

Hurley, B., Lee, D., Mott, A., Wilton, M., Liu, J., Liu, Y.C., Angers, S., Coaker, G., Guttman, D.S., and Desveaux, D. (2014). The

Pseudomonas syringae type III effector HopF2 suppresses Arabidopsis stomatal immunity. PLoS One **9**, e114921.

Jia, X., Wang, L., Zhao, H., Zhang, Y., Chen, Z., Xu, L., and Yi, K. (2023). The origin and evolution of salicylic acid signaling and biosynthesis in plants. Mol. Plant **16**:245–259.

Jiang, S., Yao, J., Ma, K.W., Zhou, H., Song, J., He, S.Y., and Ma, W. (2013). Bacterial effector activates jasmonate signaling by directly targeting JAZ transcriptional repressors. PLoS Pathog. **9**, e1003715.

Jin, L., Ham, J.H., Hage, R., Zhao, W., Soto-Hernández, J., Lee, S.Y., Paek, S.M., Kim, M.G., Boone, C., Coplin, D.L., and Mackey, D. (2016). Direct and Indirect Targeting of PP2A by Conserved Bacterial Type-III Effector Proteins. PLoS Pathog. **12**, e1005609.

Kadota, Y., Sklenar, J., Derbyshire, P., Stransfeld, L., Asai, S., Ntoukakis, V., Jones, J.D., Shirasu, K., Menke, F., Jones, A., and Zipfel, C. (2014). Direct regulation of the NADPH oxidase RBOHD by the PRR-associated kinase BIK1 during plant immunity. Mol. Cell **54**:43–55.

Kerbler, S.M., and Wigge, P.A. (2023). Temperature Sensing in Plants. Annu. Rev. Plant Biol. **74**:341–366.

Keynia, S., Jaafar, L., Zhou, Y., Anderson, C.T., and Turner, J.A. (2023). Stomatal opening efficiency is controlled by cell wall organization in Arabidopsis thaliana. PNAS Nexus **2**:pgad294.

Khokon, M.A.R., Salam, M.A., Jammes, F., Ye, W., Hossain, M.A., Okuma, E., Nakamura, Y., Mori, I.C., Kwak, J.M., and Murata, Y. (2017). MPK9 and MPK12 function in SA-induced stomatal closure in Arabidopsis thaliana. Biosci. Biotechnol. Biochem. **81**:1394–1400.

Kim, J.H., Castroverde, C.D.M., Huang, S., Li, C., Hilleary, R., Seroka, A., Sohrabi, R., Medina-Yerena, D., Huot, B., Wang, J., et al. (2022). Increasing the resilience of plant immunity to a warming climate. Nature **607**:339–344.

Kim, T.H., Hauser, F., Ha, T., Xue, S., Böhmer, M., Nishimura, N., Munemasa, S., Hubbard, K., Peine, N., Lee, B.H., et al. (2011). Chemical genetics reveals negative regulation of abscisic acid signaling by a plant immune response pathway. Curr. Biol. **21**:990–997.

Klauser, D., Desurmont, G.A., Glauser, G., Vallat, A., Flury, P., Boller, T., Turlings, T.C.J., and Bartels, S. (2015). The Arabidopsis Pep-PEPR system is induced by herbivore feeding and contributes to JA-mediated plant defence against herbivory. J. Exp. Bot. **66**:5327–5336.

Kostaki, K.I., Coupel-Ledru, A., Bonnell, V.C., Gustavsson, M., Sun, P., McLaughlin, F.J., Fraser, D.P., McLachlan, D.H., Hetherington, A.M., Dodd, A.N., and Franklin, K.A. (2020). Guard Cells Integrate Light and Temperature Signals to Control Stomatal Aperture. Plant Physiol. **182**:1404–1419.

Kwak, J.M., Mori, I.C., Pei, Z.M., Leonhardt, N., Torres, M.A., Dangl, J.L., Bloom, R.E., Bodde, S., Jones, J.D.G., and Schroeder, J.I. (2003). NADPH oxidase AtrbohD and AtrbohF genes function in ROS-dependent ABA signaling in Arabidopsis. EMBO J. **22**:2623–2633.

Lajeunesse, G., Roussin-Léveillée, C., Boutin, S., Fortin, É., Laforest-Lapointe, I., and Moffett, P. (2023). Light prevents pathogen-induced aqueous microenvironments via potentiation of salicylic acid signaling. Nat. Commun. **14**:713.

Lawson, T., and Matthews, J. (2020). Guard Cell Metabolism and Stomatal Function. Annu. Rev. Plant Biol. **71**:273–302.

Lee, D., Bourdais, G., Yu, G., Robatzek, S., and Coaker, G. (2015). Phosphorylation of the Plant Immune Regulator RPM1-INTERACTING PROTEIN4 Enhances Plant Plasma Membrane H(+)-ATPase Activity and Inhibits Flagellin-Triggered Immune Responses in Arabidopsis. Plant Cell **27**:2042–2056.

Li, L., Li, M., Yu, L., Zhou, Z., Liang, X., Liu, Z., Cai, G., Gao, L., Zhang, X., Wang, Y., et al. (2014). The FLS2-associated kinase BIK1 directly phosphorylates the NADPH oxidase RbohD to control plant immunity. Cell Host Microbe **15**:329–338.

Liang, X., Ding, P., Lian, K., Wang, J., Ma, M., Li, L., Li, L., Li, M., Zhang, X., Chen, S., et al. (2016). Arabidopsis heterotrimeric G proteins regulate immunity by directly coupling to the FLS2 receptor. Elife **5**, e13568.

Lin, P.A., Chen, Y., Ponce, G., Acevedo, F.E., Lynch, J.P., Anderson, C.T., Ali, J.G., and Felton, G.W. (2022). Stomata-mediated interactions between plants, herbivores, and the environment. Trends Plant Sci. **27**:287–300.

Lin, P.A., Chen, Y., Chaverra-Rodriguez, D., Heu, C.C., Zainuddin, N.B., Sidhu, J.S., Peiffer, M., Tan, C.W., Helms, A., Kim, D., et al. (2021). Silencing the alarm: an insect salivary enzyme closes plant stomata and inhibits volatile release. New Phytol. **230**:793–803.

Liu, H., Lu, C., Li, Y., Wu, T., Zhang, B., Liu, B., Feng, W., Xu, Q., Dong, H., He, S., et al. (2022a). The bacterial effector AvrRxo1 inhibits vitamin B6 biosynthesis to promote infection in rice. Plant Commun. **3**, 100324.

Liu, J., Elmore, J.M., Lin, Z.J.D., and Coaker, G. (2011). A receptor-like cytoplasmic kinase phosphorylates the host target RIN4, leading to the activation of a plant innate immune receptor. Cell Host Microbe **9**:137–146.

Liu, J., Huang, Y., Kong, L., Yu, X., Feng, B., Liu, D., Zhao, B., Mendes, G.C., Yuan, P., Ge, D., et al. (2020a). The malectin-like receptor-like kinase LETUM1 modulates NLR protein SUMM2 activation via MEKK2 scaffolding. Nat. Plants **6**:1106–1115.

Liu, L., Li, Y., Xu, Z., Chen, H., Zhang, J., Manion, B., Liu, F., Zou, L., Fu, Z.Q., and Chen, G. (2022b). The Xanthomonas type III effector XopAP prevents stomatal closure by interfering with vacuolar acidification. J. Integr. Plant Biol. **64**:1994–2008.

Liu, X.S., Liang, C.C., Hou, S.G., Wang, X., Chen, D.H., Shen, J.L., Zhang, W., and Wang, M. (2020b). The LRR-RLK Protein HSL3 Regulates Stomatal Closure and the Drought Stress Response by Modulating Hydrogen Peroxide Homeostasis. Front. Plant Sci. **11**, 548034.

Liu, Y., Maierhofer, T., Rybak, K., Sklenar, J., Breakspear, A., Johnston, M.G., Fliegmann, J., Huang, S., Roelfsema, M.R.G., Felix, G., et al. (2019). Anion channel SLAH3 is a regulatory target of chitin receptor-associated kinase PBL27 in microbial stomatal closure. Elife **8**:7554.

Liu, Z., Hou, S., Rodrigues, O., Wang, P., Luo, D., Munemasa, S., Lei, J., Liu, J., Ortiz-Morea, F.A., Wang, X., et al. (2022c). Phytocytokine signalling reopens stomata in plant immunity and water loss. Nature **605**:332–339.

Lolle, S., Stevens, D., and Coaker, G. (2020). Plant NLR-triggered immunity: from receptor activation to downstream signaling. Curr. Opin. Immunol. **62**:99–105.

Lopez-Moya, F., Suarez-Fernandez, M., and Lopez-Llorca, L.V. (2019). Molecular Mechanisms of Chitosan Interactions with Fungi and Plants. Int. J. Mol. Sci. **20**, 332.

Ma, X., Xu, G., He, P., and Shan, L. (2016). SERKing Coreceptors for Receptors. Trends Plant Sci. **21**:1017–1033.

Mang, H.G., Qian, W., Zhu, Y., Qian, J., Kang, H.G., Klessig, D.F., and Hua, J. (2012). Abscisic acid deficiency antagonizes high-temperature inhibition of disease resistance through enhancing nuclear accumulation of resistance proteins SNC1 and RPS4 in Arabidopsis. Plant Cell **24**:1271–1284.

Manik, M.K., Shi, Y., Li, S., Zaydman, M.A., Damaraju, N., Eastman, S., Smith, T.G., Gu, W., Masic, V., Mosaiab, T., et al. (2022). Cyclic ADP ribose isomers: Production, chemical structures, and immune signaling. Science **377**, eadc8969.

Matkowski, H., and Daszkowska-Golec, A. (2023). Update on stomata development and action under abiotic stress. Front. Plant Sci. **14**, 1270180.

Matthews, J.S.A., Vialet-Chabrand, S., and Lawson, T. (2020). Role of blue and red light in stomatal dynamic behaviour. J. Exp. Bot. **71**:2253–2269.

Melotto, M., Zhang, L., Oblessuc, P.R., and He, S.Y. (2017). Stomatal Defense a Decade Later. Plant Physiol. **174**:561–571.

Melotto, M., Underwood, W., Koczan, J., Nomura, K., and He, S.Y. (2006). Plant stomata function in innate immunity against bacterial invasion. Cell **126**:969–980.

Meng, X., Chen, X., Mang, H., Liu, C., Yu, X., Gao, X., Torii, K.U., He, P., and Shan, L. (2015). Differential Function of Arabidopsis SERK Family Receptor-like Kinases in Stomatal Patterning. Curr. Biol. **25**:2361–2372.

Miao, Y., Lv, D., Wang, P., Wang, X.C., Chen, J., Miao, C., and Song, C.P. (2006). An Arabidopsis glutathione peroxidase functions as both a redox transducer and a scavenger in abscisic acid and drought stress responses. Plant Cell **18**:2749–2766.

Mirasole, F.M., Nastasi, S.P., Cubero-Font, P., and De Angeli, A. (2023). Vacuolar control of stomatal opening revealed by 3D imaging of the guard cells. Sci. Rep. **13**:7647.

Misas-Villamil, J.C., Kolodziejek, I., Crabill, E., Kaschani, F., Niessen, S., Shindo, T., Kaiser, M., Alfano, J.R., and van der Hoorn, R.A.L. (2013). Pseudomonas syringae pv. syringae uses proteasome inhibitor syringolin A to colonize from wound infection sites. PLoS Pathog. **9**, e1003281.

Mohammed, U., Caine, R.S., Atkinson, J.A., Harrison, E.L., Wells, D., Chater, C.C., Gray, J.E., Swarup, R., and Murchie, E.H. (2019). Rice plants overexpressing OsEPF1 show reduced stomatal density and increased root cortical aerenchyma formation. Sci. Rep. **9**:5584.

Mosher, S., Moeder, W., Nishimura, N., Jikumaru, Y., Joo, S.H., Urquhart, W., Klessig, D.F., Kim, S.K., Nambara, E., and Yoshioka, K. (2010). The lesion-mimic mutant cpr22 shows alterations in abscisic acid signaling and abscisic acid insensitivity in a salicylic acid-dependent manner. Plant Physiol. **152**:1901–1913.

Mühlenbock, P., Szechynska-Hebda, M., Plaszczyca, M., Baudo, M., Mateo, A., Mullineaux, P.M., Parker, J.E., Karpinska, B., and Karpinski, S. (2008). Chloroplast signaling and LESION SIMULATING DISEASE1 regulate crosstalk between light acclimation and immunity in Arabidopsis. Plant Cell **20**:2339–2356.

Müller, H.M., Schäfer, N., Bauer, H., Geiger, D., Lautner, S., Fromm, J., Riederer, M., Bueno, A., Nussbaumer, T., Mayer, K., et al. (2017). The desert plant Phoenix dactylifera closes stomata via nitrate-regulated SLAC1 anion channel. New Phytol. **216**:150–162.

Murata, Y., Mori, I.C., and Munemasa, S. (2015). Diverse stomatal signaling and the signal integration mechanism. Annu. Rev. Plant Biol. **66**:369–392.

Mustilli, A.C., Merlot, S., Vavasseur, A., Fenzi, F., and Giraudat, J. (2002). Arabidopsis OST1 protein kinase mediates the regulation of stomatal aperture by abscisic acid and acts upstream of reactive oxygen species production. Plant Cell **14**:3089–3099.

Narula, K., Elagamey, E., Abdellatef, M.A.E., Sinha, A., Ghosh, S., Chakraborty, N., and Chakraborty, S. (2020). Chitosan-triggered immunity to Fusarium in chickpea is associated with changes in the plant extracellular matrix architecture, stomatal closure and remodeling of the plant metabolome and proteome. Plant J. **103**:561–583.

Ngou, B.P.M., Ding, P., and Jones, J.D.G. (2022). Thirty years of resistance: Zig-zag through the plant immune system. Plant Cell **34**:1447–1478.

Nomura, K., Andreazza, F., Cheng, J., Dong, K., Zhou, P., and He, S.Y. (2023). Bacterial pathogens deliver water/solute-permeable channels as a virulence strategy. Nature **10**:1038.

Okamoto, M., Tanaka, Y., Abrams, S.R., Kamiya, Y., Seki, M., and Nambara, E. (2009). High humidity induces abscisic acid 8'-hydroxylase in stomata and vasculature to regulate local and systemic abscisic acid responses in Arabidopsis. Plant Physiol. **149**:825–834.

Paauw, M., van Hulten, M., Chatterjee, S., Berg, J.A., Taks, N.W., Giesbers, M., Richard, M.M.S., and van den Burg, H.A. (2023). Hydathode immunity protects the Arabidopsis leaf vasculature against colonization by bacterial pathogens. Curr. Biol. **33**:697–710.e6.

Panchal, S., Roy, D., Chitrakar, R., Price, L., Breitbach, Z.S., Armstrong, D.W., and Melotto, M. (2016a). Coronatine Facilitates Pseudomonas syringae Infection of Arabidopsis Leaves at Night. Front. Plant Sci. **7**:880.

Panchal, S., Chitrakar, R., Thompson, B.K., Obulareddy, N., Roy, D., Hambright, W.S., and Melotto, M. (2016b). Regulation of Stomatal Defense by Air Relative Humidity. Plant Physiol. **172**:2021–2032.

Pantoja, O. (2021a). Recent Advances in the Physiology of Ion Channels in Plants. Annu. Rev. Plant Biol. **72**:463–495.

Pantoja, O. (2021b). Recent Advances in the Physiology of Ion Channels in Plants. Annu. Rev. Plant Biol. **72**:463–495.

Parys, K., Colaianni, N.R., Lee, H.S., Hohmann, U., Edelbacher, N., Trgovcevic, A., Blahovska, Z., Lee, D., Mechtler, A., Muhari-Portik, Z., et al. (2021). Signatures of antagonistic pleiotropy in a bacterial flagellin epitope. Cell Host Microbe **29**:620–634.e9.

Peng, Y., Yang, J., Li, X., and Zhang, Y. (2021). Salicylic Acid: Biosynthesis and Signaling. Annu. Rev. Plant Biol. **72**:761–791.

Pierik, R., and Ballaré, C.L. (2022). Control of Plant Growth and Defense by Photoreceptors: From Mechanisms to Opportunities in Agriculture. Mol. Plant **15**:1825.

Prodhan, M.Y., Munemasa, S., Nahar, M.N.E.N., Nakamura, Y., and Murata, Y. (2018). Guard Cell Salicylic Acid Signaling Is Integrated into Abscisic Acid Signaling via the Ca(2+)/CPK-Dependent Pathway. Plant Physiol. **178**:441–450.

Qi, J., Song, C.P., Wang, B., Zhou, J., Kangasjärvi, J., Zhu, J.K., and Gong, Z. (2018). Reactive oxygen species signaling and stomatal movement in plant responses to drought stress and pathogen attack. J. Integr. Plant Biol. **60**:805–826.

Rhodes, J., Roman, A.O., Bjornson, M., Brandt, B., Derbyshire, P., Wyler, M., Schmid, M.W., Menke, F.L.H., Santiago, J., and Zipfel, C. (2022). Perception of a conserved family of plant signalling peptides by the receptor kinase HSL3. Elife **11**, e74687.

Robert-Seilaniantz, A., Grant, M., and Jones, J.D.G. (2011). Hormone crosstalk in plant disease and defense: more than just jasmonate-salicylate antagonism. Annu. Rev. Phytopathol. **49**:317–343.

Roberts, R., Liu, A.E., Wan, L., Geiger, A.M., Hind, S.R., Rosli, H.G., and Martin, G.B. (2020). Molecular Characterization of Differences between the Tomato Immune Receptors Flagellin Sensing 3 and Flagellin Sensing 2(1)([OPEN]). Plant Physiol. **183**:1825–1837.

Roden, L.C., and Ingle, R.A. (2009). Lights, rhythms, infection: the role of light and the circadian clock in determining the outcome of plant-pathogen interactions. Plant Cell **21**:2546–2552.

Rodrigues, O., Reshetnyak, G., Grondin, A., Saijo, Y., Leonhardt, N., Maurel, C., and Verdoucq, L. (2017). Aquaporins facilitate hydrogen peroxide entry into guard cells to mediate ABA- and pathogen-triggered stomatal closure. Proc. Natl. Acad. Sci. USA **114**:9200–9205.

Roelfsema, M.R.G., and Hedrich, R. (2005). In the light of stomatal opening: new insights into 'the Watergate'. New Phytol. **167**:665–691.

Roussin-Leveillee, C., Lajeunesse, G., St-Amand, M., Veerapen, V.P., Silva-Martins, G., Nomura, K., Brassard, S., Bolaji, A., He, S.Y., and Moffett, P. (2022). Evolutionarily conserved bacterial effectors hijack abscisic acid signaling to induce an aqueous environment in the apoplast. Cell Host Microbe **30**:489–501.e484.

Roy, D., Panchal, S., Rosa, B.A., and Melotto, M. (2013). Escherichia coli O157:H7 induces stronger plant immunity than Salmonella enterica Typhimurium SL1344. Phytopathology **103**:326–332.

Rui, Y., Xiao, C., Yi, H., Kandemir, B., Wang, J.Z., Puri, V.M., and Anderson, C.T. (2017). POLYGALACTURONASE INVOLVED IN EXPANSION3 Functions in Seedling Development, Rosette Growth, and Stomatal Dynamics in Arabidopsis thaliana. Plant Cell **29**:2413–2432.

Rzemieniewski, J., and Stegmann, M. (2022). Regulation of pattern-triggered immunity and growth by phytocytokines. Curr. Opin. Plant Biol. **68**, 102230.

Saito, S., and Uozumi, N. (2019). Guard Cell Membrane Anion Transport Systems and Their Regulatory Components: An Elaborate Mechanism Controlling Stress-Induced Stomatal Closure. Plants-Basel **8**:9.

Sakata, N., Ino, T., Hayashi, C., Ishiga, T., and Ishiga, Y. (2023). Controlling stomatal aperture, a potential strategy for managing plant bacterial disease. Plant Sci. **327**, 111534.

Sanguankiattichai, N., Buscaill, P., and Preston, G.M. (2022). How bacteria overcome flagellin pattern recognition in plants. Curr. Opin. Plant Biol. **67**, 102224.

Sano, S., Aoyama, M., Nakai, K., Shimotani, K., Yamasaki, K., Sato, M.H., Tojo, D., Suwastika, I.N., Nomura, H., and Shiina, T. (2014). Light-dependent expression of flg22-induced defense genes in Arabidopsis. Front. Plant Sci. **5**:531.

Schellenberg, B., Ramel, C., and Dudler, R. (2010). Pseudomonas syringae virulence factor syringolin A counteracts stomatal immunity by proteasome inhibition. Mol. Plant Microbe Interact. **23**:1287–1293.

Schmelz, E.A., Carroll, M.J., LeClere, S., Phipps, S.M., Meredith, J., Chourey, P.S., Alborn, H.T., and Teal, P.E.A. (2006). Fragments of ATP synthase mediate plant perception of insect attack. Proc. Natl. Acad. Sci. USA **103**:8894–8899.

Schulze, S., Yu, L., Hua, C., Zhang, L., Kolb, D., Weber, H., Ehinger, A., Saile, S.C., Stahl, M., Franz-Wachtel, M., et al. (2022). The Arabidopsis TIR-NBS-LRR protein CSA1 guards BAK1-BIR3 homeostasis and mediates convergence of pattern- and effector-induced immune responses. Cell Host Microbe **30**:1717–1731.e6.

Shan, L., He, P., Li, J., Heese, A., Peck, S.C., Nürnberger, T., Martin, G.B., and Sheen, J. (2008). Bacterial effectors target the common signaling partner BAK1 to disrupt multiple MAMP receptor-signaling complexes and impede plant immunity. Cell Host Microbe **4**:17–27.

Shen, J., Diao, W., Zhang, L., Acharya, B.R., Wang, M., Zhao, X., Chen, D., and Zhang, W. (2020). Secreted Peptide PIP1 Induces Stomatal Closure by Activation of Guard Cell Anion Channels in Arabidopsis. Front. Plant Sci. **11**:1029.

Shen, W., Liu, J., and Li, J.F. (2019). Type-II Metacaspases Mediate the Processing of Plant Elicitor Peptides in Arabidopsis. Mol. Plant **12**:1524–1533.

Shimazaki, K.i., Doi, M., Assmann, S.M., and Kinoshita, T. (2007). Light regulation of stomatal movement. Annu. Rev. Plant Biol. **58**:219–247.

Singh, B.K., Delgado-Baquerizo, M., Egidi, E., Guirado, E., Leach, J.E., Liu, H., and Trivedi, P. (2023). Climate change impacts on plant pathogens, food security and paths forward. Nat. Rev. Microbiol. **21**:640–656.

Steinbrenner, A.D., Muñoz-Amatriaín, M., Chaparro, A.F., Aguilar-Venegas, J.M., Lo, S., Okuda, S., Glauser, G., Dongiovanni, J., Shi, D., Hall, M., et al. (2020). A receptor-like protein mediates plant immune responses to herbivore-associated molecular patterns. Proc. Natl. Acad. Sci. USA **117**:31510–31518.

Su, J., Zhang, M., Zhang, L., Sun, T., Liu, Y., Lukowitz, W., Xu, J., and Zhang, S. (2017). Regulation of Stomatal Immunity by Interdependent Functions of a Pathogen-Responsive MPK3/MPK6 Cascade and Abscisic Acid. Plant Cell **29**:526–542.

Takahashi, Y., Bosmans, K.C., Hsu, P.K., Paul, K., Seitz, C., Yeh, C.Y., Wang, Y.S., Yarmolinsky, D., Sierla, M., Vahisalu, T., et al. (2022). Stomatal CO(2)/bicarbonate sensor consists of two interacting protein kinases, Raf-like HT1 and non-kinase-activity requiring MPK12/MPK4. Sci. Adv. **8**, eabq6161.

Takemiya, A., Sugiyama, N., Fujimoto, H., Tsutsumi, T., Yamauchi, S., Hiyama, A., Tada, Y., Christie, J.M., and Shimazaki, K.i. (2013). Phosphorylation of BLUS1 kinase by phototropins is a primary step in stomatal opening. Nat. Commun. **4**:2094.

Tanaka, K., and Heil, M. (2021). Damage-Associated Molecular Patterns (DAMPs) in Plant Innate Immunity: Applying the Danger Model and Evolutionary Perspectives. Annu. Rev. Phytopathol. **59**:53–75.

Tateda, C., Obara, K., Abe, Y., Sekine, R., Nekoduka, S., Hikage, T., Nishihara, M., Sekine, K.T., and Fujisaki, K. (2019). The Host Stomatal Density Determines Resistance to Septoria gentianae in Japanese Gentian. Mol. Plant Microbe Interact. **32**:428–436.

Thor, K., Jiang, S., Michard, E., George, J., Scherzer, S., Huang, S., Dindas, J., Derbyshire, P., Leitão, N., DeFalco, T.A., et al. (2020). The calcium-permeable channel OSCA1.3 regulates plant stomatal immunity. Nature **585**:569–573.

Tian, W., Wang, C., Gao, Q., Li, L., and Luan, S. (2020). Calcium spikes, waves and oscillations in plant development and biotic interactions. Nat. Plants **6**:750–759.

Tian, W., Hou, C., Ren, Z., Wang, C., Zhao, F., Dahlbeck, D., Hu, S., Zhang, L., Niu, Q., Li, L., et al. (2019). A calmodulin-gated calcium channel links pathogen patterns to plant immunity. Nature **572**:131–135.

Tian, W., Hou, C., Ren, Z., Pan, Y., Jia, J., Zhang, H., Bai, F., Zhang, P., Zhu, H., He, Y., et al. (2015). A molecular pathway for CO(2) response in Arabidopsis guard cells. Nat. Commun. **6**:6057.

Toyota, M., Spencer, D., Sawai-Toyota, S., Jiaqi, W., Zhang, T., Koo, A.J., Howe, G.A., and Gilroy, S. (2018). Glutamate triggers long-distance, calcium-based plant defense signaling. Science **361**:1112–1115.

Vahisalu, T., Kollist, H., Wang, Y.F., Nishimura, N., Chan, W.Y., Valerio, G., Lamminmäki, A., Brosché, M., Moldau, H., Desikan, R., et al. (2008). SLAC1 is required for plant guard cell S-type anion channel function in stomatal signalling. Nature **452**:487–491.

Wang, C., Zhang, J., Wu, J., Brodsky, D.E., and Schroeder, J.I. (2018a). Cytosolic malate and oxaloacetate activate S-type anion channels in Arabidopsis guard cells. New Phytol. **220**:178–186.

Wang, J., Song, W., and Chai, J. (2023). Structure, biochemical function, and signaling mechanism of plant NLRs. Mol. Plant **16**:75–95.

Wang, L., Einig, E., Almeida-Trapp, M., Albert, M., Fliegmann, J., Mithöfer, A., Kalbacher, H., and Felix, G. (2018b). The systemin receptor SYR1 enhances resistance of tomato against herbivorous insects. Nat. Plants **4**:152–156.

Wang, S., Sun, J., Fan, F., Tan, Z., Zou, Y., and Lu, D. (2016). A Xanthomonas oryzae pv. oryzae effector, XopR, associates with receptor-like cytoplasmic kinases and suppresses PAMP-triggered stomatal closure. Sci. China Life Sci. **59**:897–905.

Wang, S., Li, S., Wang, J., Li, Q., Xin, X.F., Zhou, S., Wang, Y., Li, D., Xu, J., Luo, Z.Q., et al. (2021). A bacterial kinase phosphorylates OSK1 to suppress stomatal immunity in rice. Nat. Commun. **12**:5479.

Wang, Y., Pruitt, R.N., Nürnberger, T., and Wang, Y. (2022). Evasion of plant immunity by microbial pathogens. Nat. Rev. Microbiol. **20**:449–464.

Wang, Y., Li, J., Hou, S., Wang, X., Li, Y., Ren, D., Chen, S., Tang, X., and Zhou, J.M. (2010). A Pseudomonas syringae ADP-ribosyltransferase inhibits Arabidopsis mitogen-activated protein kinase kinases. Plant Cell **22**:2033–2044.

Wei, W., Cui, M.Y., Hu, Y., Gao, K., Xie, Y.G., Jiang, Y., and Feng, J.Y. (2018). Ectopic expression of FvWRKY42, a WRKY transcription factor from the diploid woodland strawberry (Fragaria vesca), enhances resistance to powdery mildew, improves osmotic stress resistance, and increases abscisic acid sensitivity in Arabidopsis. Plant Sci. **275**:60–74.

Wei, Y., Balaceanu, A., Rufian, J.S., Segonzac, C., Zhao, A., Morcillo, R.J.L., and Macho, A.P. (2020). An immune receptor complex evolved in soybean to perceive a polymorphic bacterial flagellin. Nat. Commun. **11**:3763.

Wilton, M., Subramaniam, R., Elmore, J., Felsensteiner, C., Coaker, G., and Desveaux, D. (2010). The type III effector HopF2Pto targets Arabidopsis RIN4 protein to promote Pseudomonas syringae virulence. Proc. Natl. Acad. Sci. USA **107**:2349–2354.

Wu, F., Chi, Y., Jiang, Z., Xu, Y., Xie, L., Huang, F., Wan, D., Ni, J., Yuan, F., Wu, X., et al. (2020). Hydrogen peroxide sensor HPCA1 is an LRR receptor kinase in Arabidopsis. Nature **578**:577–581.

Wu, L., Huang, Z., Li, X., Ma, L., Gu, Q., Wu, H., Liu, J., Borriss, R., Wu, Z., and Gao, X. (2018). Stomatal Closure and SA-JA/ET-Signaling Pathways Are Essential for Bacillus amyloliquefaciens FZB42 to Restrict Leaf Disease Caused by Phytophthora nicotianae in Nicotiana benthamiana. Front. Microbiol. **9**:847.

Xin, X.F., Nomura, K., Aung, K., Velásquez, A.C., Yao, J., Boutrot, F., Chang, J.H., Zipfel, C., and He, S.Y. (2016). Bacteria establish an aqueous living space in plants crucial for virulence. Nature **539**:524–529.

Xu, G., Moeder, W., Yoshioka, K., and Shan, L. (2022). A tale of many families: calcium channels in plant immunity. Plant Cell **34**:1551–1567.

Xu, Q., Wang, J., Zhao, J., Xu, J., Sun, S., Zhang, H., Wu, J., Tang, C., Kang, Z., and Wang, X. (2020). A polysaccharide deacetylase from Puccinia striiformis f. sp. tritici is an important pathogenicity gene that suppresses plant immunity. Plant Biotechnol. J. **18**:1830–1842.

Xu, Z., Jiang, Y., Jia, B., and Zhou, G. (2016). Elevated-CO2 Response of Stomata and Its Dependence on Environmental Factors. Front. Plant Sci. **7**:657.

Yamaguchi, Y., Pearce, G., and Ryan, C.A. (2006). The cell surface leucine-rich repeat receptor for AtPep1, an endogenous peptide elicitor in Arabidopsis, is functional in transgenic tobacco cells. Proc. Natl. Acad. Sci. USA **103**:10104–10109.

Yamaguchi, Y., Huffaker, A., Bryan, A.C., Tax, F.E., and Ryan, C.A. (2010). PEPR2 is a second receptor for the Pep1 and Pep2 peptides and contributes to defense responses in Arabidopsis. Plant Cell **22**:508–522.

Yan, J., Yu, H., Li, B., Fan, A., Melkonian, J., Wang, X., Zhou, T., and Hua, J. (2019). Cell autonomous and non-autonomous functions of plant intracellular immune receptors in stomatal defense and apoplastic defense. PLoS Pathog. **15**, e1008094.

Yang, Y., Kim, N.H., Cevik, V., Jacob, P., Wan, L., Furzer, O.J., and Dangl, J.L. (2022). Allelic variation in the Arabidopsis TNL CHS3/CSA1 immune receptor pair reveals two functional cell-death regulatory modes. Cell Host Microbe **30**:1701–1716.e5.

Ye, W., Munemasa, S., Shinya, T., Wu, W., Ma, T., Lu, J., Kinoshita, T., Kaku, H., Shibuya, N., and Murata, Y. (2020). Stomatal immunity against fungal invasion comprises not only chitin-induced stomatal closure but also chitosan-induced guard cell death. Proc. Natl. Acad. Sci. USA **117**:20932–20942.

Yoshioka, K., Kachroo, P., Tsui, F., Sharma, S.B., Shah, J., and Klessig, D.F. (2001). Environmentally sensitive, SA-dependent defense responses in the cpr22 mutant of Arabidopsis. Plant J. **26**:447–459.

You, Y., Koczyk, G., Nuc, M., Morbitzer, R., Holmes, D.R., von Roepenack-Lahaye, E., Hou, S., Giudicatti, A., Gris, C., Manavella, P.A., et al. (2023). The eINTACT system dissects bacterial exploitation of plant osmosignalling to enhance virulence. Nat. Plants **9**:128–141.

Yu, X., Feng, B., He, P., and Shan, L. (2017). From Chaos to Harmony: Responses and Signaling upon Microbial Pattern Recognition. Annu. Rev. Phytopathol. **55**:109–137.

Yu, X., Xie, Y., Luo, D., Liu, H., de Oliveira, M.V.V., Qi, P., Kim, S.I., Ortiz-Morea, F.A., Liu, J., Chen, Y., et al. (2023). A phospho-switch constrains BTL2-mediated phytocytokine signaling in plant immunity. Cell **186**:2329–2344.e20.

Yuan, M., Ngou, B.P.M., Ding, P., and Xin, X.F. (2021). PTI-ETI crosstalk: an integrative view of plant immunity. Curr. Opin. Plant Biol. **62**, 102030.

Zandalinas, S.I., Fritschi, F.B., and Mittler, R. (2021). Global Warming, Climate Change, and Environmental Pollution: Recipe for a Multifactorial Stress Combination Disaster. Trends Plant Sci. **26**:588–599.

Zeng, W., and He, S.Y. (2010). A prominent role of the flagellin receptor FLAGELLIN-SENSING2 in mediating stomatal response to Pseudomonas syringae pv tomato DC3000 in Arabidopsis. Plant Physiol. **153**:1188–1198.

Zeng, W., Brutus, A., Kremer, J.M., Withers, J.C., Gao, X., Jones, A.D., and He, S.Y. (2011). A genetic screen reveals Arabidopsis stomatal and/or apoplastic defenses against Pseudomonas syringae pv. tomato DC3000. PLoS Pathog. **7**, e1002291.

Zhang, C., Xie, Q., Anderson, R.G., Ng, G., Seitz, N.C., Peterson, T., McClung, C.R., McDowell, J.M., Kong, D., Kwak, J.M., and Lu, H. (2013). Crosstalk between the circadian clock and innate immunity in Arabidopsis. PLoS Pathog. **9**, e1003370.

Zhang, D., Tian, C., Yin, K., Wang, W., and Qiu, J.-L. (2019). Postinvasive bacterial resistance conferred by open stomata in rice. Mol. Plant Microbe Interact. **32**:255–266.

Zhang, J., Coaker, G., Zhou, J.M., and Dong, X. (2020). Plant Immune Mechanisms: From Reductionistic to Holistic Points of View. Mol. Plant **13**:1358–1378.

Zhang, J., Wang, N., Miao, Y., Hauser, F., McCammon, J.A., Rappel, W.J., and Schroeder, J.I. (2018). Identification of SLAC1 anion channel residues required for CO(2)/bicarbonate sensing and regulation of stomatal movements. Proc. Natl. Acad. Sci. USA **115**:11129–11137.

Zhang, Y., Berman, A., and Shani, E. (2023). Plant Hormone Transport and Localization: Signaling Molecules on the Move. Annu. Rev. Plant Biol. **74**:453–479.

Zhang, Y., Goritschnig, S., Dong, X., and Li, X. (2003). A gain-of-function mutation in a plant disease resistance gene leads to constitutive activation of downstream signal transduction pathways in suppressor of npr1-1, constitutive 1. Plant Cell **15**:2636–2646.

Zhang, Z., Wu, Y., Gao, M., Zhang, J., Kong, Q., Liu, Y., Ba, H., Zhou, J., and Zhang, Y. (2012). Disruption of PAMP-induced MAP kinase cascade by a Pseudomonas syringae effector activates plant immunity mediated by the NB-LRR protein SUMM2. Cell Host Microbe **11**:253–263.

Zheng, X., Kang, S., Jing, Y., Ren, Z., Li, L., Zhou, J.M., Berkowitz, G., Shi, J., Fu, A., Lan, W., et al. (2018). Danger-Associated Peptides

Close Stomata by OST1-Independent Activation of Anion Channels in Guard Cells. Plant Cell **30**:1132–1146.

Zheng, X.Y., Spivey, N.W., Zeng, W., Liu, P.P., Fu, Z.Q., Klessig, D.F., He, S.Y., and Dong, X. (2012). Coronatine promotes Pseudomonas syringae virulence in plants by activating a signaling cascade that inhibits salicylic acid accumulation. Cell Host Microbe **11**:587–596.

Zhou, J., Wu, S., Chen, X., Liu, C., Sheen, J., Shan, L., and He, P. (2014). The Pseudomonas syringae effector HopF2 suppresses Arabidopsis immunity by targeting BAK1. Plant J. **77**:235–245.

Zhou, M., Wang, W., Karapetyan, S., Mwimba, M., Marqués, J., Buchler, N.E., and Dong, X. (2015a). Redox rhythm reinforces the circadian clock to gate immune response. Nature **523**:472–476.

Zhou, Y., Vroegop-Vos, I., Schuurink, R.C., Pieterse, C.M.J., and Van Wees, S.C.M. (2017). Atmospheric CO(2) Alters Resistance of Arabidopsis to Pseudomonas syringae by Affecting Abscisic Acid Accumulation and Stomatal Responsiveness to Coronatine. Front. Plant Sci. **8**:700.

Zhou, Z., Wu, Y., Yang, Y., Du, M., Zhang, X., Guo, Y., Li, C., and Zhou, J.M. (2015b). An Arabidopsis Plasma Membrane Proton ATPase Modulates JA Signaling and Is Exploited by the Pseudomonas syringae Effector Protein AvrB for Stomatal Invasion. Plant Cell **27**:2032–2041.

Zhu, Y., Qian, W., and Hua, J. (2010). Temperature modulates plant defense responses through NB-LRR proteins. PLoS Pathog. **6**, e1000844.

Zuch, D.T., Doyle, S.M., Majda, M., Smith, R.S., Robert, S., and Torii, K.U. (2022). Cell biology of the leaf epidermis: Fate specification, morphogenesis, and coordination. Plant Cell **34**:209–227.