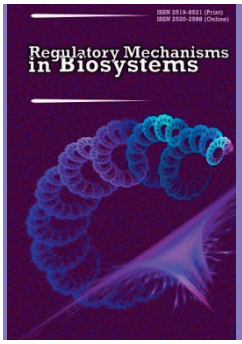

Regulatory Mechanisms in Biosystems

ISSN 2519-8521 (Print)
ISSN 2520-2588 (Online)
Regul. Mech. Biosyst.,
2024, 15(3), 527–542
doi: 10.15421/022474

The involvement of phytohormones in plant–pathogen interaction

N. R. Amrahov*, **, ***, G. A. Aghazada*, S. R. Alizada*, G. V. Mehdiyeva*,
R. B. Mammadova****, *****, S. A. Alizade*, ****, Z. M. Mammadov*

**Baku State University, Baku, Azerbaijan*
***Azerbaijan Research Institute of Crop Husbandry, Ministry of Agriculture, Baku, Azerbaijan*
****GPOGC SRI, Azerbaijan State Oil and Industry University, Baku, Azerbaijan*
*****Genetic Resources Institute, Ministry of Science and Education Republic of Azerbaijan, Baku, Azerbaijan*
******Western Caspian University, Baku, Azerbaijan*

Article info
Received 26.05.2024
Received in revised form 01.07.2024
Accepted 17.07.2024

Baku State University, Academic Zahid Khalilov st., 23, Baku, AZ1148, Azerbaijan. Tel.: +994-12-539-05-17. E-mail: nurlanamrahov@bsu.edu.az

Azerbaijan Research Institute of Crop Husbandry, Ministry of Agriculture, Pirshagi, Sovkhoz 2, Baku, AZ 1098, Azerbaijan. Tel.: +994-12-551-61-30. E-mail: nurlan00686@gmail.com

GPOGC SRI, Azerbaijan State Oil and Industry University, Dilara Aliyeva st., 227, Baku, AZ1010, Azerbaijan. Tel.: +994-12-493-45-57. E-mail: nurlan00686@gmail.com

Genetic Resources Institute, Ministry of Science and Education Republic of Azerbaijan, Azadlig Ave 155, Baku, AZ1106, Azerbaijan. Tel.: +994-12-449-92-21. E-mail: gene-res@mail.ru

Western Caspian University, Istiglaliyyat st., 31, Baku, AZ1001, Azerbaijan. Tel.: +994-12-492-74-18. E-mail: gene-res@mail.ru

Amrahov, N. R., Aghazada, G. A., Alizada, S. R., Mehdiyeva, G. V., Mammadova, R. B., Alizade, S. A., & Mammadov, Z. M. (2024). The involvement of phytohormones in plant–pathogen interaction. Regulatory Mechanisms in Biosystems, 15(3), 527–542. doi:10.15421/022474

Plant–pathogen interactions involve intricate signaling networks that coordinate the plant immune response. Recognition of pathogens through pattern recognition receptors (PRRs) triggers activation of mitogen-activated protein kinase (MAPK) pathways, initiating a cascade of defense mechanisms. Central to these responses is the synthesis of phytohormones such as salicylic acid (SA), auxins–indole-3-acetic acid (IAA), and gibberellins–gibberellic acid (GA), pivotal for immune activation. This review explores the multifaceted roles of these phytohormones in plant immunity, drawing on recent findings from *Arabidopsis thaliana* and *Gossypium hirsutum* studies. The review discusses MAPK-mediated activation of TGA1/4 (TGACG sequence-specific binding protein 1/4) transcription factors enhancing SA biosynthesis via isochorismate synthase (ICS). Increased SA levels activate NPR1, promoting gene expression in immune-related pathways including systemic acquired resistance (SAR). Concurrently, pathogen-induced IAA synthesis activates auxin-responsive genes crucial for immune responses. Elevated biosynthesis of IAA from L-tryptophan activates these genes by degrading repressor molecules. IAA acts antagonistically to SA, conserving energy during pathogen infection. Additionally, GA is vital for plant growth and development, operating DELLA (Asp–Glu–Leu–Leu–Ala) protein degradation with the formation of a complex with gibberellin insensitive dwarf 1 (GID1). Once DELLA prevents releasing GA-related response reactions, it is extremelly crucial for GA actions. In general, the review explores the intricate interplay between SA, IAA, and GA, highlighting SA's antagonistic regulation of GA signaling and the synergistic effects of auxin and GA. Understanding these hormone–mediated pathways is crucial for elucidating precise mechanisms underlying plant immunity. Insights gained could inform strategies to enhance plant resistance against pathogens, contributing to sustainable agriculture and global food security efforts.

Keywords: plant-pathogen interaction; salicylic acid; indole-3-acetic acid; gibberellic acid; *Arabidopsis thaliana*; *Gossypium hirsutum*.

Plant–pathogen interaction

In the modern agricultural industry, much attention is paid to the interaction of fungal microorganisms with plants, since pathogenic fungi cause the reduction of plant yields on a global scale. The interaction of fungi with plants is closely related to the immune response of plants against pathogenic fungi (Heuberger et al., 2014; Hua et al., 2018). Numerous attempts have been made to apply more contemporary methods to eliminate possible disruptive actions of these pathogens on plant organisms (Mehdiyeva et al., 2023).

Pathophysiological and biochemical stimulation of a plant cell by a pathogenic fungus in most cases occurs with the participation of chitin, a homopolymer of N-acetylglucosamine units that forms the cell wall of fungus. Through the cell wall, the fungal pathogen is in close contact with its host plant (Langner & Göhre, 2016). The absence of chitin or its derivatives in plant cells allows them to identify chitin as a foreign substance and initiate an immune response against fungal pathogens (Shibuya et al., 1993). This mechanism includes both mechanical protective reaction (cuticle, waxy layer on the surface of leaves and other parts of plants) and biochemical, through the activation of enzymes with antimicrobial activity and the inclusion of apoptotic processes in the area of contact of the plant cell with the pathogen) (Osbourn, 1996; Rashid et al., 2019; Amrahov et al., 2023). Ultimately, all processes lead to the activation of systemic acquired resistance (SAR), which targets a wide range of pathogens.

The primary contact of a fungal pathogen with the surface of a plant cell occurs through the involvement of surface receptors–pattern recognition receptors (PRR), responsible for recognizing the chitin of the fungal pathogen, or more precisely for recognizing microbial-associated molecular patterns (MAMP) and pathogen-associated molecular patterns (PAMPs) (Kombrink et al., 2011; Shinya et al., 2015; Vázquez–Hernández et al., 2019).

Regardless of infection strategy and lifestyle, whether the fungal pathogen was biotrophic, necrotrophic, or hemibiotrophic (Fernandez et al., 2014; Presti et al., 2015), an immune response strategy involving chitin as an elicitor was envisioned for all pathogen identifications. It was revealed that this group of receptors are membrane-localized lysin motif (LysM) proteins, which have a high affinity for chitin oligosaccharides (Kaku et al., 2006). In studies conducted on the model rice plant, a receptor-like

protein OsCEBiP was identified, this protein had a high affinity for chitin oligosaccharides of the pathogen, which indicated its biological activity and function (Kaku et al., 2006; Li et al., 2019). OsCEBiP binds to the intracellular protein OsCERK1 and LysM receptor-like kinase (RLK), which is responsible for transmitting a signal into the cell and this occurs after the interaction of 2 OsCEBiP molecules with 1 molecule of chitin oligosaccharide (Buist et al., 2008; Shimizu et al., 2010; Akamatsu et al., 2013). Different intracellular proteins responsible for chitin signal transduction have been identified in different organisms, such as AtCERK1 (AtLYK1) in *Arabidopsis thaliana* (Iizasa et al., 2010; Petutschnig et al., 2010). At the same time, extracellular proteins responsible for the chitin affinity, such as AtLYK4, AtLYK5, and the ortholog OsCEBiP, AtLYM2, also showed the ability to bind to chitin of various pathogens, while interacting with the intracellular protein AtCERK1 (Liu et al., 2012; Shinya et al., 2012; Cao et al., 2014). Similar homologues of OsCEBiP, MtLYM2 were identified in *Medicago truncatula*, and HvCEBiP – in barley (Tanaka et al., 2010; Fliegmann et al., 2011). Identical results were also demonstrated in wheat plants, in which blocking CEBiP and CERK1 analogues led to a decrease of immune resistance against pathogenic fungi (Lee et al., 2014). Chitin perception will follow the activation of extracellular and intracellular proteins and will thereby trigger signal transduction to the downstream signaling system through autophosphorylation of the cell domain of the CERK1 protein (Petutschnig et al., 2010). This protein has different homologs, identical to the CEBiP protein. In particular, we can highlight the NbCerk1 protein, already identified in *Nicotiana benthamiana,* which is a homolog of AtCERK1 from the *Arabidopsis thaliana* plant. In the subsequent process, a cascade reaction is initiated through mitogen-activated protein kinases (MAPKs), responsible for transmitting a signal to the nucleus for the expression of genes responsible for the production of protective proteins (Fig. 1a) associated with pathogenesis (PR) and other secondary metabolites (Yamaguchi et al., 2013; Shinya et al., 2014).

The ability of a host plant to recognize a fungal pathogen closely competed with the ability of the pathogen itself to destabilize microbe-associated molecular pattern (MAMP)-triggered immunity (MTI) (Jones & Dangl, 2006; Panstruga & Dodds, 2009). This process is associated with the ability of a successful fungal pathogen to produce effectors that disrupt MTI function, thereby increasing the susceptibility of the plant cell to the fungal pathogen (Panstruga & Dodds, 2009; De Jonge et al., 2011).

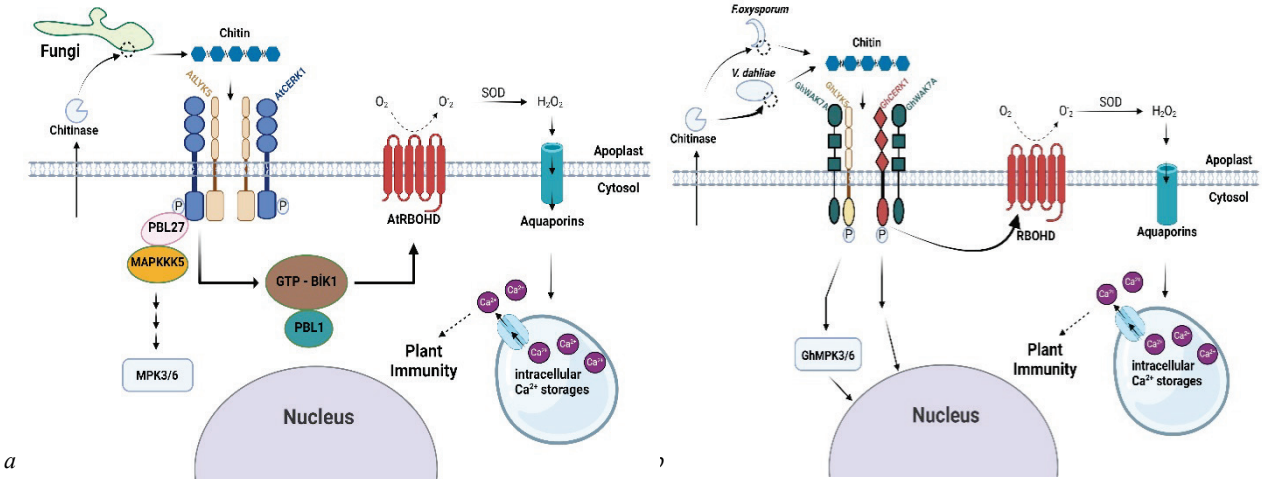

Fig. 1. The process of formation of the response after the contact of fungal elicitors with corresponding receptors: *a* – in *Arabidopsis thaliana* and *b* – in upland cotton – *Gossypium hirsutum*

Chitinase, being a protective component of plants, is mainly localized apoplastically and is responsible for the production of soluble components of fungal chitin (Han et al., 2019). Chitin, produced by the interaction of chitinase and the pathogenic fungal membrane, is a critical factor in the recognition of invading fungal pathogens (Pusztahelyi, 2018). In *Arabidopsis thaliana*, AtLYK5 receptors and AtCERK1 coreceptors are responsible for chitin recognition and signal transmission from the intermembrane space to the intracellular space (Han et al., 2019; Yang et al., 2022). Kinase domains of these receptors are involved in further transmission of extracellular pathogen-plant interaction, either through the GTP-BIK 1 complex (with the direct participation of PBL1 to NADPH oxidase (AtRBOHD)), or through activation of the MAPK cascade pathway, by preliminary signal transmission to PBL 27 and to the first component of the MAPK cascade pathway, MAPKKK5 (Liu et al., 2013; Yamada et al., 2016). Activation of AtRBOHD leads to the production of superoxide radical, which, being a substrate of SOD (superoxide dismutase), forms hydrogen peroxide (Torres et al., 2002). Hydrogen peroxide, penetrating through the aquaporins of the plasma membrane, provokes the release of calcium ions into the cytoplasm from the intracellular storage vacuole (Dangol et al., 2019; Mukherjee et al., 2024).

In *Gossypium hirsutum*, fungal pathogens of upland cotton; *Verticillium dahlia* and *Fusarium oxysporium*, were exposed to the plant's extracellular enzyme chitinase, which led to the cleavage of the pathogen's extracellular membrane and the release of chitin (Man et al., 2022). Receptors GhWAK7A, GhLYK5, and co-receptors GhCERK1 are responsible for recognizing chitin and transmitting a signal from the intermembrane space to the intracellular space (Wang et al., 2020). The rest of the signaling via intracellular domains is similar to the process in Arabidopsis. The exception is the types of MAPK kinases, which in upland cotton is GhMPK3/6 (Zhang et al., 2016). The process of formation of free radicals and their degradation, as well as the transfer of hydrogen peroxide into the cell with the involvement of the vacuole in the immune reaction, is similar in both plants. Calcium cations, being signaling molecules, are released from the vacuole into the cytoplasm (Fig. 1b), this cation directly triggers an additional immune defense reaction of plants (Gaspar et al., 2002; Jiang at al., 2013).

The process of adaptive reactions of plants, combined with an immune response against an external stressor, involves the initial diagnosis of the stressor, information about which is then transmitted to other cells and tissues through various signaling mechanisms (Sukhova & Sukhov, 2021). This process is necessary to coordinate the biochemical and physiological functions of different plant organs and tissues (Shah & Zeier, 2013; Hilleary & Gilroy, 2018; Farmer et al., 2020; Sukhova & Sukhov, 2021; Alizade et al., 2023; Mammadova et al., 2024). Depending on the concentration and type of stressor, signals can be divided into several groups: signals of a chemical nature (glutamate (Qiu et al., 2020), peptides and small proteins (Takahashi et al., 2019), phytohormones (Roychoudhury & Aftab, 2021; Tripathi et al., 2021)), signals created by reactive oxygen species (ROS) (Aliyeva et al., 2023; Amrahov et al., 2023), and transmitted with the participation of apoplasts (Suzuki & Mittler, 2012; Qi et al., 2017), Ca^{2+} induced signaling pathway (Choi et al., 2014; Kiep et al., 2015), electrical signals (Fromm & Lautner, 2007, Gallé et al., 2015; Hedrich et al., 2016; Sukhova & Sukhov, 2021), and hydraulic waves (Christmann et al., 2007; Christmann et al., 2013). Against the adverse effects of a variety of stressors, some most comprehensive research works are currently conducted, even with the use of nanoparticles (Ahmadov et al., 2020; Baran et al., 2023; Olatunbosun et al., 2023; Muradov et al., 2024; Vorobyova et al., 2024).

Effect of biotic stressor on salicylic acid biosynthesis

Activation of signaling pathways also involves phytohormones. Once the pathogen infection activates the MAPK pathway, some components of this mechanism enhance the upregulation of certain transcription factor genes, such as TGA 1/4, which in turn activates key transcription factor genes for salicylic acid synthesis, like systemic acquired resistance deficient (SARD1) and cam binding protein60-like g (CBP60g) inside the nucleus. Salicylic acid (SA) serves as a pivotal phytohormone, intricately involved in local defense mechanisms and systemic acquired resistance (SAR), as corroborated by Vlot et al. (2009) and Zhang & Li (2019). In the model *Arabidopsis* plants, when faced with pathogen infection, there is a substantial surge in SA levels due to the activated SA biosynthesis primarily through the isochorismate pathway which is affected by SARD1 and CBP60g (Wildermuth et al., 2001; Rekhter et al., 2019; Liu et al., 2020). In *Arabidopsis thaliana*, approximately 10% of SA associated with defense responses is generated through the cytosolic phenylalanine ammonia lyase pathway (Fig. 2), while the remaining 90% is derived from isochorismate, a product of the plastid-localized isochorismate synthase 1 (ICS1) (Rekhter et al., 2019).

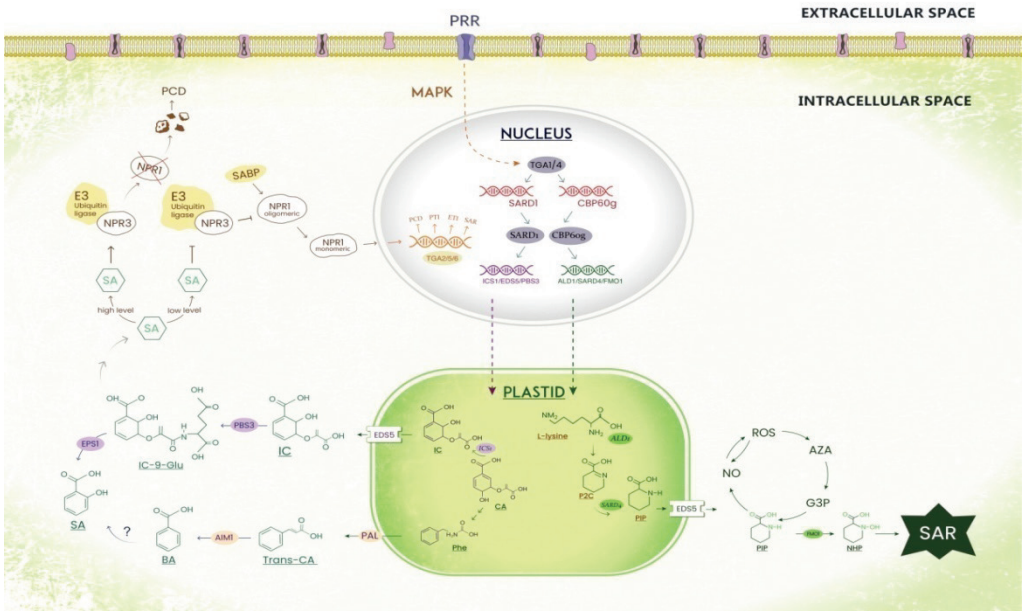

Fig. 2. Salicylic acid biosynthesis and its effector systems: PRR – pattern recognition receptors, MAPK – mitogen–activated protein kinase, TGA1/4 – TGACG sequence-specific binding protein 1/4, SARD1 – systemic acquired resistance deficient 1, Cbp60g – cam binding protein 60-like g, ICS1 – isochorismate synthase 1, EDS5 – enhanced disease susceptibility 5, PBS3 – AvrPphB susceptible 3, ALD1 – AGD2-like defense response protein 1, SARD4 – systemic acquired resistance deficient 4, FMO1 – Flavin-dependent monooxygenase 1, P2C – Δ1-piperideine-2-carboxylic acid, Pip – pipecolic acid, NHP – N-hydroxypipecolic acid, SAR – systemic acquired resistance, G3P – glycerol-3-phosphate, Aza – azelaic acid, ROS – reactive oxygen species, NO – nitric oxide, CA – chorismate, IC – isochorismate, Ic-9-Glu – isochorismate-9-glutamate, EPS1 – enhanced *Pseudomonas* susceptibility 1, SA – salicylic acid, Phe – phenylalaine, PAL – phenylalanine ammonia-lyase, Trans-CA – trans-cinnamic acid, AIM1 – abnormal inflorescence meristem 1, BA – benzoic acid, NPR3 – nonexpresser of PR genes 1-like protein 3, NPR1 – nonexpresser of PR genes 1, SABP – salicylic acid-binding proteins, (TGA2/5/6) – TGACG-sequence-specific binding protein2/5/6, PCD – programmed cell death, PTI – pattern-triggered immunity, ETI – effector triggered immunity

The expression of the SA-deficient 2 (SID2) gene, responsible for encoding the enzyme ICS1 involved in SA biosynthesis was notably facilitated by the SARD1 and CBP60g (Wang et al., 2009, 2011; Zhang et al., 2010; Liu et al., 2020). Additionally, SARD1 and CBP60g also regulate the transcription of two other SA biosynthesis genes, PBS3 and EDS5 (Sun et al., 2015; Liu et al., 2020).

Once isochorismate is synthesized from chorismate by the key enzyme ICS1 within the plastid, these newly produced molecules must be transported to the cytosol. In this crucial step, the multidrug and toxin extrusion (MATE) family transporter EDS5, localized in the chloroplast envelope, plays a pivotal role. EDS5 facilitates the export of isochorismate from the plastid into the cytosol, where PBS3 converts it into isochorismate-9-glutamate. Subsequent nonenzymatic breakdown leads to the generation of SA. Remarkably, this pathway involves only three essential proteins (ICS1, EDS5, and PBS3), their spatial separation, and unidirectional flux, potentially offering protection against evolutionary pressures and pathogenic effectors (Rekhter et al., 2019).

The loss of either SARD1 or CBP60g results in a drastic reduction in ICS1 accumulation and SA production (Liu et al., 2020). Similarly, plants carrying mutations in PBS3 gene also exhibit a reduced SA amount in response to pathogen infection. Although PBS3 has relatively low activity with SA as a substrate, the transcriptional co-regulation of PBS3 with ICS1 and EDS5 implies that all three gene products contribute in a coordinated manner to the modification, transport, or production of SA or its precursors (Liu et al., 2020).

The 2,5-dihydroxybenzoic acid (2,5-DHBA) assumes a significant role as a major catabolite of SA and its formation, so it plays a pivotal role in the intricate regulation of SA levels (Zhang et al., 2017; Liu et al., 2020). This regulatory process is crucial for maintaining SA homeostasis. At the heart of this regulation is the downy mildew resistant 6 (DMR6) gene, which encodes an SA-5-hydroxylase (S5H) responsible for the conversion of SA to 2,5-DHBA (Zhang et al., 2017; Liu et al., 2020). In plants carrying mutations in the DMR6 gene, the conversion of SA to 2,5-DHBA is impeded, resulting in elevated SA levels and heightened disease resistance (Van Damme et al., 2008; Zhang et al., 2017).

During the senescence process, *Arabidopsis* plants also accumulate substantial levels of 2,3-DHBA, a metabolite of SA. This conversion is catalyzed by an SA 3-hydroxylase (S3H) enzyme that shares similarity with DMR6/S5H (Zhang et al., 2013). In plants carrying a double mutation affecting both S5H and S3H (s5h s3h double mutant), SA levels increase even further than the dmr6 single mutant. This excessive SA accumulation leads to severe dwarfism and autoimmune responses, as evidenced by Zhang et al. (2017). Despite the critical role of DMR6 in the maintenance of SA levels, the mechanisms governing its transcriptional regulation remain a subject of inquiry and are currently not well understood (Liu et al., 2020).

İn the model plant *Arabidopsis* (*Arabidopsis thaliana*), intracellular accumulation of SA molecules' recognition hinges upon two receptor classes: NPR1 and NPR3/NPR4. These receptors orchestrate two parallel signaling pathways, thereby orchestrating the activation of defense-related

genes and the enhancement of immunity (Fu et al., 2012; Wu et al., 2012; Ding et al., 2018).

Using a unique equilibrium dialysis ligand binding technique, Wu et al. (2012) provided significant proof in the search for SA receptors, suggesting that NPR1 itself serves as a SA receptor. On the other hand, Fu et al. (2012) reported an interesting discovery, showing that in standard ligand binding tests, NPR1 failed to attach to SA directly. Rather, it was discovered that two similar proteins, NPR3 and NPR4, were SA receptors. These proteins connect to SA and coordinate its signaling processes (Attaran & He, 2012; Fu et al., 2012).

Following their inability to find SA associating to NPR1 through traditional receptor binding assays, Fu and colleagues embarked on a series of experiments to gain deeper insights into the dynamic turnover of NPR1 within the cell (Attaran & He, 2012; Fu et al., 2012). This work built upon their earlier research, revealing key structural attributes of NPR1. Broad-Complex, Tramtrack and Bric-a-brac/Pox virus and zinc finger (BTB/POZ) domains at the N-terminal end, a main ankryin repeat site, and a transactivation-capable C-terminal site make up NPR1. Interestingly, it has been discovered that certain proteins with the BTB domain connect to the Cullin 3 (CUL3) E3 ligase, making it easier for the 26S proteasome to recognize substrates, ubiquitinate them, and ultimately break them down. Expanding on this realization, a set of tests revealed that NPR4 and NPR3 relate to NPR1 and CUL3, serving as essential CUL3 adaptors for NPR1 breakdown that is regulated (Attaran & He, 2012). Based on their intriguing findings, Fu and their team further proposed a hypothesis elucidating the roles of NPR receptors in plants with a basal level of SA (Fu et al., 2012). NPR4, a component of the CUL3-NPR4 ubiquitin ligase complex, binds to NPR1 in healthy plants, where SA amounts stay at the initial state, making it easier to remove NPR1 protein. The start of energy-intensive defense responses is successfully prevented by this interaction. On the other hand, SA levels rise in diseased tissues as well as throughout the plant in response to pathogenic risks, with the greatest levels found at the location of infection. In the infected tissue, the elevated SA concentration increases the association of NPR3 with NPR1, triggering the breakdown of NPR1 and subsequently leading to PCD at the site of the pathogenic assault. The primary reason for NPR1 degradation being essential for PCD release was revealed in their previous research. They observed that the mutated forms–npr3npr4, characterized by elevated NPR1 levels, fail to undergo local tissue PCD following infection with avirulent bacteria. In contrast, in uninfected parts, such as uninfected leaves, where SA accumulates at comparatively lower levels than the infection part, NPR1 interactions with both NPR3 and NPR4 weaken. This results in the collection of NPR1, the suppression of programmed cell death (PCD), the triggering of genes related to defence action, and the activation of SAR (Attaran & He, 2012).

As the relevant amount of SA is recognized by NPR receptors, NPR1 must be transferred from the cytosol into the nucleus. NPR1 exhibits duality in its cellular existence, existing in at least two distinct forms. Under conditions of low SA concentration within the cell, as observed in the absence of pathogen infection, NPR1 adopts an oligomeric state within the cytoplasm, facilitated by intermolecular disulphide bonds. Conversely, when SA levels are elevated, particularly following pathogen incursion, redox changes within the cytosol facilitate the reduction of these disulphide bonds, causing NPR1 to transition into a monomeric form. In this monomeric state, NPR1 translocates into the nucleus, where it assumes its role as a transcriptional co-activator (Mou et al., 2003).

Four SA-binding proteins (SABPs) discovered in tobacco play a pivotal role in this translocation. They include a catalase, a methyl salicylate esterase known as SABP2, and others (Vlot et al., 2009; Attaran & He, 2012). For instance, SABP2 stands out due to its remarkably great sensitivity for SA. This protein utilizes methyl salicylate (MeSA) as a substrate, catalyzing its transformation into SA. Additionally, catalase and ascorbate peroxidase are believed to play roles in redox alterations following exposure to SA (Attaran & He, 2012). These alterations could potentially lead to the disassembly of NPR1 oligomers into monomeric forms, and help the movement of NPR1 into the nucleus (Tripathi et al., 2010).

After entering the nucleus, NPR1, lacking DNA binding domains, requires interaction with the TGACG sequence-specific binding protein (TGA) transcription factors, specifically TGA2, TGA5, and TGA6, for effective signal transduction. (Zhang et al., 1999; Després et al., 2000; Zhou et al., 2000; Zhang et al., 2006; Liu et al., 2020). The *Arabidopsis* TGA ensemble, comprising 10 members, is taxonomically stratified into five clades. Of particular interest, clade I encompasses TGA 1 and 4, while clade II is comprised of TGA 2, 5 and 6 (Gatz, 2013).

Significantly, TGA2, TGA5, and TGA6 exhibit functional redundancy in mediating immunity against necrotrophs, and biotrophic plant pathogens, contributing to SAR, and governing the mitigation of oxidative stress induced by factors such as UV-B radiation and reactive oxylipins (Zhang et al., 2003; Zander et al., 2010; Stotz et al., 2013; Herrera–Vásquez et al., 2021). NPR1 is a transcriptional co-activator for the TGA transcription factors, which are identified by the presence of a basic region/leucine zipper motif. It also produces an advanced control protein that coordinates SA-dependent defensive reactions (Attaran & He, 2012).

In contrast to NPR1, NPR3/NPR4 plays a key role as transcriptional repressors in the lacking of pathogen influence, suppressing the expression of SA-related genes (Attaran & He, 2012). On the other hand, when SA contacts with NPR3/NPR4, it hinders their ability to act as transcriptional repressors. This leads to the release of their target genes from repression and the initiation of defense mechanisms (Ding et al., 2018; Liu et al., 2020).

Fu et al. (2012) continued their investigation by assessing the binding affinities of SA for NPR3 and NPR4 through conventional ligand-receptor binding assays (Attaran & He, 2012). Interestingly, their discoveries revealed significant differences in salicylic acid (SA) binding affinities between the two receptors. NPR4 exhibited an exceptionally low dissociation constant (Kd) of 46.2 ± 2.35 nM, indicative of high SA affinity. In stark contrast, the Kd value for NPR3 was notably elevated, rendering accurate estimation challenging, but it is believed to hover around ~1000 nM. Furthermore, it was observed that SA interacts with multiple sites on both NPR3 and NPR4.

Besides SA, N-hydroxypipecolic acid (NHP) and pipecolic acid (Pip) assume pivotal roles in plant immune signaling, playing an essential role in SAR (Chen et al., 2018; Hartmann et al., 2018; Liu et al., 2020). In *Arabidopsis*, Pip is obtained through a two-step process, originating from L-Lys (Ding et al., 2016; Hartmann et al., 2017; Hartmann et al., 2018). The aminotransferase AGD2-like defense response protein 1 (ALD1) catalyzes the removal of the α-amino group from L-lys, forming 2,3-dehydropipecolic acid (2,3-DP). Consequently, 2,3-DP is converted into Pip through the action of SARD4 and an another reductive reaction (Hartmann et al., 2017; Hartmann et al., 2018). FMO1 further transforms Pip to produce NHP, a mobile signaling molecule highly likely to be involved in SAR (Chen et al., 2018; Hartmann et al., 2018; Liu et al., 2020). Upon pathogen invasion, the ALD1, SARD4, and FMO1 gene expressions are markedly induced, resulting in an increased production of Pip and NHP (Liu et al., 2020). Additionally, it is probable that the transcriptional regulation of NHP synthesis implicates SARD1 and CBPG60. This assumption arises from chromatin immunoprecipitation analyses suggesting that these factors effect the ALD1 and FMO1 gene promoters (Sun et al., 2015; Hartmann et al., 2018).

An intriguing observation from the analysis of the SA perception-deficient npr1-1 npr4-4D double mutant is that SA perception is crucial for the induction of genes involved in NHP biosynthesis and the subsequent production of NHP during pathogen infection (Liu et al., 2020). This likely occurs through SA-mediated upregulation of SARD1 and CBP60g, two transcription factors that directly govern the expression of NHP biosynthesis genes, including ALD1, SARD4, and FMO1, (Sun et al., 2015, 2018; Liu et al., 2020). Given that NHP serves as a mobile signal for SAR, one of SA's contributions to SAR is the induction of this mobile signal in local tissue (Liu et al., 2020).

The investigations revealed that NHP leads to SAR through a central transcriptional module comprising TGA2/5/6 and NPR1. This module is affected by TGA1/4 to transduce elevated NHP levels into SAR. It is worth noting that NHP has been indicated to enhance the expression of SARD1, CBP60g, and SA biosynthetic genes, likely promoting SA biosynthesis and thus activating SA-mediated immunity (Liu et al., 2020).

The characteristics of SAR-activated plants suggest that they proactively prepare for future pathogen attacks by orchestrating the upregulation of genes involved in various stages of defense signaling, encompassing

elicitor perception, signal transduction, and transcriptional gene activation. Concurrently, these plants downregulate processes related to photosynthesis and growth-associated functions (Bernsdorff et al., 2016).

In summary, NPR1, NPR3 and NPR4 indicated as SA receptors exhibit multiple roles in the plant immune system by overseeing the regulation of SA-responsive defense-related genes. The perception of SA not only promotes both PTI and ETI but is also indispensable for activating NHP production in local tissue, thereby inducing NHP-triggered defense responses during SAR (Bernsdorff et al., 2016).

Several SAR stimulators, such as SA (Gaffney et al., 1993), azelaic acid (C9 dicarboxylic acid, AzA) (Jung et al., 2009; Yu et al., 2013), nitric oxide (NO), glycerol-3-phosphate (G3P) (Chanda et al., 2011; Yu et al., 2013), reactive oxygen species (ROS) (Wang et al., 2014; Wendehenne et al., 2014; Kachroo et al., 2016), and galactolipids (Gao et al., 2014) have been determined (Wang et al., 2018). Recent studies have revealed the existence of a linear pathway involving NO⇔ROS⇒AzA⇒G3P, which operates in parallel with SA-derived signaling and is crucial for SAR activation (Wang et al., 2018). The initiation of ROS production leads to the catalytic oxidation of unbound C_{18}-unsaturated fatty acids that have been liberated from membrane lipids. The resultant oxidation produces AzA, which subsequently induces the biosynthesis of G3P by activating the genes responsible for encoding enzymes involved in G3P biosynthesis (Wang et al., 2018). In order to diminish undesired amount of nitrate species, which leads to releasing negative consequences on ROS amount, some research approaches are proposed (Valiyeva et al., 2022).

Short-distance cell-to-cell SA transport

The efficient transport of SA from the site of infection to distant tissues is a crucial aspect of SAR, which is influenced by the water potential within the infected tissue. Recent researches suggest that the movement of SA from the primary infected tissue to distant locations likely occurs through the apoplast, the space situated between the cell wall and the plasma membrane (Lim et al., 2016, 2020). When plants are infected by pathogens, there is a notable increase in the accumulation of SA in the apoplastic compartment. This accumulation appears to be independent of any deficiencies in symplastic transport through plasmodesmata (PD). In contrast to SA, other chemical signals associated with SAR, such as G3P and AzA, are primarily transported through PD (Lim et al., 2016, 2020). Disruptions or defects in the permeability of PD can hinder the efficient transport of these SAR-related signals from the infected tissue to distant tissues. AzA operates in a position preceding G3P, with both the AzA-G3P pathway and SA-derived signaling pathways functioning concurrently during the process of SAR (Chanda et al., 2011; Yu et al., 2013; Gao et al., 2014; Wang et al., 2014; Lim et al., 2016, 2020). Pip, another inducer of SAR, acts at a level preceding the AzA-G3P pathway, enhancing SAR by stimulating the production of free radicals (Wendehenne et al., 2004; Návarová et al., 2012; Wang et al., 2014; Wang et al., 2018; Lim et al., 2020). The exact relationship between NHP and other SAR signals remains a topic of current uncertainty and ongoing research (Lim et al., 2020). Significantly, the transportation of both SA and G3P plays a crucial role in facilitating the accumulation of Pip in distant plant tissues (Wang et al., 2018; Lim et al., 2020). Consequently, the synchronized transport and feedback regulation of these diverse chemical signals ultimately activates SAR in plants (Lim et al., 2020).

Exogenous SA application and long-distance SA transport

Controversially, applying SA externally, whether through seed priming (soaking seeds with SA solution before planting), adding SA to a hydroponic solution, or spraying plants with an SA solution, benefits plant growth and protection them against abiotic stresses by triggering SAR (Hayat et al., 2010; Maruri-López et al., 2019). The primary challenge lies in how plant cells accept externally introduced salicylic acid. Spraying SA allows upperground cells, like those in leaves, to absorb it, whereas applying SA through a hydroponic solution leads to uptake by root cells. Subsequently, the absorbed SA particles are transmitted through the plant's vascular system for long-distance transport.

SA moves freely within the plant, primarily using the phloem as its transportation route. SA molecules present in the phloem sap are obtained from SA accumulated cells via the phloem apoplast, known as apoplastic loaders (Bonnemain et al., 2013). The loading of SA into the phloem depends on the pH. Given SA's low pKa value (2.98), the COOH group is mostly deprotonated (COO-) in the cytosol, which tends to be neutral to slightly alkaline (pH 7.0–7.5) (Kim & Lim, 2023). To prevent swift increases in cytosolic pH due to protons released while SA accumulates, a regulated amount of SA might be moved to the apoplast, based on pH levels. This implies that the acidic pH in the apoplast significantly aids the absorption of apoplastic SA in distant tissues. This concept further proposes that transporting SA across the plasma membrane could involve a proton pump (Lim et al., 2020; Kim & Lim, 2023).

In the later case of SA, it needs to traverse the plasma membrane of the companion cell-sieve cell complex to move through the phloem. Likewise, synthetic derivatives or analogs, when applied to foliage to trigger plant defense, must also cross the plasma membrane at least once before reaching the sieve tubes. The capacity of molecules to diffuse through this membrane relies on their chemical attributes, such as molecule size, Log D (distribution coefficient), polar surface area, and the number of hydrogen bond donors. Through these characteristics, salicylic acid molecules can be conveyed to distant tissues, enhancing SAR (Bonnemain et al., 2013).

Following the detection of external SA, a shift occurs in the cellular redox system, a change associated with the SA-induced suppression of jasmonic acid (JA) responses (Lu et al., 2016; Maruri-López et al., 2019). This leads to the dissociation of the NPR1 complex into monomers by reducing disulfide links (Tada et al., 2008; Maruri-López et al., 2019). These monomeric forms are then transported to the nucleus, where they contact with basic leucine zipper (bZIP) TGA-type transcription factors. This interaction triggers modifications in the transcriptome, notably activating the transcription of defense-related genes like PR-1 (Fu & Dong, 2013; Birkenbihl et al., 2017; Maruri-López et al., 2019). From this stage, the subsequent steps in SAR activation follow the mechanisms mentioned earlier (Amrahov et al., 2022).

Auxine phytohormones–IAA/IBA and their effector systems during pathogen–plant interaction

Plant hormones play a crucial role in coordinating responses to both developmental signals and environmental stimuli, thus mitigating the fitness costs associated with defense. Among these hormones, those governing plant growth responses are particularly notable (Denancé et al., 2013; Ludwig-Müller, 2015). In the context of plant-pathogen interactions, the term "arms race" has been coined to depict the ongoing coevolutionary dynamics between defense and colonization strategies employed by both partners (Anderson et al., 2010; Ludwig-Müller, 2015). This concept can also be extended to encompass the interplay between growth promotion, such as nitrogen fixation (Gresshoff et al., 2015) and defense responses (Ludwig-Müller, 2015). The outcome of this "race" is contingent upon the hormonal balance: when favoring the plant, this plant will be the winner; however, if the pathogen can manipulate the hormonal system in its favor, it emerges as the victory of the pathogen (Ludwig-Müller, 2015). Hormones govern various pathways, including direct defense mechanisms, nutritional aspects, and the maintenance of cell wall integrity (López et al., 2008; Ludwig-Müller, 2015). These pathways collectively influence the plant's response during interactions with pathogens, delineating the outcome of this intricate interplay.

Auxins exert diverse influences on plant growth and development, just as extensively (Davies, 2004; Ludwig-Müller, 2015). At the cellular level, they intricately regulate various fundamental processes such as cell division, expansion, differentiation, and polarity. On a broader scale, they significantly contribute to organ development, including roots (both lateral and adventitious), shoots (like apical dominance), leaves, flower organs, and fruits. Additionally, auxins play pivotal roles in vascular patterning and orientation in response to environmental cues, such as gravitropism and phototropism. These varied examples underscore the indispensable roles of auxins across all major developmental processes in plants. Moreover, auxins are integral in modulating alterations in growth processes lin-

ked to both pathogens and symbiotic relationships. Pathogens can manipulate the auxin response, inducing specific disease symptoms during infection and disease progression. Conversely, beneficial microorganisms interfere with the host plant's auxin metabolism, triggering plant growth for their own advantage (Ludwig-Müller, 2014, 2015).

Auxin biosynthesis plays a dual role in plant health: it contributes to the symptoms of certain plant diseases while also being indispensable for normal plant development and environmental responses. Pathogens can exploit this biosynthetic system by either hijacking the host plant's system or producing auxin themselves. A prime illustration of the latter is demonstrated by the soil bacterium *Agrobacterium tumefaciens*, which induces tumor formation by genetically incorporating auxin and cytokinin biosynthesis genes into the plant tissue (Zupan & Zambryski, 1995; Ludwig-Müller, 2015). Other bacteria exhibit the ability to produce IAA via different ways, contributing to the modulation of plant growth and development (Spaepen et al., 2007; Ludwig-Müller, 2015).

The involvement of auxin in biotrophic interactions has received considerable attention in past research works, yet its role in the resistance response to necrotrophic pathogens remains less understood. Some reports indicate that hemibiotrophic or necrotrophic fungi have the capability to produce IAA, the primary auxin molecule (Ludwig-Müller, 2015). For instance, the rice blast fungus *Magnaporthe oryzae* generates IAA during its biotrophic phase, particularly in the region of infection hyphae (Tanaka et al., 2011; Ludwig-Müller, 2015). However, the precise purpose of this IAA production by the fungus – whether to manipulate the host plant or for the fungus's own advantage – is not yet definitively established. Studies using an auxin-inducible promoter have shown that the host plant responds transcriptionally to the IAA secreted by the fungus. Similarly, observations of auxin biosynthesis in two *Colletotrichum* species (Chung et al., 2003; Maor et al., 2004; Ludwig-Müller, 2015) further support the idea that fungal-produced auxin might serve to maintain the host plant in a state conducive to the fungus's nutrient acquisition. This strategy potentially keeps the plant in a growth stage where the fungus can derive necessary nutrients, especially during stages when the pathogen does not rely on the host plant's survival (necrotrophic stages). It seems that manipulating the plant by utilizing its own growth-promoting signaling molecules, like auxin, may be a widespread strategy employed by pathogens. Additionally, auxin is known to induce the expression of numerous genes, thereby potentially steering the cellular program favorably for the pathogenic fungus (Ludwig-Müller, 2015).

IBA serves as another example of auxin phytohormones. Historically, IBA has been characterized as a 'synthetic auxin', demonstrating similar effects such as root growth initiation, stem curling and leaf epinasty. Notably, IBA is widely employed as the active ingredient in plant propagation media (Frick & Strader, 2018).

The existence of a four-carbon side chain at position 3 of the indole ring, as opposed to IAA's two-carbon side chain, is one characteristic that sets IBA apart from IAA. The molecule's inability to adopt a conformation that would allow it to attach into the TIR1-Aux/IAA co-receptor pocket is probably due to its extended side chain (Uzunova et al., 2016; Frick & Strader, 2018). This idea is corroborated by surface plasmon resonance studies, which shows that IBA lacks detectable interaction activity (Uzunova et al., 2016). This is consistent with genetic research (Strader & Bartel, 2011; Frick & Strader, 2018) that suggests IBA's action is manifested via its transition to IAA. IBA is likely transformed into IAA through a process reminiscent of fatty acid β-oxidation. Various plant species, including *Arabidopsis* (Růžička et al., 2010), hazelnut, and elm (Kreiser et al., 2016) have been reported to convert IBA to IAA (Epstein & Ludwig-Müller, 1993; Frick & Strader, 2018). In *Arabidopsis*, this conversion is dependent on peroxisomes (Růžička et al., 2010). It has been found that a large number of mutants with reduced peroxisome production and peroxisomal proteins are resistant to IBA but still sensitive to the active auxin IAA (Hu et al., 2012; Frick & Strader, 2018).

The peroxisomal transporter1/comatose/ABCD1 (PXA1/CTS/ABCD1) transporter probably facilitates the transport of IBA into the peroxisome for its conversion into active auxin (Strader & Bartel, 2011; Frick & Strader, 2018). Some peroxisomal enzymes appear to be specialized to IBA β-oxidation (Frick & Strader, 2018), whereas others, such as the PED 1 3-ketoacyl-CoA thiolase, are thought to be involved in both fatty acid (Hayashi et al., 1998) and IBA β-oxidation (Zolman et al., 2000). Notably, certain enzymes, including acyl-CoA dehydrogenase/oxidase-like IBR3 (Zolman et al., 2007), enoyl-CoA hydratase IBR10 (Zolman et al., 2008), and enoyl-CoA hydratase enoyl-CoA hydratase 2 (ECH2) (Strader et al., 2011), are predicted to play specific roles in the transformation (Fig. 3) of the auxin predecessors IBA to the active form IAA (Frick & Strader, 2018).

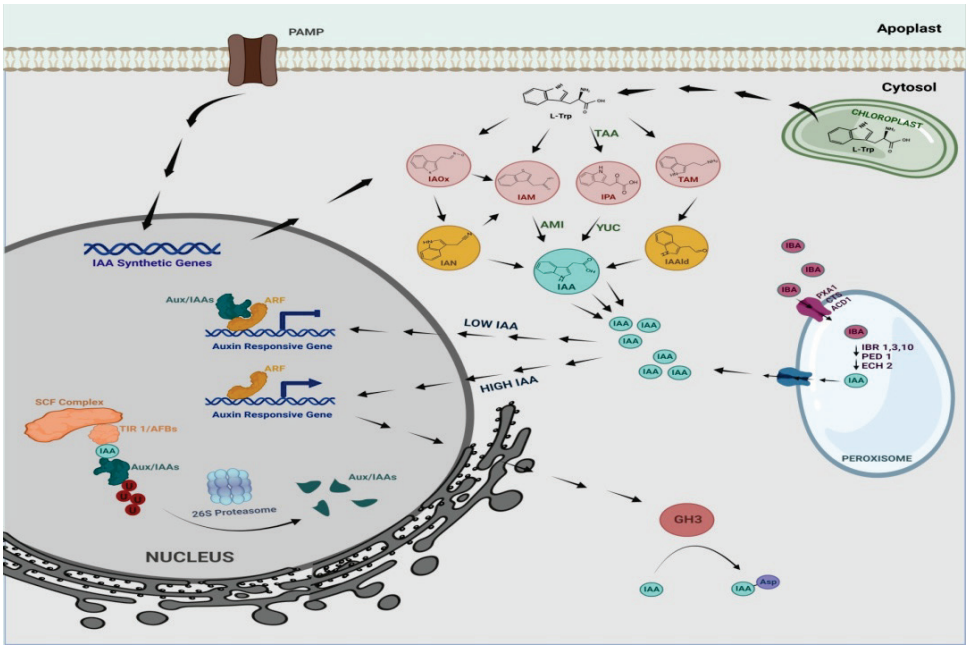

Fig. 3. The main pathways of IAA (Indole-3-acetic acid) metabolism and effector systems in plants: IBA – indole-3-butyric acid, PXA1/CTS/ABCD1 – peroxisomal transporter1/comatose/ABCD1, IBR1,3,10 – indole-3-butyric acid response 1,3,10, PED – peroxisome defective, ECH2 – enoyl-CoA hydratase 2, IAA – indole-3-acetic acid, L-Trp – L-tryptophan, IAOx – indole-3-acetaldoxime, IAM – indole-3-acetamide, IPyA – indole-3-pyruvic acid, TAM – tryptamine, IAN – indole-3-acetonitrile, IAAld – indole-3-acetaldehyde, TAA – tryptophan aminotransferase of *Arabidopsis*, YUC – *Yucca* flavin monooxygenase-like proteins, AMI – *Arabidopsis* amidase, PAMP – pathogen-associated molecular patterns, Aux/IAA – auxin/indole-3-acetic acid transcriptional corepressor, ARF – auxin response factor, SCF Complex – auxin-receptor S-phase kinase associated protein 1 (SKP1) – cullin-F-box complex, TIR/AFB – transport inhibitor resistant/auxin signaling F-box proteins, U – ubiquitin, GH3 – Gretchen Hagen 3

Apart from the conversion of IBA to IAA, IAA synthesis involves several steps originating from tryptophan, with only a few genes responsible for this process thoroughly understood (Woodward & Bartel, 2005; Reineke et al., 2008). Tryptophan-dependent auxin biosynthesis includes intermediates such as IAOx, IAM, IPyA, and TAM. While the pathways for IAOx and IAM to auxin production remain unclear, the IPyA pathway is indicated as the primary route for IAA synthesis in plants. This pathway involves Trp being deaminated to IPyA by tryptophan aminotransferase Of Arabidopsis 1 (TAA1) and TAA1-related proteins (TARs) (Stepanova et al., 2008; Tao et al., 2008; Yamada et al., 2009; Casanova-Sáez et al., 2021), followed by decarboxylation of IPyA to IAA catalyzed by Flavin-containing monooxygenases from the YUC family (Mashiguchi et al., 2011; Stepanova et al., 2011; Won et al., 2011; Casanova-Sáez et al., 2021). Another product of Trp, called IAOx, is an intermediary in an IAA biosynthesis pathway that is still being worked out. It involves the cytochrome P450 (CYP) monooxygenase family members CYP79B2 and CYP79B3 (Hull et al., 2000; Mikkelsen et al., 2000; Zhao et al., 2002; Casanova-Sáez et al., 2021). The conversion of indole-3-acetonitrile (IAN) to IAA with the use of plant nitrilases (NITs) is proposed as the route for IAOx-dependent auxin biosynthesis, although direct genetic and biochemical proof is lacking (Lehmann et al., 2017; Casanova-Sáez et al., 2021). An alternative route involves IAAld originating from tryptamine (TAM) (Zhao et al., 2001, 2002; Reineke et al., 2008). A similar pathway has been proposed in *Arabidopsis* based on the presence of indole-3-acetamide (IAM) and the IAM hydrolase activity expressed by the AMI1 gene (Pollmann et al., 2002, 2003; Reineke et al., 2008).

Auxin perception involves the action of F-box proteins TIR1, AFB1, 2 and 3. These receptors play a crucial role within a nuclear-localized SCF ubiquitin ligase complex. Mutations in genes responsible for auxin receptors or core SCF subunits lead to declined sensitivity to auxin. When cellular auxin amounts fall below a threshold level, AUX/IAA repressor proteins come into play, inhibiting auxin responses. These repressors hinder ARFs, essential for activating gene expression in response to auxin, by interacting with the conserved sequence ARE detected in the promoters of auxin-responsive genes. Under conditions of elevated auxin concentrations, the binding of auxin to its receptor enhances the ubiquitin-mediated disruption of AUX/IAA. This process liberates ARFs from transcriptional repression, ultimately inducing auxin responses (Woodward & Bartel, 2005; Paciorek & Friml, 2006; Vieten et al., 2007; Delker et al., 2008; Mockaitis & Estelle, 2008; Strader & Bartel, 2008; Chandler, 2009; Kazan & Manners, 2009; Sugawara et al., 2009).

During their interaction with host plants, many plant pathogens induce an increase in auxin levels. In the non-infection state or steady state, plant cells maintain a low, essential concentration of auxin. In this state, the AUX/IAA repressor protein contacts with ARFs, effectively repressing the transcription of auxin genes (Kazan & Manners, 2009).

Upon pathogen infection, the plant's auxin levels rise, facilitating the binding of auxin to the auxin-receptor SKP-Cullin-F-box (SCF) complex (Naseem et al., 2015). This binding leads to the removal and cleavage of the repressor AUX/IAA via the 26S proteasome and associated proteases. As a consequence, ARFs are released from repression and can bind to the promoters of auxin-responsive genes, activating their transcription (O'Donnell et al., 2003; Dharmasiri et al., 2005; Thilmony et al., 2006; Kazan & Lyons, 2014; Naseem et al., 2015).

The concentration of active IAA in specific tissues undergoes strict regulation, influenced by a variety of metabolic processes. These processes include the control of IAA synthesis, its transport to or from specific cells or tissues, inactivation and reactivation of IAA, and degradation through oxidative pathways (Normanly, 2010; González-Lamothe et al., 2012). The last two mechanisms involve the modification of IAA to glycosyl esters and amide-linked conjugates with various amino acids and peptides. In *Arabidopsis thaliana*, a significant portion of the conjugated forms is associated with amino acids or small peptides. IAA-amino acid conjugates are categorized as either storage or catabolism conjugates (Ludwig-Müller, 2011; González-Lamothe et al., 2012). Storage conjugates, such as IAA-Ala and IAA-Leu can be enzymatically hydrolyzed by plant enzymes to release free auxin. On the other hand, catabolism conjugates like IAA-Asp and IAA-Glu are not substrates for IAA-amino acid aminohydrolases. Given that free IAA is recognized as the biologically active form of auxin, only specific hydrolysable conjugates have demonstrated activity in auxin bioassays (Woodward & Bartel, 2005; Ludwig-Müller, 2011; González-Lamothe et al., 2012). While IAA-Asp has been suggested to play a role in abiotic stress and ripening in henbane (*Hyoscyamus niger*) and grape (*Vitis vinifera*) (Oetiker & Aeschbacher, 1997; Böttcher et al., 2010; González-Lamothe et al., 2012), a direct biological function for IAA-Asp and IAA-Glu has not been conclusively demonstrated. These conjugates are generally regarded as the starting point of auxin catabolism (Woodward & Bartel, 2005; Ludwig-Müller, 2011; González-Lamothe et al., 2012). The conjugation of auxin to amino acids is facilitated by the GH3 protein family, consisting of 19 members, with at least seven confirmed to catalyze the synthesis of IAA-amino acid combinations (Staswick et al., 2005; González-Lamothe et al., 2012).

Similar to the mechanisms governing IAA levels (Zazimalova et al., 2010; Korasick et al., 2013; Frick & Strader, 2018), the regulation of IBA levels involves processes such as the formation of IBA conjugates and IBA transport. IBA is found in both amide- and ester-linked forms (Woodward & Bartel, 2005; Bajguz & Piotrowska, 2009; Ludwig-Müller, 2011; Frick & Strader, 2018). While some members of the *Arabidopsis* GH3 amino acid synthetase family – GH3.4, GH3.5, GH3.6, and GH3.17 – show evidence of adenylation action with IBA and IAA (Staswick et al., 2005), no particular enzymes have been identified to date that are in charge of conjugating IBA to amino acids (Frick & Strader, 2018).

The precise mechanism through which auxin signaling contributes to disease remains unclear. One notable response triggered by the activation of auxin signaling is the synthesis of conjugated forms of auxin, facilitated by GH3 proteins (González-Lamothe et al., 2012). Previous studies have demonstrated the involvement of certain members of the GH3 gene family in the interaction between plants and pathogens, particularly in *Arabidopsis* and rice (*Oryza sativa*) (Jagadeeswaran et al., 2007; Nobuta et al., 2007; Park et al., 2007; Zhang et al., 2007; Ding et al., 2008; Domingo et al., 2009; Kazan & Manners, 2009; Fu et al., 2011; González-Lamothe et al., 2012). In each of these instances, the role of GH3 proteins has been elucidated by their capacity to modulate the concentration of free IAA or other defense-related hormones (González-Lamothe et al., 2012).

The detection of IAA-Asp, as opposed to free IAA, may have evolved as a strategic adaptation to enhance virulence. The observed induction of GH3.2 by the pathogen suggests that the pathogen itself is capable of initiating this process. It is plausible that in the evolutionary history, the pathogen originally utilized IAA to boost its virulence by manipulating other defense-related hormone pathways. Following infection, the plant may have responded by inducing GH3 proteins to decrease free IAA levels. Subsequently, the pathogen could have evolved to recognize and exploit IAA-Asp for its own advantage (González-Lamothe et al., 2012).

The induction of GH3.2 by auxin, as observed in previous studies (Takase et al., 2004; González-Lamothe et al., 2012), suggests that auxin signaling may activate the conjugation process through the transcriptional activation of GH3.2. Consequently, the repression of auxin signaling by SA could lead to a decrease in IAA conjugation and, consequently, a reduction in pathogen virulence (Wang et al., 2007; González-Lamothe et al., 2012). Moreover, there is a proposed link between auxin signaling and SAR, with auxin playing an essential role in establishing SAR (Truman et al., 2010). Specific compounds involved in auxin signaling might be crucial in regulating the interaction between IAA and SA, the enhancing of IAA conjugation, or possibly both aspects (González-Lamothe et al., 2012).

Although auxin's role as a regulator of plant defense has long been acknowledged, the precise molecular mechanisms involved have only recently come under investigation. Similar to the signaling pathways associated with the defense chemicals SA and JA, auxin signaling has also effects on resistance to different pathogen species (Kazan & Manners, 2009; Ludwig-Müller, 2015). Recent evidence suggests an antagonistic relationship between auxin and the SA pathway during plant defense responses, while similarities exist between auxin and jasmonate pathways in terms of plant defense reactions (Kazan & Manners, 2009; Ludwig-Müller, 2015). Additionally, auxin may indirectly influence disease outcomes by influencing plant development (Gil et al., 2001; Ludwig-Müller, 2015).

The opposing interactions between SA and auxin could be evolutionarily driven by the need for plants to allocate limited resources to defense

processes at the expense of growth when facing pathogen attacks (Kazan & Manners, 2009). Plant growth relies on energy, primarily derived from photosynthesis and respiration. The SA-mediated activation of PR (pathogenesis-related) proteins is contingent upon intact photoreceptors, establishing a link between light and defense (Karpinski et al., 2003; Ludwig-Müller, 2015). A noteworthy connection between SA and photosynthesis involves the protein isochorismate synthase, crucial for SA synthesis and also contributing to the production of phylloquinone, which integrates into photosystem I (Szechynska-Hebda & Karpinski, 2013). Excessive excitation energy exhibits similar effects on the expression of nuclear genes associated with systemic-acquired acclimation and systemic-acquired resistance, both intricately connected to programmed cell death (Szechynska-Hebda & Karpinski, 2013; Ludwig-Müller, 2015). However, recent findings demonstrate that auxin and SA exhibit a systemic co-increase during *Arabidopsis thaliana* infection with Cucumber mosaic virus, challenging the expectation of a consistent antagonistic relationship between auxin and SA (Likic´ et al., 2014). Hence, not all instances necessarily involve an antagonism between auxin and SA (Ludwig-Müller, 2015).

SA exerts its inhibitory influence on auxin signaling through various mechanisms. One such mechanism involves the transcriptional repression of genes responsible for encoding auxin receptors, including TIR1 and related F-box proteins (Wang et al., 2007; Kazan & Manners, 2009). By suppressing the expression of these auxin receptors, SA facilitates the auxin-dependent degradation of the AUX/IAA repressors. Consequently, this process leads to the stabilization of AUX/IAA and the stabilizing of their repressive effects on auxin response genes. The overall outcome is the inhibition of disease development (Kazan & Manners, 2009).

As previously discussed, auxin stands as one of the fundamental signals governing plant cellular developmental programs, and any disruption in auxin signaling can have adverse effects on normal plant development (Santner & Estelle, 2009; Grones & Friml, 2015; Naseem et al., 2015; Schaller et al., 2015). The inhibited plant growth phenotypes observed with constitutively active SA defenses (Clarke et al., 2000; Zhang et al., 2003; Huot et al., 2014) may be, in part, ascribed to the SA-associated inactivation of auxin signaling (Naseem et al., 2015).

Besides the already mentioned effects of internally synthesized auxin molecules, externally applied phytohormones, including IBA have been detected as implicating similar effects inside plant cells (Amrahov et al., 2023).

IBA/IAA transport

The active transport of auxin in cambium is primarily basipetal, while within plant tissues, it adopts a polar pattern (Uggla et al., 1998; Yu et al., 2017). This polar auxin transport (PAT) relies on the proton motive force across the plasma membrane and employs specialized membrane carriers (Galweiler et al., 1998; Müller et al., 1998; Schrader et al., 2003). Despite the close chemical resemblance between IAA and IBA, investigations into numerous IAA carriers have revealed their incapacity to transport IBA. Similar to IAA, IBA entry exhibits saturation kinetics in *Arabidopsis* (Ludwig-Muller et al., 1995; Rashotte et al., 2003; Strader & Bartel, 2011), implying the involvement of protein carrier molecules instead of passive diffusion. The entry of IAA into cells is facilitated by the auxin resistant 1 (AUX1) protein family (Vieten et al., 2007). Comprising AUX1, like AUX1 (LAX1), LAX2 and 3, these members share structural similarities with amino acid permeases, featuring 11 transmembrane domains (Swarup et al., 2004). Although AUX1 role as an IBA uptake carrier seems improbable, other members of the AUX1 family stand as plausible candidates for this function (Strader & Bartel, 2011).

Unlike the inflow, IBA export seems to use different carriers than those that support IAA efflux. PIN-formed (PIN) proteins and ABCB class type proteins of ATP-Binding-Cassette (ABC) transporters are the two types of proteins that facilitate IAA efflux. The endoplasmic reticulum membrane or the plasma membrane is where the eight PIN family members are located (Grunewald & Friml, 2010; Strader & Bartel, 2011). According to Grunwald & Friml (2010), plasma-membrane PIN proteins frequently display distinct polar localization to particular cellular faces, suggesting their function in controlling the direction of IAA flow within cells and tissues. While they are also found in the plasma membrane, members of the ABCB/multidrug resistance/P-glycoprotein family, such as ABCB1, ABCB4, and ABCB19, exhibit a more symmetrical dispersion than PIN proteins. As part of long-distance IAA movement and perhaps transporting IAA to PIN proteins, they could promote non-polar IAA efflux (Bandyopadhyay et al., 2007; Blakeslee et al., 2007; Bailly et al., 2008; Mravec et al., 2008; Strader & Bartel, 2011). Nevertheless, none of the ABCB or PIN family mediates IBA efflux. Rather, at least two members of the pleiotropic drug resistance (PDR) molecules of the ABCG family of ABC transporters facilitate the removal of IBA from root cells (Strader & Bartel, 2011).

Gibberellic acid

The gibberellins include a diverse group of diterpenoid carboxylic acids with the most biologically active forms being GA1, GA3, and GA4 (MacMillan & Takahashi, 1968; Hedden & Thomas, 2012). As a plant hormone, GAs have crucial roles in growth and development. They also involve in major developmental transitions, e.g. the shift between seed dormancy and germination, juvenile and adult growth phases, and vegetative and reproductive development. The execution of these functions is dependent on developmental and environmental cues, which may regulate various aspects of the GA pathway, including biosynthesis, turnover, perception, or signal transduction, often acting at multiple points in the pathway (Hedden & Thomas, 2012).

In higher plants, GA biosynthesis is initiated in plastids, continues in the endoplasmic reticulum, and finally, the biologically active GAs are synthesized in the cytosol. The synthesis starts with the conversion of geranylgeranyl diphosphate (GGPP) to ent-copalyl diphosphate by ent-copalyl diphosphate synthase (CPS), then to ent-kaurene by ent-kaurene synthase (KS) in proplastids (Olszewski et al., 2002; Mizutani & Ohta, 2010). In the past, it was believed that ent-kaurene competes for a shared pool of GGPP with other GGPP-derived metabolites (Fray et al., 1995). Later, studies showed that there is a separate GGPP pool for ent-kaurene production, facilitated by a GGPS (GGPP synthase) that uses GPP as a substrate (Fig. 4), which differs from other GGPS isozymes involved in pigment synthesis (van Schie et al., 2007; Hedden & Thomas, 2012).

GA12 is considered the main precursor of all active GA forms and its formation from ent-kaurene involves the action of membrane–associated P450s and soluble 2-oxoglutarate-dependent dioxygenases (ODDs) in the endoplasmic reticulum. The production of GA12 involves two monooxygenases, KO and KAO (Morrone et al., 2010; Hedden & Thomas, 2012). GA12 is converted into GA53 by GA13ox (GA 13-oxidase). Next, GA12 and 53 are oxidized on C-20 by GA20ox (GA 20-oxidase), an ODD, converting these substrates into GA9 and GA20 in the cytosol, respectively (Hedden & Thomas, 2012).

The end stage of the formation of biologically active GAs is the production of GA4 and GA1 from GA9 and GA20, respectively, which involves the action of GA3ox (GA 3-oxidase). In dicotyledonous species, this reaction yields a single product (Martin et al., 1997; Lester et al., 1997; Williams et al., 1998), whereas in several monocotyledons, GA1 and GA3 are formed from GA20 via the intermediate GA5 (Itoh et al., 2001; Appleford et al., 2006).

Gibberellin insensitive dwarf 1 (GID1), the GA receptor, DELLA growth inhibitors (DELLAs), and the F-box proteins SLEEPY1 (SLY1) and SNEEZY (SNZ) in *Arabidopsis* and gibberellin insensitive dwarf 2 (GID2) in rice are the primary components of the gibberellic acid signaling pathway (Davière & Achard, 2013). GID1 is a soluble nuclear receptor that detects GA signals. In contrast to *Arabidopsis*, which has three functioning GID1 orthologs – GID1a, 1b, and 1c – rice only has one copy of the GID1 gene (Ueguchi-Tanaka et al., 2005; Griffiths et al., 2006; Iuchi et al., 2007; Willige et al., 2007; Hauvermale et al., 2012). The GID1 receptor lacks hydrolase function but resembles human hormone-sensitive lipases (Ueguchi-Tanaka et al., 2005; Ueguchi–Tanaka & Matsuoka, 2010; Davière & Achard, 2013). GID1 has a flexible N-terminal extension and a GA-binding pocket (Murase et al., 2008; Shimada et al., 2008; Davière & Achard, 2013). The closure of the GA pocket is caused by a conformational shift in the N-terminal tail following the attachment of bioactive GA to GID1 (Murase et al., 2008; Shimada et al., 2008). The outermost layer of the lid bonds with the DELLA only after selecting the GA

pocket, forming the GA-GID1-DELLA complex (Griffiths et al., 2006; Ueguchi-Tanaka et al., 2007; Willige et al., 2007; Davière & Achard, 2013). The ubiquitin-proteasome pathway facilitates the degradation of DELLAs, which is facilitated by the formation of the GA-GID1-DELLA complex. According to Hirano et al. (2010), this complex formation modifies the structure of DELLA's GRAS domain, improving the interaction between DELLA's VHIID and LHRII motifs and the F-box protein SLEEPY1 (SLY1) in rice and *Arabidopsis*/GID2. The F-box proteins are components of the SCF (SKP1, cullin, F-box) E3 ubiquitin ligase complexes (Fig. 5), which help polyubiquitin chains connect to target proteins so that the 26S proteasome can degrade them (Lechner et al., 2006).

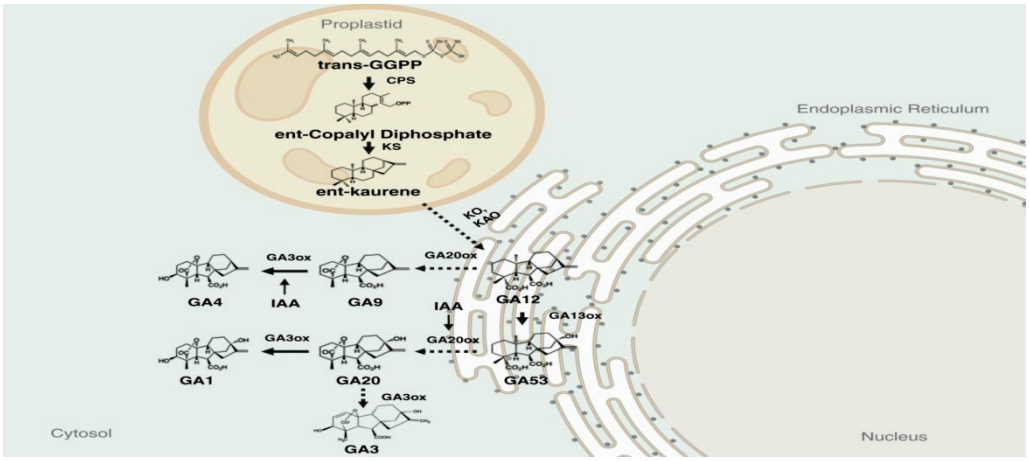

Fig. 4. Synthesis of active foms of GA: trans-GGPP – geranylgeranyl diphosphate, CPS – ent-copalyl diphosphate synthase, KS – ent-kaurene synthase, KO – entkaurene oxidase, KAO – ent-kaurenoic acid oxidase, GA20ox – GA 20-oxidase, GA13ox – GA 13-oxidase, GA3ox – Ga 3-oxidase

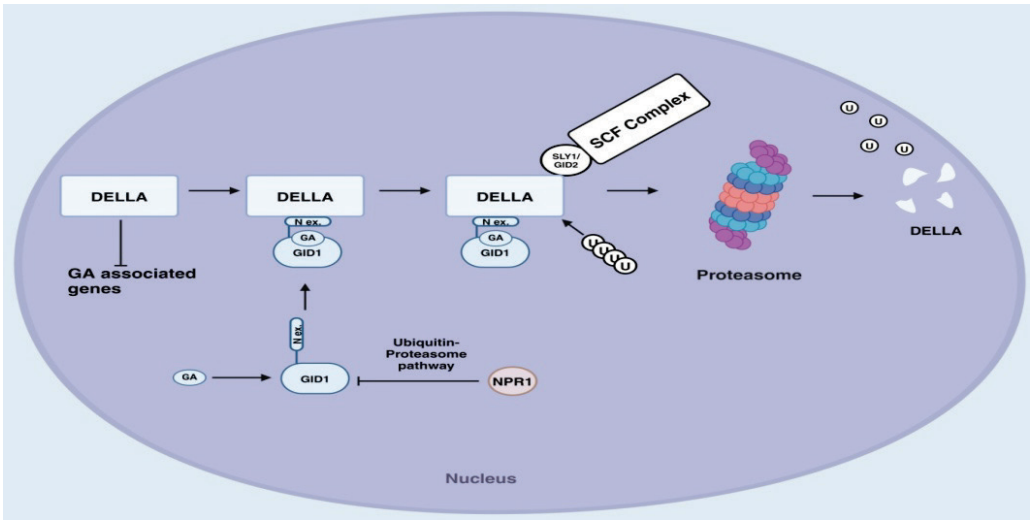

Fig. 5. GA–mediated degradation of DELLA proteins: GID1 – gibberellin insensitive dwarf 1, SLY1 – SLEEPY1, GID2 – gibberellin insensitive dwarf 2, SCF – SKP1 cullin F-box, NPR1 – nonexpressor of pathogenesis related genes 1, DELLA – Asp-Glu-Leu-Leu-Ala

As members of the plant-specific GRAS family of putative transcription regulators, DELLAs are nuclear-localized inhibitors of GA responses (Peng et al., 1997; Silverstone et al., 1998; Ogawa et al., 2000; Ikeda et al., 2001; Chandler et al., 2002). DELLAs inhibit nearly every known GA-dependent function as well as seed germination and growth (Achard & Genschik, 2009).

Like other GRAS proteins, DELLAs have a conserved C-terminal GRAS domain that is important for controlling transcription. This domain is made up of three conserved structures (VHIID, PFYRE, and SAW) and two leucine heptad repeats (LHRI and LHRII) (Bolle, 2004). DELLAs are unique members of the GRAS family because of their unique N-terminal sequence, which contains two conserved domains: the TVHYNP domain and the DELLA domain, which gives them their name (Davière & Achard, 2013). Five DELLAs (GA–insensitive, GAI, repressor of GA1-3, RGA; RGA-LIKE1, RGL1; RGL2 and RGL3) are encoded in the *Arabidopsis* genome and work together to repress GA responses in different ways (Peng et al., 1997; Ikeda et al., 2001; Lee et al., 2002; Wen & Chang, 2002; Tyler et al., 2004; Davière & Achard, 2013). A pivotal aspect of DELLAs lies in their capacity to interact with various classes of regulatory proteins. DELLAs block transcription factors' DNA-binding capacities (like PIFs do) or the activity of regulatory molecules through these interactions (Feng et al., 2008).

Gibberellins are found to interact with other phytohormones, including ABA, SA and auxins. Gibberellic acid and abscisic acid (ABA) have antagonistic roles in the control of various developmental processes, and their interactions are important. GA is recognized for its ability to stimulate germination, development, and flowering, while ABA is known to impede these essential activities. The transition from embryogenesis to seed germination is regulated by the relative presence and balance of these hormones (Razem et al., 2006; Weiss & Ori, 2007).

The growing embryo releases GAs to aleurone cells during cereal seed germination, which causes the transcription of genes encoding hydrolytic enzymes, such as a-amylase. Following their secretion, these enzymes are found in the endosperm, where they catalyze the breakdown of proteins and carbohydrates, supplying vital nutrients for the growing embryo. Conversely, ABA exerts a suppressive effect on a-amylase expression, introducing a regulatory dynamic to the process (Weiss & Ori, 2007).

SA negatively regulates GA signaling by the degradation of GID1 receptors. The ubiquitin-proteosome pathway degrades the GID1 receptor in the presence of SA by interacting with it through NPR1, a critical regu-

lator in SA signaling (Cao et al., 1997; Ryals et al., 1997; Shah et al., 1997; Yu et al., 2022). NPR1 and CUL3 (Cullin 3) combine to generate a ubiquitin E3 ligase that facilitates GID1 degradation and polyubiquitination (Yu et al., 2022).

The cross-talk between auxins and GAs occurs at many levels, including signaling, metabolism or gene expression, and in many cases in a tissue-specific manner. However, a clear relationship between both hormones cannot be stated, as they have a positive effect on some aspects of development, and they seem to present a synergistic character (Castro–Camba et al., 2022). IAA upregulates GA synthesis by activating GA3ox and GA20ox and inhibiting GA2ox (Frigerio et al., 2006; Yin et al., 2007; O'Neill et al., 2010). On the other hand, GAs modulate auxin-related genes, although the outcomes of these responses depend on the specific set of GA-induced auxin response factors (ARFs) (Castro-Camba et al., 2022). In addition, GAs modify the expressions of several auxin transporters (Salanenka et al., 2018). It has been found that GAs are required for the proper function of PIN-formed (PIN) protein auxin transporters (PIN1, PIN2, PIN3) (Willige et al., 2011; Castro-Camba et al., 2022).

Studies have shown that after infection with a pathogen the endogenous GA content significantly decreased (Zhu et al., 2005; Ding et al., 2013; De Bruyne et al., 2014). Furthermore, exogenous GA application to infected plants restored pathogen-induced GA decline partially, and exogenous GA lowered resistance to hemibiotrophic fungi *Magnaporthe oryzae* in rice (Ding et al., 2013; Qin et al., 2013; De Bruyne et al., 2014).

Some of MAPKKKs and MAPK molecules have been identified to influence GA content (Xie et al., 2023). Overexpression of MAPKKK5 of *O. sativa*, a MAP3K, belonging to the Raf subfamily, involves in regulation of plant cell size by increasing concentrations of GA. The pathway through which MAP3K5 upregulates GA content has not been determined yet (Liu et al., 2019; Xie et al., 2023). GhMPK11, a group B MAPK gene isolated from *Gossypium hirsutum*, increases GA3 concentrations by upregulating GA-related genes and also causes susceptibility to certain pathogens through the GA3 signaling pathway via down-regulation of ROS detoxification enzymes (Wang & Guo, 2016). After infection with a pathogen due to elevated levels of ROS, resistance to a pathogen decreases (Yoshioka et al., 2009). The concentrations of ROS detoxifying enzymes can be regulated by the SCFSLY 1/GID2 complex through the GA signaling pathway (Grant & Jones, 2009). Thus, increased GA content may decrease resistance to pathogens by decreased ROS detoxfying enzymes and by contributing to ROS accumulation.

Conclusion

The growing body of knowledge about plant-pathogen interactions reveals increasingly complex underlying processes. Upon recognition of pathogen-associated molecular patterns (PAMPs) by pattern recognition receptors (PRRs), the downstream MAPK signaling pathway is activated, leading to various immune responses. One significant aspect of these mechanisms is the enhanced synthesis of phytohormones, such as salicylic acid (SA). Research using the model plant *Arabidopsis thaliana* has shown that the activation of TGA1/4 transcription factors, as part of the MAPK-related pathways, leads to the induction of isochorismate synthase (ICS), the primary enzyme for SA biosynthesis. This, in turn, triggers other critical immune processes, including systemic acquired resistance (SAR). Simultaneously, pathogen recognition stimulates genes involved in indole-3-acetic acid (IAA) synthesis, leading to increased IAA levels. The accumulation of IAA activates auxin-responsive genes, which play a crucial role in subsequent immune responses. Additionally, gibberellins (GAs) can modulate the effects of these phytohormones. While elevated SA levels may negatively regulate GA signaling by degrading GID1 receptors, auxin and GA often act synergistically. The interactions between pathogens and plant cells, and their effects on various phytohormones, present numerous uncertainties regarding the exact mechanisms and propagation of signaling molecules to systemic tissues for activating secondary immune responses. Understanding these signals and transduction pathways is highly relevant for potential applications in plant engineering programs aimed at enhancing resistance to phytopathogens.

This work was supported by the Azerbaijan Science Foundation – Grant No. AEF-MCG-2022-1(42)-12/11/4-M-11.

References

Achard, P., & Genschik, P. (2009). Releasing the brakes of plant growth: how GAs shutdown DELLA proteins. Journal of Experimental Botany, 60(4), 1085–1092.

Ahmadov, I. S., Ramazanov, M. A., Gasimov, E. K., Rzayev, F. H., & Veliyeva, S. B. (2020). The migration study of nanoparticles from soil to the leaves of plants. Biointerface Research in Applied Chemistry, 10(5), 6101–6111.

Akamatsu, A., Wong, H. L., Fujiwara, M., Okuda, J., Nishide, K., Uno, K., Imai, K., Umemura, K., Kawasaki, T., Kawano, Y., & Shimamoto, K. (2013). An OsCEBiP/OsCERK1-OsRacGEF1-OsRac1 module is an essential early component of chitin-induced rice immunity. Cell Host and Microbe, 13(4), 465–476.

Aliyeva, D. R., Gurbanova, U. A., Rzayev, F. H., Gasimov, E. K., & Huseynova, I. M. (2023). Biochemical and ultrastructural changes in wheat plants during drought stress. Biochemistry, 88(11), 1944–1955.

Alizade, S., Mammadova, R., & Sirajli, N. (2023). Evaluation of morphometric traits of upland cotton genotypes under different concentration of NaCl. Advances in Biology and Earth Sciences, 8(3), 301–307.

Amrahov, N. R., Allahverdiyev, V. Y., Agharzayeva, Y. I., Mammadova, R. B., Omarova, S. N., Khudayev, F. A., Nasibova, A. N., Shoaib, M., Khalilov R. İ., & Mammadov, Z. M. (2023). Effect of *Verticillium* wilt on the antioxidant system and formation of iron nanoparticles in cotton genotypes. Journal of Animal and Plant Sciences, 33(6), 1322–1332.

Amrahov, N. R., Mammadova, R. B., Allahverdiyeva, S. N., Aliyev, E. I., Alizada, S. R., Aghazada, G. A., Ojagverdiyeva, S. Y., & Mammadov, Z. M. (2023). Effect of indole-3-butyric acid on the antioxidant enzymes, NO and chlorophyll content of Agdash-3 and AP-317 genotypes of upland cotton (*Gossypium hirsutum* L.). Advances in Biology and Earth Sciences, 8(2), 147–156.

Amrahov, N., Bayramova, S., Mammadova, R., Ojagverdiyeva, S., Aghazada, G., Alizada, S., & Mammadov, Z. (2022). The effect of salicylic acid on the content of photosynthetic pigments and the activity of antioxidant enzymes in cotton seedlings. Advances in Biology and Earth Sciences, 7(3), 169–177.

Anderson, J. P., Gleason, C. A., Foley, R. C., Thrall, P. H., Burdon, J. B., & Singh, K. B. (2010). Plants versus pathogens: An evolutionary arms race. Functional Plant Biology, 37(6), 499–512.

Appleford, N. E., Evans, D. J., Lenton, J. R., Gaskin, P., Croker, S. J., Devos, K. M., Phillips, A. L., & Hedden, P. (2006). Function and transcript analysis of gibberellin-biosynthetic enzymes in wheat. Planta, 223(3), 568–582.

Attaran, E., & He, S. Y. (2012). The long-sought-after salicylic acid receptors. Molecular Plant, 5(5), 971–973.

Bailly, A., Sovero, V., Vincenzetti, V., Santelia, D., Bartnik, D., Koenig, B. W., Mancuso, S., Martinoia, E., & Geisler, M. (2008). Modulation of P-glycoproteins by auxin transport inhibitors is mediated by interaction with immunophilins. Journal of Biological Chemistry, 283(31), 21817–21826.

Bajguz, A., & Piotrowska, A. (2009). Conjugates of auxin and cytokinin. Phytochemistry, 70(8), 957–969.

Bandyopadhyay, A., Blakeslee, J. J., Lee, O. R., Mravec, J., Sauer, M., Titapiwatanakun, B., Makam, S. N., Bouchard, R., Geisler, M., Martinoia, E., Friml, J., Peer, W. A., & Murphy, A. S. (2007). Interactions of PIN and PGP auxin transport mechanisms. Biochemical Society Transactions, 35(1), 137–141.

Baran, M. F., Keskin, C., Baran, A., Eftekhari, A., Omarova, S., Khalilov, R., Adican, M. T., Rosić, G., Selakovic, D., Yıldıztekin, M., Kurt, K., Ava, C. A., & Atalar, M. N. (2023). The investigation of the chemical composition and applicability of gold nanoparticles synthesized with *Amygdalus communis* (almond) leaf aqueous extract as antimicrobial and anticancer agents. Molecules, 28(6), 2428.

Bernsdorff, F., Döring, A. C., Gruner, K., Schuck, S., Bräutigam, A., & Zeier, J. (2016). Pipecolic acid orchestrates plant systemic acquired resistance and defense priming via salicylic acid-dependent and-independent pathways. The Plant Cell, 28(1), 102–129.

Birkenbihl, R. P., Liu, S., & Somssich, I. E. (2017). Transcriptional events defining plant immune responses. Current Opinion in Plant Biology, 38, 1–9.

Blakeslee, J. J., Bandyopadhyay, A., Lee, O. R., Mravec, J., Titapiwatanakun, B., Sauer, M., Makam S. N., Cheng, Y., Bouchard, R., Adamec, J., Geisler, M., Nagashima, A., Sakai, T., Martinoia, E., Friml, J., Peer, W. A., & Murphy, A. S. (2007). Interactions among PIN-FORMED and P-glycoprotein auxin transporters in *Arabidopsis*. The Plant Cell, 19(1), 131–147.

Bolle, C. (2004). The role of GRAS proteins in plant signal transduction and development. Planta, 218(5), 683–692.

Bonnemain, J. L., Chollet, J. F., & Rocher, F. (2013). Transport of salicylic acid and related compounds. In: Hayat, S., Ahmad, A., & Alyemeni, M. (Eds.). Salicylic acid. Springer, Dordrecht. Pp. 43–59.

Böttcher, C., Keyzers, R. A., Boss, P. K., & Davies, C. (2010). Sequestration of auxin by the indole-3-acetic acid-amido synthetase GH3-1 in grape berry (*Vitis vinifera* L.) and the proposed role of auxin conjugation during ripening. Journal of Experimental Botany, 61(13), 3615–3625.

Buist, G., Steen, A., Kok, J., & Kuipers, O. P. (2008). LysM, a widely distributed protein motif for binding to (peptido) glycans. Molecular Microbiology, 68(4), 838–847.
Cao, H., Glazebrook, J., Clarke, J. D., Volko, S., & Dong, X. (1997). The *Arabidopsis* NPR1 gene that controls systemic acquired resistance encodes a novel protein containing ankyrin repeats. Cell, 88(1), 57–63.
Cao, Y., Liang, Y., Tanaka, K., Nguyen, C. T., Jedrzejczak, R. P., Joachimiak, A., & Stacey, G. (2014). The kinase LYK5 is a major chitin receptor in *Arabidopsis* and forms a chitin-induced complex with related kinase CERK1. eLife, 3, e03766.
Casanova-Sáez, R., Mateo-Bonmatí, E., & Ljung, K. (2021). Auxin metabolism in plants. Cold Spring Harbor Perspectives in Biology, 13(3), a039867.
Castro-Camba, R., Sánchez, C., Vidal, N., & Vielba, J. (2022). Interactions of gibberellins with phytohormones and their role in stress responses. Horticulturae, 8(3), 241.
Chanda, B., Xia, Y. E., Mandal, M. K., Yu, K., Sekine, K. T., Gao, Q. M., Selote, D., Hu, Y., Stromberg, A., Navarre, D., Kachroo, A., & Kachroo, P. (2011). Glycerol-3-phosphate is a critical mobile inducer of systemic immunity in plants. Nature Genetics, 43(5), 421–427.
Chandler, J. W. (2009). Local auxin production: a small contribution to a big field. Bioessays, 31(1), 60–70.
Chandler, P. M., Marion-Poll, A., Ellis, M., & Gubler, F. (2002). Mutants at the Slender1 locus of barley cv Himalaya. Molecular and Physiological Characterization. Plant Physiology, 129(1), 181–190.
Chen, Y. C., Holmes, E. C., Rajniak, J., Kim, J. G., Tang, S., Fischer, C. R., Mudgett, M. B., & Sattely, E. S. (2018). N-hydroxy-pipecolic acid is a mobile metabolite that induces systemic disease resistance in *Arabidopsis*. Proceedings of the National Academy of Sciences, 115(21), e4920–e4929.
Choi, W. G., Toyota, M., Kim, S. H., Hilleary, R., & Gilroy, S. (2014). Salt stress-induced Ca^{2+} waves are associated with rapid, long-distance root-to-shoot signaling in plants. Proceedings of the National Academy of Sciences, 111(17), 6497–6502.
Christmann, A., Grill, E., & Huang, J. (2013). Hydraulic signals in long-distance signaling. Current Opinion in Plant Biology, 16(3), 293–300.
Christmann, A., Weiler, E. W., Steudle, E., & Grill, E. (2007). A hydraulic signal in root-to-shoot signalling of water shortage. The Plant Journal, 52(1), 167–174.
Chung, K. R., Shilts, T., Ertürk, Ü., Timmer, L. W., & Ueng, P. P. (2003). Indole derivatives produced by the fungus *Colletotrichum acutatum* causing lime anthracnose and postbloom fruit drop of citrus. FEMS Microbiology Letters, 226(1), 23–30.
Clarke, J. D., Volko, S. M., Ledford, H., Ausubel, F. M., & Dong, X. (2000). Roles of salicylic acid, jasmonic acid, and ethylene in cpr-induced resistance in *Arabidopsis*. The Plant Cell, 12(11), 2175–2190.
Dangol, S., Chen, Y., Hwang, B. K., & Jwa, N. S. (2019). Iron-and reactive oxygen species-dependent ferroptotic cell death in rice-*Magnaporthe oryzae* interactions. The Plant Cell, 31(1), 189–209.
Davière, J. M., & Achard, P. (2013). Gibberellin signaling in plants. Development, 140(6), 1147–1151.
Davies, P. J. (Ed.). (2004). Plant hormones: Biosynthesis, signal transduction, action! 3rd edition. Springer, Dordrecht.
De Bruyne, L., Höfte, M., & De Vleesschauwer, D. (2014). Connecting growth and defense: The emerging roles of brassinosteroids and gibberellins in plant innate immunity. Molecular Plant, 7(6), 943–959.
De Jonge, R., Bolton, M. D., & Thomma, B. P. (2011). How filamentous pathogens co-opt plants: The ins and outs of fungal effectors. Current Opinion in Plant Biology, 14(4), 400–406.
Delker, C., Raschke, A., & Quint, M. (2008). Auxin dynamics: The dazzling complexity of a small molecule's message. Planta, 227, 929–941.
Denancé, N., Sánchez-Vallet, A., Goffner, D., & Molina, A. (2013). Disease resistance or growth: The role of plant hormones in balancing immune responses and fitness costs. Frontiers in Plant Science, 4, 155.
Després, C., DeLong, C., Glaze, S., Liu, E., & Fobert, P. R. (2000). The *Arabidopsis* NPR1/NIM1 protein enhances the DNA binding activity of a subgroup of the TGA family of bZIP transcription factors. The Plant Cell, 12(2), 279–290.
Dharmasiri, N., Dharmasiri, S., & Estelle, M. (2005). The F-box protein TIR1 is an auxin receptor. Nature, 435(7041), 441–445.
Ding, P., Rekhter, D., Ding, Y., Feussner, K., Busta, L., Haroth, S., Xu, S., Li, X., Jetter, R., Feussner, I., & Zhang, Y. (2016). Characterization of a pipecolic acid biosynthesis pathway required for systemic acquired resistance. The Plant Cell, 28(10), 2603–2615.
Ding, X., Cao, Y., Huang, L., Zhao, J., Xu, C., Li, X., & Wang, S. (2008). Activation of the indole-3-acetic acid - amido synthetase GH3-8 suppresses expansin expression and promotes salicylate-and jasmonate-independent basal immunity in rice. The Plant Cell, 20(1), 228–240.
Ding, Y., Sun, T., Ao, K., Peng, Y., Zhang, Y., Li, X., & Zhang, Y. (2018). Opposite roles of salicylic acid receptors NPR1 and NPR3/NPR4 in transcriptional regulation of plant immunity. Cell, 173(6), 1454–1467.
Ding, Y., Wei, W., Wu, W., Davis, R. E., Jiang, Y., Lee, I.-M., Hammond, R. W., Shen, L., Sheng, J.-P., & Zhao, Y. (2013). Role of gibberellic acid in tomato defence against potato purple top phytoplasma infection. Annals of Applied Biology, 162(2), 191–199.
Domingo, C., Andrés, F., Tharreau, D., Iglesias, D. J., & Talón, M. (2009). Constitutive expression of OsGH3.1 reduces auxin content and enhances defense response and resistance to a fungal pathogen in rice. Molecular Plant-Microbe Interactions, 22(2), 201–210.
Epstein, E., & Ludwig-Müller, J. (1993). Indole-3-butyric acid in plants: Occurrence, synthesis, metabolism and transport. Physiologia Plantarum, 88(2), 382–389.
Farmer, E. E., Gao, Y. Q., Lenzoni, G., Wolfender, J. L., & Wu, Q. (2020). Wound- and mechanostimulated electrical signals control hormone responses. New Phytologist, 227(4), 1037–1050.
Feng, S., Martinez, C., Gusmaroli, G., Wang, Y., Zhou, J., Wang, F., Chen, L., Yu, L., Iglesias-Pedraz, J. M., Kircher, S., Schäfer, E., Fu, X., Fan, L. M., & Deng, X. W. (2008). Coordinated regulation of *Arabidopsis thaliana* development by light and gibberellins. Nature, 451(7177), 475–479.
Fernandez, J., Marroquin-Guzman, M., Nandakumar, R., Shijo, S., Cornwell, K. M., Li, G., & Wilson, R. A. (2014). Plant defence suppression is mediated by a fungal sirtuin during rice infection by *Magnaporthe oryzae*. Molecular Microbiology, 94(1), 70–88.
Fliegmann, J., Uhlenbroich, S., Shinya, T., Martinez, Y., Lefebvre, B., Shibuya, N., & Bono, J. J. (2011). Biochemical and phylogenetic analysis of CEBiP-like LysM domain-containing extracellular proteins in higher plants. Plant Physiology and Biochemistry, 49(7), 709–720.
Fray, R. G., Wallace, A., Fraser, P. D., Valero, D., Hedden, P., Bramley, P. M., & Grierson, D. (1995). Constitutive expression of a fruit phytoene synthase gene in transgenic tomatoes causes dwarfism by redirecting metabolites from the gibberellin pathway. The Plant Journal, 8(5), 693–701.
Frick, E. M., & Strader, L. C. (2018). Roles for IBA-derived auxin in plant development. Journal of Experimental Botany, 69(2), 169–177.
Frigerio, M., Alabadí, D., Pérez-Gómez, J., García-Cárcel, L., Phillips, A. L., Hedden, P., & Blázquez, M. A. (2006). Transcriptional regulation of gibberellin metabolism genes by auxin signaling in *Arabidopsis*. Plant Physiology, 142(2), 553–563.
Fromm, J., & Lautner, S. (2007). Electrical signals and their physiological significance in plants. Plant, Cell and Environment, 30(3), 249–257.
Fu, J., Liu, H., Li, Y., Yu, H., Li, X., Xiao, J., & Wang, S. (2011). Manipulating broad-spectrum disease resistance by suppressing pathogen-induced auxin accumulation in rice. Plant Physiology, 155(1), 589–602.
Fu, Z. Q., & Dong, X. (2013). Systemic acquired resistance: turning local infection into global defense. Annual Review of Plant Biology, 64, 839–863.
Fu, Z. Q., Yan, S., Saleh, A., Wang, W., Ruble, J., Oka, N., Mohan, R., Spoel, S. H., Tada, Y., Zheng, N., & Dong, X. (2012). NPR3 and NPR4 are receptors for the immune signal salicylic acid in plants. Nature, 486(7402), 228–232.
Gaffney, T., Friedrich, L., Vernooij, B., Negrotto, D., Nye, G., Uknes, S., Ward, E., Kessmann, H., & Ryals, J. (1993). Requirement of salicylic acid for the induction of systemic acquired resistance. Science, 261(5122), 754–756.
Gallé, A., Lautner, S., Flexas, J., & Fromm, J. (2015). Environmental stimuli and physiological responses: The current view on electrical signalling. Environmental and Experimental Botany, 114, 15–21.
Galweiler, L., Guan, C., Müller, A., Wisman, E., Mendgen, K., Yephremov, A., & Palme, K. (1998). Regulation of polar auxin transport by AtPIN1 in *Arabidopsis* vascular tissue. Science, 282(5397), 2226–2230.
Gao, Q. M., Yu, K., Xia, Y., Shine, M. B., Wang, C., Navarre, D., Kachroo, A., & Kachroo, P. (2014). Mono- and digalactosyldiacylglycerol lipids function nonredundantly to regulate systemic acquired resistance in plants. Cell Reports, 9(5), 1681–1691.
Gaspar, T., Franck, T., Bisbis, B., Kevers, C., Jouve, L., Hausman, J. F., & Dommes, J. (2002). Concepts in plant stress physiology. Application to plant tissue cultures. Plant Growth Regulation, 37, 263–285.
Gatz, C. (2013). From pioneers to team players: TGA transcription factors provide a molecular link between different stress pathways. Molecular Plant-Microbe Interactions, 26(2), 151–159.
Gil, P., Dewey, E., Friml, J., Zhao, Y., Snowden, K. C., Putterill, J., Palme, K., Estelle, M., & Chory, J. (2001). BIG: A calossin-like protein required for polar auxin transport in *Arabidopsis*. Genes and Development, 15(15), 1985–1997.
González-Lamothe, R., El Oirdi, M., Brisson, N., & Bouarab, K. (2012). The conjugated auxin indole-3-acetic acid – aspartic acid promotes plant disease development. The Plant Cell, 24(2), 762–777.
Grant, M. R., & Jones, J. D. (2009). Hormone (dis) harmony moulds plant health and disease. Science, 324(5928), 750–752.
Gresshoff, P. M., Hayashi, S., Biswas, B., Mirzaei, S., Indrasumunar, A., Reid, D., Samuel, S., Tollenaere, A., van Hameren, B., Hastwell, A., Scott P., & Ferguson, B. J. (2015). The value of biodiversity in legume symbiotic nitrogen fixation and nodulation for biofuel and food production. Journal of Plant Physiology, 172, 128–136.
Griffiths, J., Murase, K., Rieu, I., Zentella, R., Zhang, Z. L., Powers, S. J., Gong, F., Phillips, A. L., Hedden, P., Sun, T. P., & Thomas, S. G. (2006). Genetic charac-

terization and functional analysis of the GID1 gibberellin receptors in *Arabidopsis*. The Plant Cell, 18(12), 3399–3414.
Grones, P., & Friml, J. (2015). Auxin transporters and binding proteins at a glance. Journal of Cell Science, 128(1), 1–7.
Grunewald, W., & Friml, J. (2010). The march of the PINs: Developmental plasticity by dynamic polar targeting in plant cells. The EMBO Journal, 29(16), 2700–2714.
Han, L. B., Li, Y. B., Wang, F. X., Wang, W. Y., Liu, J., Wu, J. H., Zhong, N. Q., Wu, S. J., Jiao, G. L., Wang, H. Y., & Xia, G. X. (2019). The cotton apoplastic protein CRR1 stabilizes chitinase 28 to facilitate defense against the fungal pathogen *Verticillium dahliae*. The Plant Cell, 31(2), 520–536.
Hartmann, M., Kim, D., Bernsdorff, F., Ajami-Rashidi, Z., Scholten, N., Schreiber, S., Zeier,T., Schuck, S., Reichel-Deland, V., & Zeier, J. (2017). Biochemical principles and functional aspects of pipecolic acid biosynthesis in plant immunity. Plant Physiology, 174(1), 124–153.
Hartmann, M., Zeier, T., Bernsdorff, F., Reichel-Deland, V., Kim, D., Hohmann, M., Scholten, N., Schuck S., Bräutigam, A., Hölzel, T., Ganter, C., & Zeier, J. (2018). Flavin monooxygenase-generated N-hydroxypipecolic acid is a critical element of plant systemic immunity. Cell, 173(2), 456–469.
Hauvermale, A. L., Ariizumi, T., & Steber, C. M. (2012). Gibberellin signaling: A theme and variations on DELLA repression. Plant Physiology, 160(1), 83–92.
Hayashi, M., Toriyama, K., Kondo, M., & Nishimura, M. (1998). 2, 4-Dichlorophenoxybutyric acid-resistant mutants of *Arabidopsis* have defects in glyoxysomal fatty acid β-oxidation. The Plant Cell, 10(2), 183–195.
Hayat, Q., Hayat, S., Irfan, M., & Ahmad, A. (2010). Effect of exogenous salicylic acid under changing environment: A review. Environmental and Experimental Botany, 68(1), 14–25.
Hedden, P., & Thomas, S. G. (2012). Gibberellin biosynthesis and its regulation. The Biochemical Journal, 444(1), 11–25.
Hedrich, R., Salvador-Recatalà, V., & Dreyer, I. (2016). Electrical wiring and long-distance plant communication. Trends in Plant Science, 21(5), 376–387.
Herrera-Vásquez, A., Fonseca, A., Ugalde, J. M., Lamig, L., Seguel, A., Moyano, T. C., Gutiérrez, R. A., Salinas, P., Vidal, E. A., & Holuigue, L. (2021). TGA class II transcription factors are essential to restrict oxidative stress in response to UV-B stress in *Arabidopsis*. Journal of Experimental Botany, 72(5), 1891–1905.
Heuberger, A. L., Robison, F. M., Lyons, S. M. A., Broeckling, C. D., & Prenni, J. E. (2014). Evaluating plant immunity using mass spectrometry-based metabolomics workflows. Frontiers in Plant Science, 5, 291.
Hilleary, R., & Gilroy, S. (2018). Systemic signaling in response to wounding and pathogens. Current Opinion in Plant Biology, 43, 57–62.
Hirano, K., Asano, K., Tsuji, H., Kawamura, M., Mori, H., Kitano, H., Ueguchi-Tanaka, M., & Matsuoka, M. (2010). Characterization of the molecular mechanism underlying gibberellin perception complex formation in rice. The Plant Cell, 22(8), 2680–2696.
Hu, J., Baker, A., Bartel, B., Linka, N., Mullen, R. T., Reumann, S., & Zolman, B. K. (2012). Plant peroxisomes: biogenesis and function. The Plant Cell, 24(6), 2279–2303.
Hua, C., Zhao, J. H., & Guo, H. S. (2018). Trans-kingdom RNA silencing in plant-fungal pathogen interactions. Molecular Plant, 11(2), 235–244.
Hull, A. K., Vij, R., & Celenza, J. L. (2000). *Arabidopsis* cytochrome P450s that catalyze the first step of tryptophan-dependent indole-3-acetic acid biosynthesis. Proceedings of the National Academy of Sciences, 97(5), 2379–2384.
Huot, B., Yao, J., Montgomery, B. L., & He, S. Y. (2014). Growth-defense tradeoffs in plants: A balancing act to optimize fitness. Molecular Plant, 7(8), 1267–1287.
Iizasa, E. I., Mitsutomi, M., & Nagano, Y. (2010). Direct binding of a plant LysM receptor-like kinase, LysM RLK1/CERK1, to chitin *in vitro*. Journal of Biological Chemistry, 285(5), 2996–3004.
Ikeda, A., Ueguchi-Tanaka, M., Sonoda, Y., Kitano, H., Koshioka, M., Futsuhara, Y., Matsuoka, M., & Yamaguchi, J. (2001). slender rice, a constitutive gibberellin response mutant, is caused by a null mutation of the SLR1 gene, an ortholog of the height-regulating gene GAI/RGA/RHT/D8. The Plant Cell, 13(5), 999–1010.
Itoh, H., Ueguchi-Tanaka, M., Sentoku, N., Kitano, H., Matsuoka, M., & Kobayashi, M. (2001). Cloning and functional analysis of two gibberellin 3 beta-hydroxylase genes that are differently expressed during the growth of rice. Proceedings of the National Academy of Sciences, 98(15), 8909–8914.
Iuchi, S., Suzuki, H., Kim, Y. C., Iuchi, A., Kuromori, T., Ueguchi-Tanaka, M., Asami, T., Yamaguchi, I., Matsuoka, M., Kobayashi, M., & Nakajima, M. (2007). Multiple loss-of-function of *Arabidopsis* gibberellin receptor AtGID1s completely shuts down a gibberellin signal. The Plant Journal for Cell and Molecular Biology, 50(6), 958–966.
Jagadeeswaran, G., Raina, S., Acharya, B. R., Maqbool, S. B., Mosher, S. L., Appel, H. M., Schultz, J. C., Klessig, D. F., & Raina, R. (2007). *Arabidopsis* GH3-like defense gene 1 is required for accumulation of salicylic acid, activation of defense responses and resistance to *Pseudomonas syringae*. The Plant Journal, 51(2), 234–246.
Jiang, Z., Zhu, S., Ye, R., Xue, Y., Chen, A., An, L., & Pei, Z. M. (2013). Relationship between NaCl- and H_2O_2-induced cytosolic Ca^{2+} increases in response to stress in *Arabidopsis*. PLoS One, 8(10), e76130.
Jones, J. D., & Dangl, J. L. (2006). The plant immune system. Nature, 444(7117), 323–329.
Jung, H. W., Tschaplinski, T. J., Wang, L., Glazebrook, J., & Greenberg, J. T. (2009). Priming in systemic plant immunity. Science, 324(5923), 89–91.
Kachroo, P., Lim, G. H., & Kachroo, A. (2016). Nitric oxide-mediated chemical signaling during systemic acquired resistance. Advances in Botanical Research, 77, 245–261.
Kaku, H., Nishizawa, Y., Ishii-Minami, N., Akimoto-Tomiyama, C., Dohmae, N., Takio, K., Minami, E., & Shibuya, N. (2006). Plant cells recognize chitin fragments for defense signaling through a plasma membrane receptor. Proceedings of the National Academy of Sciences, 103(29), 11086–11091.
Karpinski, S., Gabrys, H., Mateo, A., Karpinska, B., & Mullineaux, P. M. (2003). Light perception in plant disease defence signalling. Current Opinion in Plant Biology, 6(4), 390–396.
Kazan, K., & Lyons, R. (2014). Intervention of phytohormone pathways by pathogen effectors. The Plant Cell, 26(6), 2285–2309.
Kazan, K., & Manners, J. M. (2009). Linking development to defense: Auxin in plant-pathogen interactions. Trends in Plant Science, 14(7), 373–382.
Kiep, V., Vadassery, J., Lattke, J., Maaß, J. P., Boland, W., Peiter, E., & Mithöfer, A. (2015). Systemic cytosolic Ca^{2+} elevation is activated upon wounding and herbivory in *Arabidopsis*. New Phytologist, 207(4), 996–1004.
Kim, T. J., & Lim, G. H. (2023). Salicylic acid and mobile regulators of systemic immunity in plants: Transport and metabolism. Plants, 12(5), 1013.
Kombrink, A., Sánchez-Vallet, A., & Thomma, B. P. (2011). The role of chitin detection in plant-pathogen interactions. Microbes and Infection, 13(14–15), 1168–1176.
Korasick, D. A., Enders, T. A., & Strader, L. C. (2013). Auxin biosynthesis and storage forms. Journal of Experimental Botany, 64(9), 2541–2555.
Kreiser, M., Giblin, C., Murphy, R., Fiesel, P., Braun, L., Johnson, G., Wyse, D., & Cohen, J. D. (2016). Conversion of indole-3-butyric acid to indole-3-acetic acid in shoot tissue of hazelnut (*Corylus*) and elm (*Ulmus*). Journal of Plant Growth Regulation, 35, 710–721.
Langner, T., & Göhre, V. (2016). Fungal chitinases: Function, regulation, and potential roles in plant/pathogen interactions. Current Genetics, 62, 243–254.
Lechner, E., Achard, P., Vansiri, A., Potuschak, T., & Genschik, P. (2006). F-box proteins everywhere. Current Opinion in Plant Biology, 9(6), 631–638.
Lee, S., Cheng, H., King, K. E., Wang, W., He, Y., Hussain, A., Lo, J., Harberd, N. P., & Peng, J. (2002). Gibberellin regulates *Arabidopsis* seed germination via RGL2, a GAI/RGA-like gene whose expression is up-regulated following imbibition. Genes and Development, 16(5), 646–658.
Lee, W. S., Rudd, J. J., Hammond-Kosack, K. E., & Kanyuka, K. (2014). *Mycosphaerella graminicola* LysM effector-mediated stealth pathogenesis subverts recognition through both CERK1 and CEBiP homologues in wheat. Molecular Plant-Microbe Interactions, 27(3), 236–243.
Lehmann, T., Janowitz, T., Sánchez-Parra, B., Alonso, M. M. P., Trompetter, I., Piotrowski, M., & Pollmann, S. (2017). Arabidopsis NITRILASE 1 contributes to the regulation of root growth and development through modulation of auxin biosynthesis in seedlings. Frontiers in Plant Science, 8, 36.
Lester, D. R., Ross, J. J., Davies, P. J., & Reid, J. B. (1997). Mendel's stem length gene (Le) encodes a gibberellin 3 beta-hydroxylase. The Plant Cell, 9(8), 1435–1443.
Li, W., Chern, M., Yin, J., Wang, J., & Chen, X. (2019). Recent advances in broad-spectrum resistance to the rice blast disease. Current Opinion in Plant Biology, 50, 114–120.
Likić, S., Šola, I., Ludwig-Müller, J., & Rusak, G. (2014). Involvement of kaempferol in the defence response of virus infected *Arabidopsis thaliana*. European Journal of Plant Pathology, 138, 257–271.
Lim, G. H., Liu, H., Yu, K., Liu, R., Shine, M. B., Fernandez, J., Burch-Smith, T., Mobley, J. K., Mcletchie, N., Kachroo, A., & Kachroo, P. (2020). The plant cuticle regulates apoplastic transport of salicylic acid during systemic acquired resistance. Science Advances, 6(19), eaaz0478.
Lim, G. H., Shine, M. B., de Lorenzo, L., Yu, K., Cui, W., Navarre, D., Navarre, D., Hunt, A. G., Lee, J-Y., Kachroo, A., & Kachroo, P. (2016). Plasmodesmata localizing proteins regulate transport and signaling during systemic acquired immunity in plants. Cell Host and Microbe, 19(4), 541–549.
Liu, T., Liu, Z., Song, C., Hu, Y., Han, Z., She, J., Fan, F., Wang, J., Jin, C., Chang, J., Zhou, J-M., & Chai, J. (2012). Chitin-induced dimerization activates a plant immune receptor. Science, 336(6085), 1160–1164.
Liu, Y., Sun, T., Sun, Y., Zhang, Y., Radojičić, A., Ding, Y., Tian, H., Huang, X., Lan, J., Chen, S., Orduna, A. R., Zhang, K., Jetter, R., Li, X. & Zhang, Y. (2020). Diverse roles of the salicylic acid receptors NPR1 and NPR3/NPR4 in plant immunity. The Plant Cell, 32(12), 4002–4016.
Liu, Y., Zhu, Y., Xu, X., Sun, F., Yang, J., Cao, L., & Luo, X. (2019). OsMAPKKK5, a truncated mitogen-activated protein kinase kinase kinase 5, positively regulates plant height and yield in rice. The Crop Journal, 7(5), 707–714.
Liu, Z., Wu, Y., Yang, F., Zhang, Y., Chen, S., Xie, Q., Tian, X., & Zhou, J. M. (2013). BIK1 interacts with PEPRs to mediate ethylene-induced immunity. Proceedings of the National Academy of Sciences, 110(15), 6205–6210.

Lo Presti, L., Lanver, D., Schweizer, G., Tanaka, S., Liang, L., Tollot, M., Zuccaro, A., Reissmann, S., & Kahmann, R. (2015). Fungal effectors and plant susceptibility. Annual Review of Plant Biology, 66(1), 513–545.
López, M. A., Bannenberg, G., & Castresana, C. (2008). Controlling hormone signaling is a plant and pathogen challenge for growth and survival. Current Opinion in Plant Biology, 11(4), 420–427.
Lu, H., Greenberg, J. T., & Holuigue, L. (2016). Salicylic acid signaling networks. Frontiers in Plant Science, 7, 238.
Ludwig-Müller, J. (2011). Auxin conjugates: their role for plant development and in the evolution of land plants. Journal of Experimental Botany, 62(6), 1757–1773.
Ludwig-Müller, J. (2014). Auxin and the interaction between plants and microorganisms. In: Zažímalová, E., Petrášek, J., & Benková, E. (Eds.). Auxin and its role in plant development. Springer, Vienna.
Ludwig-Müller, J. (2015). Bacteria and fungi controlling plant growth by manipulating auxin: Balance between development and defense. Journal of Plant Physiology, 172, 4–12.
Ludwig-Müller, J., Raisig, A., & Hilgenberg, W. (1995). Uptake and transport of indole-3-butyric acid in *Arabidopsis thaliana*: Comparison with other natural and synthetic auxins. Journal of Plant Physiology, 147(3–4), 351–354.
MacMillan, J., & Takahashi, N. (1968). Proposed procedure for the allocation of trivial names to the gibberellins. Nature, 217(5124), 170–171.
Mammadova, R., Amri, A., Akparov, Z., Alo, F., Sheikhzamanova, F., Abbasov, M., Amrahov, N., Hajiyev, E., & Alizade, S. (2024). Evaluation of genetic diversity using SSR markers and link with drought response of Azerbaijani durum wheat (*Triticum durum* Desf.) genotypes. Genetic Resources and Crop Evolution, 71(5), 2203–2212.
Man, M., Zhu, Y., Liu, L., Luo, L., Han, X., Qiu, L., Li, F., Ren, M., & Xing, Y. (2022). Defense mechanisms of cotton *Fusarium* and *Verticillium* wilt and comparison of pathogenic response in cotton and humans. International Journal of Molecular Sciences, 23(20), 12217.
Maor, R., Haskin, S., Levi-Kedmi, H., & Sharon, A. (2004). In planta production of indole-3-acetic acid by *Colletotrichum gloeosporioides* f. sp. *aeschynomene*. Applied and Environmental Microbiology, 70(3), 1852–1854.
Martin, D. N., Proebsting, W. M., & Hedden, P. (1997). Mendel's dwarfing gene: cDNAs from the Le alleles and function of the expressed proteins. Proceedings of the National Academy of Sciences, 94(16), 8907–8911.
Maruri-López, I., Aviles-Baltazar, N. Y., Buchala, A., & Serrano, M. (2019). Intra- and extracellular journey of the phytohormone salicylic acid. Frontiers in Plant Science, 10, 423.
Mashiguchi, K., Tanaka, K., Sakai, T., Sugawara, S., Kawaide, H., Natsume, M., Hanada, A., Yaeno, T., Shirasu, K., Yao, H., McSteen, P., Zhao, Y., Hayashi, K., Kamiya, Y., & Kasahara, H. (2011). The main auxin biosynthesis pathway in *Arabidopsis*. Proceedings of the National Academy of Sciences, 108(45), 18512–18517.
Mehdiyeva, G. M., Bairamov, M. R., Gasimov, E. K., & Rzayev, F. H. (2023). Synthesis and antimicrobial activity of a number of 3-substituted-8-allylbenzoxazines and their hydrobromic salts. Journal of the Chemical Society of Pakistan, 45(6), 540–550.
Mikkelsen, M. D., Hansen, C. H., Wittstock, U., & Halkier, B. A. (2000). Cytochrome P450 CYP79B2 from *Arabidopsis* catalyzes the conversion of tryptophan to indole-3-acetaldoxime, a precursor of indole glucosinolates and indole-3-acetic acid. Journal of Biological Chemistry, 275(43), 33712–33717.
Mizutani, M., & Ohta, D. (2010). Diversification of P450 genes during land plant evolution. Annual Review of Plant Biology, 61, 291–315.
Mockaitis, K., & Estelle, M. (2008). Auxin receptors and plant development: A new signaling paradigm. Annual Review of Cell and Developmental Biology, 24, 55–80.
Morrone, D., Chen, X., Coates, R. M., & Peters, R. J. (2010). Characterization of the kaurene oxidase CYP701A3, a multifunctional cytochrome P450 from gibberellin biosynthesis. The Biochemical Journal, 431(3), 337–344.
Mou, Z., Fan, W., & Dong, X. (2003). Inducers of plant systemic acquired resistance regulate NPR1 function through redox changes. Cell, 113(7), 935–944.
Mravec J., Kubeš M., Bielach A., Gaykova V., Petrášek J., Skupa P., Chand S., Benková E., Zažímalová E., & Friml, J. (2008). Interaction of PIN and PGP transport mechanisms in auxin distribution-dependent development. Development, 135(20), 3345–3354.
Mukherjee, S., Roy, S., & Corpas, F. J. (2024). Aquaporins: A vital nexus in H_2O_2-gasotransmitter signaling. Trends in Plant Science, 29(6), 681–693.
Müller, A., Guan, C., Gälweiler, L., Tänzler, P., Huijser, P., Marchant, A., Parry, G., Bennett, M., Wisman, E., & Palme, K. (1998). AtPIN2 defines a locus of *Arabidopsis* for root gravitropism control. The EMBO Journal, 17(23), 6903–6911.
Muradov, M. B., Mammadyarova, S. J., Eyvazova, G. M., Balayeva, O. O., Melikova, S. Z., Sadigov, N., Abdullayev, M. I., Musayeva, N., Gasimov, E. K., Rzayev, F. H., & Hasanova, I. (2024). Sonochemical synthesis and characterization of structural, optical and dielectric properties of Ag-doped Co_3O_4 nanoparticles. Journal of Cluster Science, 35(3), 845–861.
Murase, K., Hirano, Y., Sun, T. P., & Hakoshima, T. (2008). Gibberellin-induced DELLA recognition by the gibberellin receptor GID1. Nature, 456(7221), 459–463.
Naseem, M., Kaltdorf, M., & Dandekar, T. (2015). The nexus between growth and defence signalling: auxin and cytokinin modulate plant immune response pathways. Journal of Experimental Botany, 66(16), 4885–4896.
Návarová, H., Bernsdorff, F., Döring, A. C., & Zeier, J. (2012). Pipecolic acid, an endogenous mediator of defense amplification and priming, is a critical regulator of inducible plant immunity. The Plant Cell, 24(12), 5123–5141.
Nobuta, K., Okrent, R. A., Stoutemyer, M., Rodibaugh, N., Kempema, L., Wildermuth, M. C., & Innes, R. W. (2007). The GH3 acyl adenylase family member PBS3 regulates salicylic acid-dependent defense responses in *Arabidopsis*. Plant Physiology, 144(2), 1144–1156.
Normanly, J. (2010). Approaching cellular and molecular resolution of auxin biosynthesis and metabolism. Cold Spring Harbor Perspectives in Biology, 2(1), a001594.
O'Donnell, P. J., Schmelz, E. A., Moussatche, P., Lund, S. T., Jones, J. B., & Klee, H. J. (2003). Susceptible to intolerance – a range of hormonal actions in a susceptible *Arabidopsis* pathogen response. The Plant Journal, 33(2), 245–257.
Oetiker, J. H., & Aeschbacher, G. (1997). Temperature-sensitive plant cells with shunted indole-3-acetic acid conjugation. Plant Physiology, 114(4), 1385–1395.
Ogawa, M., Kusano, T., Katsumi, M., & Sano, H. (2000). Rice gibberellin-insensitive gene homolog, OsGAI, encodes a nuclear-localized protein capable of gene activation at transcriptional level. Gene, 245(1), 21–29.
Olatunbosun, A., Nigar, H., Rovshan, K., Nurlan, A., Boyukhanim, J., Narmina, A., & Ibrahim, A. (2023). Comparative impact of nanoparticles on salt resistance of wheat plants. MethodsX, 11, 102371.
Olszewski, N., Sun, T. P., & Gubler, F. (2002). Gibberellin signaling: Biosynthesis, catabolism, and response pathways. The Plant Cell, 14(suppl. 1), S61–S80.
O'Neill, D. P., Davidson, S. E., Clarke, V. C., Yamauchi, Y., Yamaguchi, S., Kamiya, Y., Reid, J. B., & Ross, J. J. (2010). Regulation of the gibberellin pathway by auxin and DELLA proteins. Planta, 232(5), 1141–1149.
Osbourn, A. E. (1996). Preformed antimicrobial compounds and plant defense against fungal attack. The Plant Cell, 8(10), 1821–1831.
Paciorek, T., & Friml, J. (2006). Auxin signaling. Journal of Cell Science, 119(7), 1199–1202.
Panstruga, R., & Dodds, P. N. (2009). Terrific protein traffic: The mystery of effector protein delivery by filamentous plant pathogens. Science, 324(5928), 748–750.
Park, J. E., Park, J. Y., Kim, Y. S., Staswick, P. E., Jeon, J., Yun, J., Kim, S. Y., Kim, J., Lee, Y. H., & Park, C. M. (2007). GH3-mediated auxin homeostasis links growth regulation with stress adaptation response in *Arabidopsis*. Journal of Biological Chemistry, 282(13), 10036–10046.
Peng, J., Carol, P., Richards, D. E., King, K. E., Cowling, R. J., Murphy, G. P., & Harberd, N. P. (1997). The *Arabidopsis* GAI gene defines a signaling pathway that negatively regulates gibberellin responses. Genes and Development, 11(23), 3194–3205.
Petutschnig, E. K., Jones, A. M., Serazetdinova, L., Lipka, U., & Lipka, V. (2010). The lysin motif receptor-like kinase (LysM-RLK) CERK1 is a major chitin-binding protein in *Arabidopsis thaliana* and subject to chitin-induced phosphorylation. Journal of Biological Chemistry, 285(37), 28902–28911.
Pollmann, S., Müller, A., Piotrowski, M., & Weiler, E. W. (2002). Occurrence and formation of indole-3-acetamide in *Arabidopsis thaliana*. Planta, 216, 155–161.
Pollmann, S., Neu, D., & Weiler, E. W. (2003). Molecular cloning and characterization of an amidase from *Arabidopsis thaliana* capable of converting indole-3-acetamide into the plant growth hormone, indole-3-acetic acid. Phytochemistry, 62(3), 293–300.
Pusztahelyi, T. (2018). Chitin and chitin-related compounds in plant-fungal interactions. Mycology, 9(3), 189–201.
Qi, J., Wang, J., Gong, Z., & Zhou, J. M. (2017). Apoplastic ROS signaling in plant immunity. Current Opinion in Plant Biology, 38, 92–100.
Qin, X., Liu, J. H., Zhao, W. S., Chen, X. J., Guo, Z. J., & Peng, Y. L. (2013). Gibberellin 20-oxidase gene OsGA20ox3 regulates plant stature and disease development in rice. Molecular Plant-Microbe Interactions, 26(2), 227–239.
Qiu, X. M., Sun, Y. Y., Ye, X. Y., & Li, Z. G. (2020). Signaling role of glutamate in plants. Frontiers in Plant Science, 10, 1743.
Rashid, A. N., Janda, M., Mahmud, M. Z., Valentová, O., Burketová, L., & Alekber, Q. A. (2019). Influence of salt stress on the flg22 induced ROS production in *Arabidopsis thaliana* leaves. Pakistan Journal of Botany, 53(5), 1605–1610.
Rashotte, A. M., Poupart, J., Waddell, C. S., & Muday, G. K. (2003). Transport of the two natural auxins, indole-3-butyric acid and indole-3-acetic acid, in *Arabidopsis*. Plant Physiology, 133(2), 761–772.
Razem, F. A., Baron, K., & Hill, R. D. (2006). Turning on gibberellin and abscisic acid signaling. Current Opinion in Plant Biology, 9(5), 454–459.
Reineke, G., Heinze, B., Schirawski, J., Buettner, H., Kahmann, R., & Basse, C. W. (2008). Indole-3-acetic acid (IAA) biosynthesis in the smut fungus *Ustilago maydis* and its relevance for increased IAA levels in infected tissue and host tumour formation. Molecular Plant Pathology, 9(3), 339–355.

Rekhter, D., Lüdke, D., Ding, Y., Feussner, K., Zienkiewicz, K., Lipka, V., Wiermer, M., Zhang, Y., & Feussner, I. (2019). Isochorismate-derived biosynthesis of the plant stress hormone salicylic acid. Science, 365(6452), 498–502.
Roychoudhury, A., & Aftab, T. (2021). Phytohormones, plant growth regulators and signaling molecules: Cross-talk and stress responses. Plant Cell Reports, 40, 1301–1303.
Růžička, K., Strader, L. C., Bailly, A., Yang, H., Blakeslee, J., Łangowski, Ł., Nejedlá, E., Fujita, H., Itoh, H., Syōno, K., Hejátko, J., Gray, W. M., Martinoia, E., Geisler, M., Bartel, B., Murphy, A. S., & Friml, J. (2010). *Arabidopsis* PIS1 encodes the ABCG37 transporter of auxinic compounds including the auxin precursor indole-3-butyric acid. Proceedings of the National Academy of Sciences, 107(23), 10749–10753.
Ryals, J., Weymann, K., Lawton, K., Friedrich, L., Ellis, D., Steiner, H. Y., Johnson, J., Delaney, T. P., Jesse, T., Vos, P., & Uknes, S. (1997). The *Arabidopsis* NIM1 protein shows homology to the mammalian transcription factor inhibitor I kappa B. The Plant Cell, 9(3), 425–439.
Salanenka, Y., Verstraeten, I., Löfke, C., Tabata, K., Naramoto, S., Glanc, M., & Friml, J. (2018). Gibberellin DELLA signaling targets the retromer complex to redirect protein trafficking to the plasma membrane. Proceedings of the National Academy of Sciences, 115(14), 3716–3721.
Santner, A., & Estelle, M. (2009). Recent advances and emerging trends in plant hormone signalling. Nature, 459(7250), 1071–1078.
Schaller, G. E., Bishopp, A., & Kieber, J. J. (2015). The yin-yang of hormones: Cytokinin and auxin interactions in plant development. The Plant Cell, 27(1), 44–63.
Schrader, J., Baba, K., May, S. T., Palme, K., Bennett, M., Bhalerao, R. P., & Sandberg, G. (2003). Polar auxin transport in the wood-forming tissues of hybrid aspen is under simultaneous control of developmental and environmental signals. Proceedings of the National Academy of Sciences, 100(17), 10096–10101.
Shah, J., & Zeier, J. (2013). Long-distance communication and signal amplification in systemic acquired resistance. Frontiers in Plant Science, 4(30), 43095.
Shah, J., Tsui, F., & Klessig, D. F. (1997). Characterization of a salicylic acid-insensitive mutant (sai1) of *Arabidopsis thaliana*, identified in a selective screen utilizing the SA-inducible expression of the tms2 gene. Molecular Plant-Microbe Interactions, 10(1), 69–78.
Shibuya, N., Kaku, H., Kuchitsu, K., & Maliarik, M. J. (1993). Identification of a novel high-affinity binding site for N-acetylchitooligosaccharide elicitor in the membrane fraction from suspension-cultured rice cells. Febs Letters, 329(1–2), 75–78.
Shimada, A., Ueguchi-Tanaka, M., Nakatsu, T., Nakajima, M., Naoe, Y., Ohmiya, H., Kato, H., & Matsuoka, M. (2008). Structural basis for gibberellin recognition by its receptor GID1. Nature, 456(7221), 520–523.
Shimizu, T., Nakano, T., Takamizawa, D., Desaki, Y., Ishii- Minami, N., Nishizawa, Y., Minami, E., Okada, K., Yamane, H., Kaku, H., & Shibuya, N. (2010). Two LysM receptor molecules, CEBiP and OsCERK1, cooperatively regulate chitin elicitor signaling in rice. The Plant Journal, 64(2), 204–214.
Shinya, T., Motoyama, N., Ikeda, A., Wada, M., Kamiya, K., Hayafune, M., Kaku, H., & Shibuya, N. (2012). Functional characterization of CEBiP and CERK1 homologs in *Arabidopsis* and rice reveals the presence of different chitin receptor systems in plants. Plant and Cell Physiology, 53(10), 1696–1706.
Shinya, T., Nakagawa, T., Kaku, H., & Shibuya, N. (2015). Chitin-mediated plant-fungal interactions: Catching, hiding and handshaking. Current Opinion in Plant Biology, 26, 64–71.
Shinya, T., Yamaguchi, K., Desaki, Y., Yamada, K., Narisawa, T., Kobayashi, Y., Maeda, K., Suzuki, M., Tanimoto, T., Takeda, J., Nakashima, M., Funama, R., Narusaka, M., Narusaka, Y., Kaku, H., Kawasaki T., & Shibuya, N. (2014). Selective regulation of the chitin-induced defense response by the *Arabidopsis* receptor-like cytoplasmic kinase PBL 27. The Plant Journal, 79(1), 56–66.
Silverstone, A. L., Ciampaglio, C. N., & Sun, T. (1998). The Arabidopsis RGA gene encodes a transcriptional regulator repressing the gibberellin signal transduction pathway. The Plant Cell, 10(2), 155–169.
Spaepen, S., Vanderleyden, J., & Remans, R. (2007). Indole-3-acetic acid in microbial and microorganism-plant signaling. FEMS Microbiology Reviews, 31(4), 425–448.
Staswick, P. E., Serban, B., Rowe, M., Tiryaki, I., Maldonado, M. T., Maldonado, M. C., & Suza, W. (2005). Characterization of an *Arabidopsis* enzyme family that conjugates amino acids to indole-3-acetic acid. The Plant Cell, 17(2), 616–627.
Stepanova, A. N., Robertson-Hoyt, J., Yun, J., Benavente, L. M., Xie, D. Y., Doležal, K., Schlereth, A., Jürgens, G., & Alonso, J. M. (2008). TAA1-mediated auxin biosynthesis is essential for hormone crosstalk and plant development. Cell, 133(1), 177–191.
Stepanova, A. N., Yun, J., Robles, L. M., Novak, O., He, W., Guo, H., Ljung, K., & Alonso, J. M. (2011). The *Arabidopsis* YUCCA1 flavin monooxygenase functions in the indole-3-pyruvic acid branch of auxin biosynthesis. The Plant Cell, 23(11), 3961–3973.
Stotz, H. U., Mueller, S., Zoeller, M., Mueller, M. J., & Berger, S. (2013). TGA transcription factors and jasmonate-independent COI1 signalling regulate specific plant responses to reactive oxylipins. Journal of Experimental Botany, 64(4), 963–975.
Strader, L. C., & Bartel, B. (2008). A new path to auxin. Nature Chemical Biology, 4(6), 337–339.
Strader, L. C., & Bartel, B. (2011). Transport and metabolism of the endogenous auxin precursor indole-3-butyric acid. Molecular Plant, 4(3), 477–486.
Sugawara, S., Hishiyama, S., Jikumaru, Y., Hanada, A., Nishimura, T., Koshiba, T., Zhao, Y., Kamiya, Y., & Kasahara, H. (2009). Biochemical analyses of indole-3-acetaldoxime-dependent auxin biosynthesis in *Arabidopsis*. Proceedings of the National Academy of Sciences, 106(13), 5430–5435.
Sukhova, E., & Sukhov, V. (2021). Electrical signals, plant tolerance to actions of stressors, and programmed cell death: is interaction possible? Plants, 10(8), 1704.
Sun, T., Busta, L., Zhang, Q., Ding, P., Jetter, R., & Zhang, Y. (2018). TGACG–binding factor 1 (TGA 1) and TGA 4 regulate salicylic acid and pipecolic acid biosynthesis by modulating the expression of Systemic Acquired Resistance Deficient 1 (SARD 1) and Calmodulin-Binding Protein 60g (CBP 60g). New Phytologist, 217(1), 344–354.
Sun, T., Zhang, Y., Li, Y., Zhang, Q., Ding, Y., & Zhang, Y. (2015). ChIP-seq reveals broad roles of SARD1 and CBP60g in regulating plant immunity. Nature Communications, 6(1), 10159.
Suzuki, N., & Mittler, R. (2012). Reactive oxygen species-dependent wound responses in animals and plants. Free Radical Biology and Medicine, 53(12), 2269–2276.
Swarup, R., Kargul, J., Marchant, A., Zadik, D., Rahman, A., Mills, R., Yemm, A., May, S., Williams, L., Millner, P., Tsurumi, S., Moore, I., Napier, R., Kerr, I. D., & Bennett, M. J. (2004). Structure-function analysis of the presumptive *Arabidopsis* auxin permease AUX1. The Plant Cell, 16(11), 3069–3083.
Szechyńska-Hebda, M., & Karpiński, S. (2013). Light intensity-dependent retrograde signalling in higher plants. Journal of Plant Physiology, 170(17), 1501–1516.
Tada, Y., Spoel, S. H., Pajerowska-Mukhtar, K., Mou, Z., Song, J., Wang, C., Zuo, J., & Dong, X. (2008). Plant immunity requires conformational charges of NPR1 via S-nitrosylation and thioredoxins. Science, 321(5891), 952–956.
Takahashi, F., Hanada, K., Kondo, T., & Shinozaki, K. (2019). Hormone-like peptides and small coding genes in plant stress signaling and development. Current Opinion in Plant Biology, 51, 88–95.
Takase, T., Nakazawa, M., Ishikawa, A., Kawashima, M., Ichikawa, T., Takahashi, N., Shimada, H., Manabe, K., & Matsui, M. (2004). ydk1-D, an auxin–responsive GH3 mutant that is involved in hypocotyl and root elongation. The Plant Journal, 37(4), 471–483.
Tanaka, E., Koga, H., Mori, M., & Mori, M. (2011). Auxin production by the rice blast fungus and its localization in host tissue. Journal of Phytopathology, 159(7–8), 522–530.
Tanaka, S., Ichikawa, A., Yamada, K., Tsuji, G., Nishiuchi, T., Mori, M., Koga, H., Nishizawa, Y., O'Connell, R., & Kubo, Y. (2010). HvCEBiP, a gene homologous to rice chitin receptor CEBiP, contributes to basal resistance of barley to *Magnaporthe oryzae*. BMC Plant Biology, 10, 288.
Tao, Y., Ferrer, J. L., Ljung, K., Pojer, F., Hong, F., Long, J. A., Li, L., Moreno, J. E., Bowman, M. E., Ivans, L. J., Cheng, Y., Lim, J., Zhao, Y., Ballaré, C. L., Sandberg, G., Noel, J. P., & Chory, J. (2008). Rapid synthesis of auxin via a new tryptophan-dependent pathway is required for shade avoidance in plants. Cell, 133(1), 164–176.
Thilmony, R., Underwood, W., & He, S. Y. (2006). Genome-wide transcriptional analysis of the *Arabidopsis thaliana* interaction with the plant pathogen *Pseudomonas syringae* pv. *tomato* DC3000 and the human pathogen *Escherichia coli* O157: H7. The Plant Journal, 46(1), 34–53.
Torres, M. A., Dangl, J. L., & Jones, J. D. (2002). Arabidopsis gp91phox homologues AtrbohD and AtrbohF are required for accumulation of reactive oxygen intermediates in the plant defense response. Proceedings of the National Academy of Sciences, 99(1), 517–522.
Tripathi, D. K., Vishwakarma, K., Singh, V. P., Prakash, V., Sharma, S., Muneer, S., Nikolic, M., Deshmukh, R., Vaculík, M., & Corpas, F. J. (2021). Silicon cross-talk with reactive oxygen species, phytohormones and other signaling molecules. Journal of Hazardous Materials, 408, 124820.
Tripathi, D., Jiang, Y. L., & Kumar, D. (2010). SABP2, a methyl salicylate esterase is required for the systemic acquired resistance induced by acibenzolar-S-methyl in plants. FEBS Letters, 584(15), 3458-3463.
Truman, W. M., Bennett, M. H., Turnbull, C. G., & Grant, M. R. (2010). *Arabidopsis* auxin mutants are compromised in systemic acquired resistance and exhibit aberrant accumulation of various indolic compounds. Plant Physiology, 152(3), 1562–1573.
Tyler, L., Thomas, S. G., Hu, J., Dill, A., Alonso, J. M., Ecker, J. R., & Sun, T. P. (2004). Della proteins and gibberellin-regulated seed germination and floral development in *Arabidopsis*. Plant Physiology, 135(2), 1008–1019.
Ueguchi-Tanaka, M., & Matsuoka, M. (2010). The perception of gibberellins: Clues from receptor structure. Current Opinion in Plant Biology, 13(5), 503–508.
Ueguchi-Tanaka, M., Ashikari, M., Nakajima, M., Itoh, H., Katoh, E., Kobayashi, M., Chow, T. Y., Hsing, Y. I., Kitano, H., Yamaguchi, I., & Matsuoka, M. (2005). Gibberellin insensitive DWARF1 encodes a soluble receptor for gibberellin. Nature, 437(7059), 693–698.

Ueguchi-Tanaka, M., Nakajima, M., Katoh, E., Ohmiya, H., Asano, K., Saji, S., Hongyu, X., Ashikari, M., Kitano, H., Yamaguchi, I., & Matsuoka, M. (2007). Molecular interactions of a soluble gibberellin receptor, GID1, with a rice DELLA protein, SLR1, and gibberellin. The Plant Cell, 19(7), 2140–2155.

Uzunova, V. V., Quareshy, M., Del Genio, C. I., & Napier, R. M. (2016). Tomographic docking suggests the mechanism of auxin receptor TIR1 selectivity. Open Biology, 6(10), 160139.

Valiyeva, G. G., Di Palma, L., Hajiyeva, S. R., Ramazanov, M. A., Hajiyeva, F. V., Gasimov, E. K., & Rzayev, F. H. (2022). Reuse of bimetallic nanoparticles for nitrate reduction. IEEE Transactions on Nanotechnology, 22, 613–619.

Van Damme, M., Huibers, R. P., Elberse, J., & Van den Ackerveken, G. (2008). *Arabidopsis* DMR6 encodes a putative 2OG-Fe (II) oxygenase that is defense-associated but required for susceptibility to downy mildew. The Plant Journal, 54(5), 785–793.

van Schie, C. C., Ament, K., Schmidt, A., Lange, T., Haring, M. A., & Schuurink, R. C. (2007). Geranyl diphosphate synthase is required for biosynthesis of gibberellins. The Plant Journal, 52(4), 752–762.

Vázquez-Hernández, M. C., Parola-Contreras, I., Montoya-Gómez, L. M., Torres-Pacheco, I., Schwarz, D., & Guevara-González, R. G. (2019). Eustressors: Chemical and physical stress factors used to enhance vegetables production. Scientia Horticulturae, 250, 223–229.

Vieten, A., Sauer, M., Brewer, P. B., & Friml, J. (2007). Molecular and cellular aspects of auxin-transport-mediated development. Trends in Plant Science, 12(4), 160–168.

Vlot, A. C., Dempsey, D. M. A., & Klessig, D. F. (2009). Salicylic acid, a multifaceted hormone to combat disease. Annual Review of Phytopathology, 47, 177–206.

Vorobyova, V., Skiba, M., Vinnichuk, K., & Vasyliev, G. (2024). Synthesis of gold nanoparticles using plum waste extract with green solvents. Sustainable Chemistry for the Environment, 6, 100086.

Wang, C., El-Shetehy, M., Shine, M. B., Yu, K., Navarre, D., Wendehenne, D., Kachroo, A., & Kachroo, P. (2014). Free radicals mediate systemic acquired resistance. Cell Reports, 7(2), 348–355.

Wang, C., Liu, R., Lim, G. H., de Lorenzo, L., Yu, K., Zhang, K., Hunt, A. G., Kachroo, A., & Kachroo, P. (2018). Pipecolic acid confers systemic immunity by regulating free radicals. Science Advances, 4(5), eaar4509.

Wang, D., Pajerowska-Mukhtar, K., Culler, A. H., & Dong, X. (2007). Salicylic acid inhibits pathogen growth in plants through repression of the auxin signaling pathway. Current Biology, 17(20), 1784–1790.

Wang, F., Wang, C., Yan, Y., Jia, H., & Guo, X. (2016). Overexpression of cotton GhMPK11 decreases disease resistance through the gibberellin signaling pathway in transgenic *Nicotiana benthamiana*. Frontiers in Plant Science, 7(689), 177978.

Wang, L., Tsuda, K., Sato, M., Cohen, J. D., Katagiri, F., & Glazebrook, J. (2009). *Arabidopsis* CaM binding protein CBP60g contributes to MAMP-induced SA accumulation and is involved in disease resistance against Pseudomonas syringae. PLoS Pathogens, 5(2), e1000301.

Wang, L., Tsuda, K., Truman, W., Sato, M., Nguyen, L. V., Katagiri, F., & Glazebrook, J. (2011). CBP60g and SARD1 play partially redundant critical roles in salicylic acid signaling. The Plant Journal, 67(6), 1029–1041.

Wang, P., Zhou, L., Jamieson, P., Zhang, L., Zhao, Z., Babilonia, K., Shao, W., Wu, L., Mustafa, R., Amin, I., Diomaiuti, A., Pontiggia, D., Ferrari, S., Hou, Y., He, P., & Shan, L. (2020). The cotton wall-associated kinase GhWAK7A mediates responses to fungal wilt pathogens by complexing with the chitin sensory receptors. The Plant Cell, 32(12), 3978–4001.

Weiss, D., & Ori, N. (2007). Mechanisms of cross talk between gibberellin and other hormones. Plant Physiology, 144(3), 1240–1246.

Wen, C. K., & Chang, C. (2002). *Arabidopsis* RGL1 encodes a negative regulator of gibberellin responses. The Plant Cell, 14(1), 87–100.

Wendehenne, D., Durner, J., & Klessig, D. F. (2004). Nitric oxide: A new player in plant signalling and defence responses. Current Opinion in Plant Biology, 7(4), 449–455.

Wendehenne, D., Gao, Q. M., Kachroo, A., & Kachroo, P. (2014). Free radical-mediated systemic immunity in plants. Current Opinion in Plant Biology, 20, 127–134.

Wildermuth, M. C., Dewdney, J., Wu, G., & Ausubel, F. M. (2001). Isochorismate synthase is required to synthesize salicylic acid for plant defence. Nature, 414(6863), 562–565.

Williams, J., Phillips, A. L., Gaskin, P., & Hedden, P. (1998). Function and substrate specificity of the gibberellin 3beta-hydroxylase encoded by the *Arabidopsis* GA4 gene. Plant Physiology, 117(2), 559–563.

Willige, B. C., Ghosh, S., Nill, C., Zourelidou, M., Dohmann, E. M., Maier, A., & Schwechheimer, C. (2007). The DELLA domain of GA INSENSITIVE mediates the interaction with the GA INSENSITIVE DWARF1A gibberellin receptor of *Arabidopsis*. The Plant Cell, 19(4), 1209–1220.

Willige, B. C., Isono, E., Richter, R., Zourelidou, M., & Schwechheimer, C. (2011). Gibberellin regulates PIN-FORMED abundance and is required for auxin transport-dependent growth and development in *Arabidopsis thaliana*. The Plant Cell, 23(6), 2184–2195.

Won, C., Shen, X., Mashiguchi, K., Zheng, Z., Dai, X., Cheng, Y., Kasahara, H., Kamiya, Y., Chory, J., & Zhao, Y. (2011). Conversion of tryptophan to indole-3-acetic acid by tryptophan aminotransferases of *Arabidopsis* and Yuccas in *Arabidopsis*. Proceedings of the National Academy of Sciences, 108(45), 18518–18523.

Woodward, A. W., & Bartel, B. (2005). Auxin: Regulation, action, and interaction. Annals of Botany, 95(5), 707–735.

Wu, Y., Zhang, D., Chu, J. Y., Boyle, P., Wang, Y., Brindle, I. D., De Luca, V., & Després, C. (2012). The *Arabidopsis* NPR1 protein is a receptor for the plant defense hormone salicylic acid. Cell Reports, 1(6), 639–647.

Xie, C., Yang, L., & Gai, Y. (2023). MAPKKKs in plants: Multidimensional regulators of plant growth and stress responses. International Journal of Molecular Sciences, 24(4), 4117.

Yamada, K., Yamaguchi, K., Shirakawa, T., Nakagami, H., Mine, A., Ishikawa, K., Fujiwara, M., Narusaka, M., Narusaka, Y., Ichimura, K., Kobayashi, Y., Matsui, H., Nomura, Y., Nomoto, M., Tada, Y., Fukao, Y., Fukamizo, T., Tsuda, K., Shirasu, K., Shibuya, N., & Kawasaki, T. (2016). The *Arabidopsis* CERK 1-associated kinase PBL 27 connects chitin perception to MAPK activation. The EMBO Journal, 35(22), 2468–2483.

Yamada, M., Greenham, K., Prigge, M. J., Jensen, P. J., & Estelle, M. (2009). The Transport Inhibitor Response2 gene is required for auxin synthesis and diverse aspects of plant development. Plant Physiology, 151(1), 168–179.

Yamaguchi, K., Yamada, K., Ishikawa, K., Yoshimura, S., Hayashi, N., Uchihashi, K., Ishihama, N., Kishi-Kaboshi, M., Takahashi, A., Tsuge, S., Ochiai, H., Tada, Y., Shimamoto, K., Yoshioka, H., & Kawasaki, T. (2013). A receptor-like cytoplasmic kinase targeted by a plant pathogen effector is directly phosphorylated by the chitin receptor and mediates rice immunity. Cell Host and Microbe, 13(3), 347–357.

Yang, C., Wang, E., & Liu, J. (2022). CERK1, more than a co-receptor in plant-microbe interactions. New Phytologist, 234(5), 1606–1613.

Yin, C., Gan, L., Ng, D., Zhou, X., & Xia, K. (2007). Decreased panicle-derived indole-3-acetic acid reduces gibberellin A1 level in the uppermost internode, causing panicle enclosure in male sterile rice Zhenshan 97A. Journal of Experimental Botany, 58(10), 2441–2449.

Yoshioka, H., Asai, S., Yoshioka, M., & Kobayashi, M. (2009). Molecular mechanisms of generation for nitric oxide and reactive oxygen species, and role of the radical burst in plant immunity. Molecules and Cells, 28(4), 321–329.

Yu, K., Soares, J. M., Mandal, M. K., Wang, C., Chanda, B., Gifford, A. N., Fowler, J. S., Navarre, D., Kachroo, A., & Kachroo, P. (2013). A feedback regulatory loop between G3P and lipid transfer proteins DIR1 and AZI1 mediates azelaic-acid-induced systemic immunity. Cell Reports, 3(4), 1266–1278.

Yu, M., Liu, K., Liu, S., Chen, H., Zhou, L., & Liu, Y. (2017). Effect of exogenous IAA on tension wood formation by facilitating polar auxin transport and cellulose biosynthesis in hybrid poplar (*Populus deltoids* × *Populus nigra*) wood. Holzforschung, 71(2), 179–188.

Yu, X., Cui, X., Wu, C., Shi, S., & Yan, S. (2022). Salicylic acid inhibits gibberellin signaling through receptor interactions. Molecular Plant, 15(11), 1759–1771.

Zander, M., La Camera, S., Lamotte, O., Métraux, J. P., & Gatz, C. (2010). *Arabidopsis thaliana* class-II TGA transcription factors are essential activators of jasmonic acid/ethylene-induced defense responses. The Plant Journal, 61(2), 200–210.

Zazimalova, E., Murphy, A. S., Yang, H., Hoyerova, K., & Hosek, P. (2010). Auxin transporters – why so many? Cold Spring Harbor Perspectives in Biology, 2(3), a001552.

Zhang, K., Halitschke, R., Yin, C., Liu, C. J., & Gan, S. S. (2013). Salicylic acid 3-hydroxylase regulates *Arabidopsis* leaf longevity by mediating salicylic acid catabolism. Proceedings of the National Academy of Sciences, 110(36), 14807–14812.

Zhang, X., Xu, X., Yu, Y., Chen, C., Wang, J., Cai, C., & Guo, W. (2016). Integration analysis of MKK and MAPK family members highlights potential MAPK signaling modules in cotton. Scientific Reports, 6(1), 29781.

Zhang, Y., & Li, X. (2019). Salicylic acid: biosynthesis, perception, and contributions to plant immunity. Current Opinion in Plant Biology, 50, 29–36.

Zhang, Y., Cheng, Y. T., Qu, N., Zhao, Q., Bi, D., & Li, X. (2006). Negative regulation of defense responses in *Arabidopsis* by two NPR1 paralogs. The Plant Journal, 48(5), 647–656.

Zhang, Y., Fan, W., Kinkema, M., Li, X., & Dong, X. (1999). Interaction of NPR1 with basic leucine zipper protein transcription factors that bind sequences required for salicylic acid induction of the PR-1 gene. Proceedings of the National Academy of Sciences, 96(11), 6523–6528.

Zhang, Y., Goritschnig, S., Dong, X., & 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. The Plant Cell, 15(11), 2636–2646.

Zhang, Y., Tessaro, M. J., Lassner, M., & Li, X. (2003). Knockout analysis of *Arabidopsis* transcription factors TGA2, TGA5, and TGA6 reveals their redundant

and essential roles in systemic acquired resistance. The Plant Cell, 15(11), 2647–2653.
Zhang, Y., Xu, S., Ding, P., Wang, D., Cheng, Y. T., He, J., Gao, M., Xu, F., Li, Y., Zhu, Z., Li, X., & Zhang, Y. (2010). Control of salicylic acid synthesis and systemic acquired resistance by two members of a plant-specific family of transcription factors. Proceedings of the National Academy of Sciences, 107(42), 18220–18225.
Zhang, Y., Zhao, L., Zhao, J., Li, Y., Wang, J., Guo, R., Gan, S., Liu, C., & Zhang, K. (2017). S5H/DMR6 encodes a salicylic acid 5-hydroxylase that fine-tunes salicylic acid homeostasis. Plant Physiology, 175(3), 1082–1093.
Zhang, Z., Li, Q., Li, Z., Staswick, P. E., Wang, M., Zhu, Y., & He, Z. (2007). Dual regulation role of GH3. 5 in salicylic acid and auxin signaling during *Arabidopsis – Pseudomonas syringae* interaction. Plant Physiology, 145(2), 450–464.
Zhao, Y., Christensen, S. K., Fankhauser, C., Cashman, J. R., Cohen, J. D., Weigel, D., & Chory, J. (2001). A role for flavin monooxygenase-like enzymes in auxin biosynthesis. Science, 291(5502), 306–309.
Zhao, Y., Hull, A. K., Gupta, N. R., Goss, K. A., Alonso, J., Ecker, J. R., Normanly, J., Chory, J., & Celenza, J. L. (2002). Trp-dependent auxin biosynthesis in *Arabidopsis*: Involvement of cytochrome P450s CYP79B2 and CYP79B3. Genes and Development, 16(23), 3100–3112.
Zhou, J. M., Trifa, Y., Silva, H., Pontier, D., Lam, E., Shah, J., & Klessig, D. F. (2000). NPR1 differentially interacts with members of the TGA/OBF family of transcription factors that bind an element of the PR-1 gene required for induction by salicylic acid. Molecular Plant – Microbe Interactions, 13(2), 191–202.
Zhu, S., Gao, F., Cao, X., Chen, M., Ye, G., Wei, C., & Li, Y. (2005). The rice dwarf virus P2 protein interacts with ent-kaurene oxidases *in vivo*, leading to reduced biosynthesis of gibberellins and rice dwarf symptoms. Plant Physiology, 139(4), 1935–1945.
Zolman, B. K., Martinez, N., Millius, A., Adham, A. R., & Bartel, B. (2008). Identification and characterization of *Arabidopsis* indole-3-butyric acid response mutants defective in novel peroxisomal enzymes. Genetics, 180(1), 237–251.
Zolman, B. K., Nyberg, M., & Bartel, B. (2007). IBR3, a novel peroxisomal acyl-CoA dehydrogenase-like protein required for indole-3-butyric acid response. Plant Molecular Biology, 64, 59–72.
Zolman, B. K., Yoder, A., & Bartel, B. (2000). Genetic analysis of indole-3-butyric acid responses in *Arabidopsis thaliana* reveals four mutant classes. Genetics, 156(3), 1323–1337.
Zupan, J. R., & Zambryski, P. (1995). Transfer of T-DNA from *Agrobacterium* to the plant cell. Plant Physiology, 107(4), 1041–1047.