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Towards an integrated molecular understanding of plant hormones∞

Louise Vilain*, Marie-Laure Fauconnier† and Manon Genva†

Laboratory of Chemistry of Natural Molecules, Gembloux Agro-Bio Tech, University of Liège, Gembloux 5030, Belgium
†These authors contributed equally to this work.
*Correspondence: Louise Vilain (louise.vilain@uliege.be)

Louise Vilain

Marie-Laure Fauconnier

ABSTRACT

From seed dormancy to pathogen attacks and senescence, many events in a plant's life are critically controlled by low-molecular-weight signaling molecules, called plant hormones. These master regulators include abscisic acid, auxins, brassinosteroids, cytokinins, ethylene, gibberellins, jasmonates, salicylates, and strigolactones. Years of research have unveiled multiple aspects of their biosynthesis, transport, signaling, functions, and interactions. With this knowledge, complex molecular networks have been established and refined over time in an attempt to explain how plant hormones collectively mediate physiological processes, under a wide range of contexts and at various developmental stages. In this context, we aim to shed light on the global molecular framework of plant hormonal signaling. To do so, we first step back and offer a deconvolution of plant hormonal pathways, by addressing their biosynthesis, transport, and signaling. We then explore how hormonal pathways interfere with each other at a molecular level, mostly through transcriptional regulation and protein–protein interactions. We further discuss directions to fine-tune our fundamental understanding of plant hormonal control, and finally, we highlight how this knowledge can be translated into applied prospects to tackle unprecedented agricultural challenges.

Keywords: auxins, biosynthesis, brassinosteroids, crosstalk, jasmonates, plant hormones, signaling

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INTRODUCTION

Plant hormones are low-molecular-weight signaling compounds that regulate, at very low concentrations (<1 μM), critical aspects of plant growth, development, and responses to external stresses (Yu et al., 2020; Waadt et al., 2022). Nine classes of compounds are considered plant hormones: abscisic acid (ABA) and derivatives, auxins, brassinosteroids (BRs), cytokinins (CKs), ethylene (ET) and precursors, gibberellins (GAs), jasmonates (JAs), salicylates (SAs), and strigolactones (SLs) (Verma et al., 2016). While every family of plant hormones usually revolves around only a few bioactive molecules (Figure 1), each of them encompasses a much broader range of compounds in their metabolism.

Hormonal networks are crucial at every stage in the life of a plant, and hence, understanding how they work is of outstanding significance for both fundamental and applied research. The mechanisms by which plant hormones operate are, nonetheless, far from as simple as originally thought. For a long time indeed, plant hormones were labeled as either stress hormones – JAs, SAs, ABA, ET – or growth promotion hormones – auxins, GAs, CKs, BRs, and SLs (Verma et al., 2016). This notion was challenged by accumulating evidence on crosstalk between plant hormones and on their high context-dependency (Robert-Seilaniantz et al., 2011; Vanstraelen and Benková, 2012). Since then, the number of studies investigating how plant hormones collectively drive changes in various plant species, development stages, and

processes has skyrocketed. With this multitude of novel and multifaceted insights on hormonal (cross-)regulation, it is now well acknowledged that it is not just one hormone controlling one specific event in a plant's life, but all of them communicating and working together to meet the plant's needs (Robert-Seilaniantz et al., 2011; Vanstraelen and Benková, 2012). It is thus becoming imperative to adopt a more comprehensive approach that addresses plant hormones as a whole rather than individually.

In line with this view, we aim here to offer a global molecular unraveling of plant hormonal control. Our review draws on three subsequent questions that successively build complexity in our understanding of plant hormones:

1. What is the fundamental basis of plant hormonal regulation? Because each of the nine plant hormones is primarily governed by its own core pathway, we first detail their biosynthesis, transport, and signaling.

2. What are the molecular mechanisms enabling hormonal crosstalk? Because plant hormonal signaling pathways are highly interconnected rather than operating independently, we then explore their context-dependent points of intersection, focusing on transcriptional regulation and protein–protein interactions.

3. What additional features must be considered for a more comprehensive view of plant hormones? Because hormonal (cross-)regulation is highly specific, this must be integrated into a wider framework that notably encompasses triggering upon distinctive cues, tissue-specificity, crosstalking with other chemical messengers, and interferences from non-plant organisms. These aspects are developed in the final section of our review.

Understanding all these superimposed layers is critical to better understand why and how plant hormones act as a whole to drive context-specific changes in plants. To conclude, we elaborate on the translation of this fundamental knowledge into applied perspectives that could be leveraged in the future to tackle agricultural challenges.

For improved readability, all proteins are thereafter mentioned in their abbreviated names. Their corresponding full names are provided in Table S1.

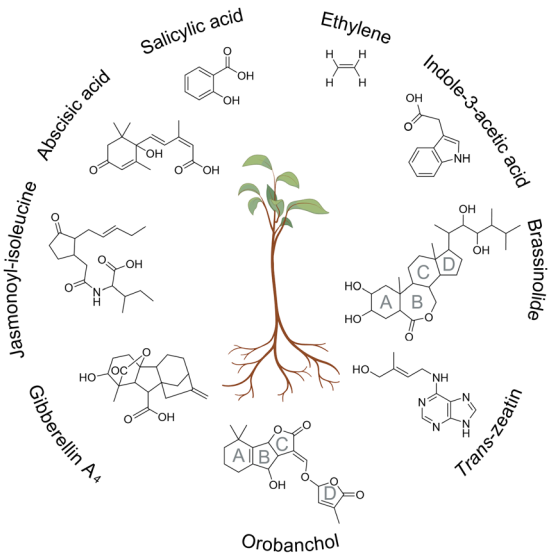

Figure 1. Structure of the most common and bioactive molecule for each class of plant hormones

Created with BioRender: Genva, M. (2026) https://BioRender.com/rob31g0. [Correction added on 15 April 2026, after first online publication: In Figure 1, the structure of indole-3-acetic acid (IAA) was incorrect and one oxygen atom was missing from gibberellin A4; "Gibberelin A4" has been corrected to "Gibberellin A4" and "Orobranchol" to "Orobanchol". Figure 1 has been corrected accordingly.]

THE CORE PATHWAYS OF PLANT HORMONES

Abscisic acid

ABA (Figure 1) is a terpenoid plant hormone most well-known for its control of seed dormancy, stomatal aperture, and stress responses to drought and pathogen attacks (Chen et al., 2020).

Biosynthesis and transport

The biosynthesis of ABA starts in plastids with the mevalonate (MVA) and/or methylerythritol phosphate (MEP) pathways, which produce dimethylallylpyrophosphate (DMAPP) and isopentylpyrophosphate (IPP) (Bajguz and Piotrowska-Niczyporuk, 2023). These compounds are turned into β-carotene, a C40 carotenoid, from which ABA is obtained after a multi-step conversion (Nambara and Marion-Poll, 2005; Chen et al., 2020). In chloroplasts, β-carotene is first converted into zeaxanthin, violaxanthin, and neoxanthin. Violaxanthin and neoxanthin are then processed in chloroplasts by NCEDs, which are rate-limiting enzymes in ABA biosynthesis, to produce the C15 xanthoxin (Ng et al., 2014; Chen et al., 2020). Xanthoxin is finally converted in the cytosol into ABA by the successive action of ABA2 and AAO3 (Nambara and Marion-Poll, 2005; Chen et al., 2020). Further catabolism of ABA is critical for its inactivation, notably by CYP707As enzymes (Chen et al., 2020).

In seeds, ABA biosynthesis mostly occurs in the endosperm and is delivered to the embryo after endosperm export via AtABCG25 and AtABCG31, and embryo import via AtABCG30 and AtABCG40 (Kang et al., 2015). In grown plants, ABA is produced in both roots and shoots, and its biosynthesis localizes more specifically in vascular tissues, especially in phloem companion cells (Kuromori et al., 2014). ABA is then relocated throughout the plant via long-distance transport in the xylem and phloem (Kuromori et al., 2018). In this context, ABA export into vascular tissues is mediated by AtABCG25 and AtDTX50 in shoots and roots (Kuromori et al., 2010; Zhang et al., 2014c), while AtNRT1.2 imports ABA in shoots and roots, together with the dual ABA/GA transporters AtNPF2.12 in roots and AtNPF2.13 in shoots (Kanno et al., 2012; Binenbaum et al., 2023). ABA is then redistributed from vascular tissues to other plant tissues, notably with AtABCG40 importing ABA in guard cells (Kang et al., 2010). It is noteworthy to add, though, that guard cells also

produce ABA (Kuromori et al., 2014, 2018). In addition, other members from the ABCG family also function as ABA transporters in various contexts (Zhang et al., 2023a). Within cells, ABA is stored in vacuoles as an inactive glucose ester, and its vacuolar import seems to be mediated by AtABCC1 and AtABCC2 in guard cells, as well as by AtNPF2.14 in the endodermis (Burla et al., 2013; Binenbaum et al., 2023).

Signalization

Signal transduction of ABA largely relies on reversible protein phosphorylation (Figure 2A), with both nuclear and cytosolic targets downstream of PYR1/PYLs, PP2Cs, and SnRK2s.

Without ABA, the cytosolic PYR1/PYLs receptors are inactive and phosphorylated by TOR (Wang et al., 2018a). This enables PP2Cs to bind and dephosphorylate SnRK2s, thereby immobilizing and inactivating SnRK2s (Umezawa et al., 2009). As a consequence, the cytosolic and nuclear targets of SnRK2s remain unphosphorylated, and ABA signaling is thus blocked.

Under high ABA concentrations, however, PYR1/PYLs sense ABA, and this binding of ABA triggers conformational changes in PYR1/PYLs (Miyakawa et al., 2013), thereby enabling their docking of PP2Cs, like ABI1-2 (Ma et al., 2009; Park et al., 2009; Miyakawa et al., 2013). The binding of PP2Cs by PYR1/PYLs results in the release of SnRK2s (Umezawa et al., 2009), which are thereafter activated either by auto-phosphorylation or after phosphorylation by Ralf-like kinases (Fàbregas et al., 2020; Lin et al., 2021a). Through their kinase domain, SnRK2s then (in)activate their targets, which consist of ion channels and NADPH oxidases in the cytosol, as well as ABA-responsive transcription factors (TFs) in the nucleus (Ng et al., 2014; Jung et al., 2020). These ABA responsive TFs consist of ABI3-5 as well as AREB/ABFs, and their activation induces the expression of ABA-responsive genes through binding to the ABA-responsive element (ABRE; PyACGTGGC) (Himmelbach et al., 2003). It is noteworthy to add that ABA optimal response depends on the presence of at least one ABRE and one coupling element, which may be another ABRE or an element recognized by another TF (Himmelbach et al., 2003; Antoni et al., 2011).

Auxins

Auxins are indole derivatives most well-known for their control of cell expansion, division, and differentiation, making them critical in many plant developmental processes, including embryogenesis, root and leaf patterning, as well as tropic responses (Du et al., 2020). Among auxins, indole-3-acetic acid (IAA; Figure 1) is usually the most active and abundant in plants, although other endogenous and bioactive auxins exist, including 4-chloroindole-3-acetic acid, phenylacetic acid, and indole-3-butyric acid (Simon and Petrášek, 2011).

Biosynthesis and transport

The biosynthesis of IAA is particularly intricate, as five different pathways have been described up to now. In the tryptophan-independent pathway, anthranilates are transformed into indole-3-glycerol phosphate, which is thought to be the precursor of IAA, although precise mechanisms remain to be uncovered (Zhao, 2010). In tryptophan-dependent routes, anthranilates are converted into tryptophan in chloroplasts. IAA is then obtained from tryptophan through four different pathways involving either indole-3-acetaldoxime, indole-3-acetamide, tryptamine, or indole-3-pyruvic acid (Mano and Nemoto, 2012; Tivendale et al., 2014; Kasahara, 2016). Interestingly, the indole-3-acetaldoxime pathway seems limited to Brassicaceae and is also responsible for the production of camalexin and indole glucosinolates (Mano and Nemoto, 2012). On the contrary, the endoplasmic reticulum (ER)-located pathway of indole-3-pyruvic acid seems to be the most conserved IAA biosynthetic route in plants (Zhao, 2012; Blakeslee et al., 2019). In this pathway, tryptophan is transformed by TAAs enzymes into indole-3-pyruvate, which is then turned into IAA after oxidative decarboxylation by YUCs/YUCCA enzymes (Zhao, 2012; Blakeslee et al., 2019). This last step is rate-limiting in tryptophan-dependent IAA biosynthesis (Zhao et al., 2001).

While auxins are synthesized in both roots and shoots, auxin production is highly localized and mainly occurs in apical meristems (Vernoux et al., 2010). Initially, auxins were thought to be predominantly synthesized in shoots, then redistributed in roots through long-distance transport (Zhao, 2010). More recent studies, however, invalidated this view as shoot-derived auxins cannot compensate for the lack of root-derived auxins in various contexts (Chen et al., 2014; Brumos et al., 2018; Matosevich et al., 2020). Thus, auxin distribution in plants largely results from both local biosynthesis and polar transport, which is a unique feature of auxins. In a nutshell, auxins synthesized in a cell are exported through PIN transporters in the apoplast, where they are imported into the next cell either by passive diffusion and/or by active uptake via AUX1/LAX carriers (Luschnig and Friml, 2024; Vanneste et al., 2025). This unidirectional transport continues from adjacent cell to adjacent cell, thereby generating along cells an auxin gradient, of which the rate-limiting factor is auxin export by PINs (Petrášek et al., 2006). While other auxin intercellular transporters from the ABCB and NPF families are known, their roles remain less understood (Hammes and Pedersen, 2024; Vanneste et al., 2025). Plasmodesmata also mediate auxin diffusion between cells in root tips, but the extent of such an involvement remains to be determined (Mellor et al., 2020). Besides cell-to-cell transport, auxins are transported intracellularly to peroxisomes, vacuoles, and ER by various carriers (Zhang et al., 2023a; Hammes and Pedersen, 2024). Whether IAA requires a transporter for translocation in the nucleus, nonetheless, still remains unknown.

Signalization

Interestingly, auxin signaling (Figure 2B) occurs at both intracellular and extracellular levels through different routes (Vanneste et al., 2025). Auxin intracellular signaling is mostly based on the proteasomal degradation of Aux/IAAs following auxin recognition by their TIR1/AFBs receptors

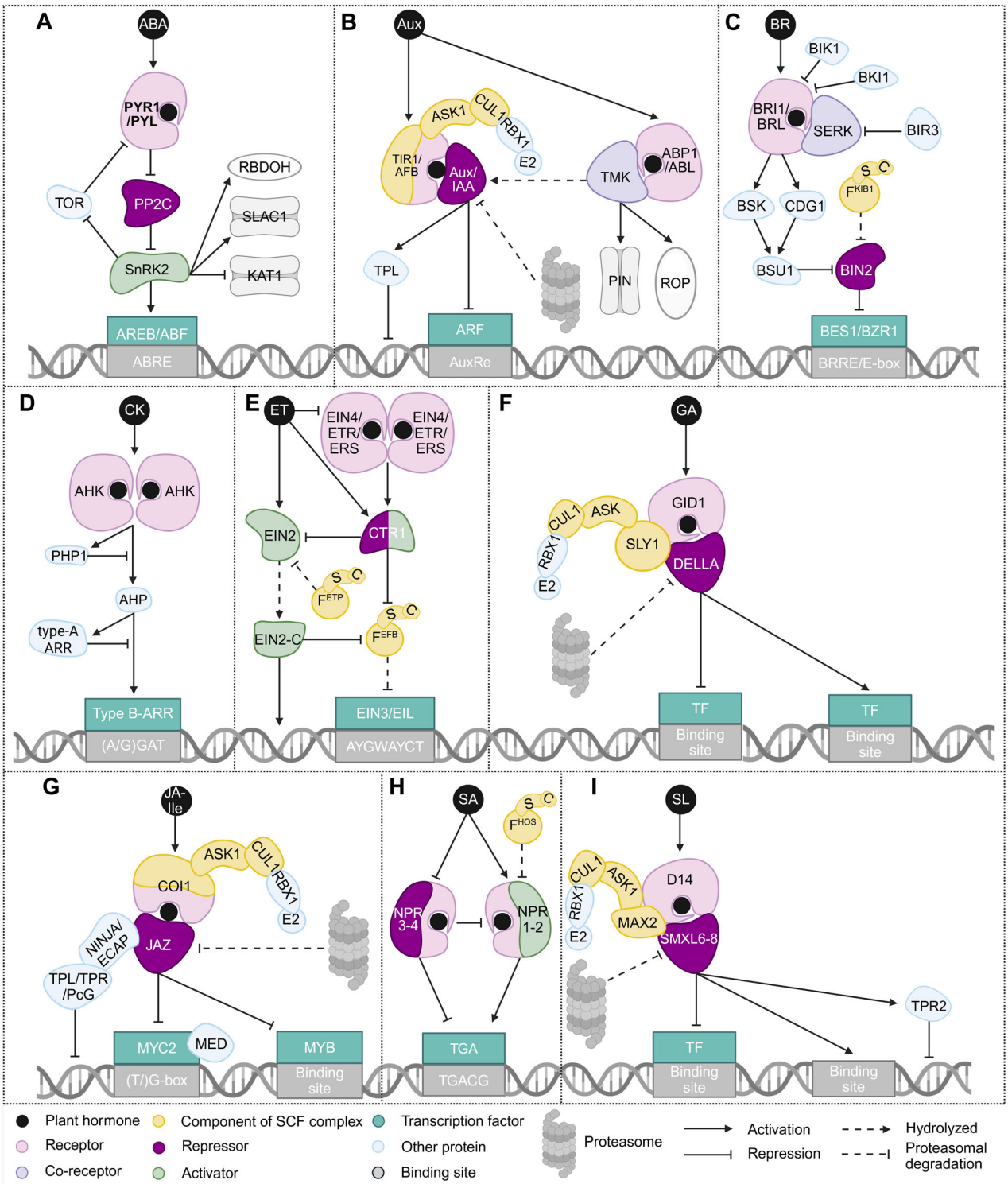

Figure 2. Core signaling pathways of plant hormones

(A) abscisic acid, **(B)** auxins, **(C)** brassinosteroids, **(D)** cytokinins, **(E)** ethylene, **(F)** gibberellins, **(G)** jasmonates, **(H)** salicylates, **(I)** strigolactones. Abbreviations: ABA, abscisic acid; ABRE, ABA-responsive element; Aux, auxins; AuxRE, Aux-responsive element; BR, brassinosteroids; BRRE, BR-responsive element; CK, cytokinins; ET, ethylene; GA, gibberellins; JA-Ile, jasmonoyl-isoleucine; SA, salicylic acid; SCF, SKP1–CUL1–F-box; SL, strigolactones; TFs, transcription factors. Full protein names are provided in Table S1. Created with BioRender: Genva, M. (2026) https://BioRender.com/gpkbkeh.

(Yu et al., 2022a). Besides TIR1/AFBs, two unconventional intracellular auxin receptors exist: ETT/ARF3 is an auxin-responsive TF, which cannot interact with Aux/IAAs and instead directly activates gene expression upon auxin binding (Simonini et al., 2017); meanwhile, SKP2A seems to function similarly to TIR1 but otherwise mostly remains enigmatic (Yu et al., 2022a). In contrast, auxin extracellular signaling rather relies on protein phosphorylation through ABP1 receptor and TMKs co-receptors, eventually converging with intracellular transcriptional signaling (Monzer and Friml, 2025). Intracellular signaling is first discussed here, with a focus on the TIR1/AFBs receptors.

In the absence of auxins within cells, Aux/IAAs bind and thereby repress auxin-responsive TFs, known as ARFs, to block gene regulation (Tiwari et al., 2001; Tiwari et al., 2004). Moreover, they recruit the transcriptional co-repressor TPL to reduce DNA accessibility through histone deacetylases, and repress this way the regulation of auxin-responsive genes (Szemenyei et al., 2008; Nguyen et al., 2020). Aux/IAAs thus globally prevent the transcription of auxin-responsive genes by acting on both ARFs and DNA accessibility.

Under high intracellular auxin concentrations, however, auxins bind their nucleus-localized TIR1/AFBs receptors and favor their interaction with Aux/IAAs through hydrophobic interaction forces (Dharmasiri et al., 2005; Tan et al., 2007). This triggers the polyubiquitination and subsequent proteasomal degradation of Aux/IAA proteins. TIR1/AFBs are indeed part of an SCF complex responsible for the polyubiquitination of Aux/IAAs (Gray et al., 2001). This $SCF^{TIR1/AFB}$ complex is an E3 ubiquitin ligase with three components: TIR1/AFBs F-box proteins enables auxin recognition, CUL1 is a scaffold protein, and ASK1 acts as an adapter between CUL1 and TIR1/AFBs (Gray et al., 2001). CUL1 furthermore recruits RBX1, which promotes the E2-mediated transfer of ubiquitin molecules to Aux-IAAs, leading to their polyubiquitination and further degradation by the 26S proteasome (Lavy and Estelle, 2016; De Roij et al., 2024). As Aux/IAA proteins bind and repress ARFs, their degradation releases ARFs from repression and enables them to regulate the expression of auxin-responsive genes through binding with auxin-responsive elements (AuxRE; TGTCNN) (Li et al., 2023).

Outside of plant cells, auxin signaling occurs through the ABP1 receptor, its ABLs homologs, and their TMKs co-receptors (Monzer and Friml, 2025; Vanneste et al., 2025). While ABP1 and ABLs are mostly found in the ER, a fraction is located in the apoplast near the plasma membrane (PM), where they sense extracellular auxins (Friml et al., 2022; Yu et al., 2023). Upon high auxin levels, auxin binding with ABP1 favors its interaction with PM-located TMKs, which thereby serve as docking proteins for ABP1 (Xu et al., 2014; Yu et al., 2023). Signaling downstream of ABP1-TMKs seemingly occurs via at least three routes. First, ABP1-activated TMKs are able to phosphorylate multiple proteins and thereby regulate their activities, as for AHAs, TAA1, PIN1, and PIN2 notably (Wang et al., 2020a; Lin et al., 2021b; Li et al., 2021c; Friml et al., 2022; Rodriguez et al., 2025; Huang et al., 2026). Second, TMK1 affects transcriptional regulation by phosphorylating and thereby stabilizing IAA32 and IAA34 after cleavage and nucleus translocation of its kinase domain (Gu et al., 2022; Wang et al., 2024c). Third, ABP1-TMKs activate ROP signaling pathways (Xu et al., 2010, 2014). In the absence of extracellular auxins, ABP1 remains in the apoplast and is not connected to the PM, as there is no binding with TMKs, which remain inactive. Most interestingly, ABP1-TMKs signaling seems to be the most conserved auxin signaling route within the plant kingdom (Kuhn et al., 2024).

Brassinosteroids

BRs are sterol-derived master regulators of the cell cycle that are largely involved in vascular differentiation, root growth, and fertility, notably (Planas-Riverola et al., 2019). BRs display a basic skeleton consisting of four ABCD-rings and a side chain (Tang et al., 2016). More than 70 BRs exist naturally and are divided into three classes according to their number of carbon atoms (C27-BRs, C28-BRs, and C29-BRs), with a wide diversity of oxygenated functions in rings A and B and of functional groups in the side chain (Bajguz et al., 2020). Among BRs, the C28-BR brassinolide (Figure 1) is the most active found so far (Tang et al., 2016).

Biosynthesis and transport

The biosynthesis of BRs starts with the obtention of DMAPP and IPP from the MVA and/or the MEP pathways. DMAPP and IPP are transformed into squalene, which is further converted into cycloartenol, the central sterol precursor. Cycloartenol can be turned into campesterol and β-sitosterol, which are the precursors of C28-BRs and C29-BRs, respectively. For C27-BRs, however, cycloartenol must first be reduced into cycloartenal, which is then converted into cholesterol and finally C27-BRs. This is a much-condensed summary of the complex biosynthesis of BRs, which likely occurs in the ER: for more details, we refer to (Bajguz et al., 2020; Zebosi et al., 2024).

BRs are found in all plant tissues, and they are synthesized in shoots and roots. However, they display a relatively low mobility and do not seem to be transported between plant tissues, unlike other plant hormones (Hu et al., 2025). This does nonetheless not imply that BRs are homogenously produced in all plant tissues: instead, the distribution of BRs depends on both local differential biosynthesis and short-distance transport, at least in roots, where it is mostly localized in the elongation zone (Vukašinović et al., 2021). Furthermore, not all cells display the entire enzyme machinery required for full biosynthesis of BRs, and hence, the precursors of BRs seem to undergo cell-to-cell transport for biosynthesis completion (Vukašinović et al., 2021). This cell-to-cell transport, interestingly, seems to rely on plasmodesmata (Wang et al., 2023e). Because BRs are nonetheless perceived in the apoplast, they must be exported from cells after biosynthesis for sensing and signaling: this is mediated at least by AtABCB1 and AtABCB19 (Ying et al., 2024; Wei et al., 2025). Currently, little is known about the intracellular transport of BRs.

Signalization

Reversible protein phosphorylation is key in the signaling pathway of BRs (Figure 2C), in which key players consist of BR1/BRLs receptors, SERKs co-receptors, BIN2 kinase, and BZR1/BES1 TFs (Kim and Russinova, 2020).

In the absence of BRs, BRI1/BRLs receptors are either autoinhibited or inhibited by BKI and BIK1 (Wang et al., 2005; Wang and Chory, 2006; Lin et al., 2013). BRI1/BRLs co-receptors, SERK1/3/4 (He et al., 2007; Wang et al., 2008; Santiago et al., 2013), are moreover kept away through interaction with BIR3 (Imkampe et al., 2017). As a consequence of the inactive state of BRI1/BRLs, the BIN2 kinase is constitutively active and blocks BRs signaling by repressing BRs–responsive TFs, that is, BES1 and BZR1 (Zhao et al., 2002; Kim and Russinova, 2020). Phosphorylation of BES1 and BZR1 by BIN2 indeed results in their inactivation, as it reduces their DNA binding ability, favors their nuclear export/cytoplasmic retention, and enhances their proteasome-dependent degradation (He et al., 2002; Yin et al., 2002; Delesalle et al., 2024).

Upon high BR levels, BRs are perceived outside the cell at the PM by BRI1 and its BRL homologs (Wang et al., 2001; Caño-Delgado et al., 2004; Lozano-Elena and Caño-Delgado, 2019). After binding of BRs, BRI1 phosphorylates BKI1, which then dissociates from the PM (Wang and Chory, 2006). BRs binding also switches the affinity of SERK3 (better known as BAK1) from BIR3 to BRI1: this results in BIR3 release and in heterodimerization of BAK1 with BRI1 (Imkampe et al., 2017). After sequential transphosphorylation between BAK1 and BRI1 (Wang et al., 2008), BRI1 becomes fully activated and phosphorylates two families of receptor-like cytoplasmic kinases PM-located, CDG1 and BSKs (Tang et al., 2008; Kim et al., 2011). In turn, the so-activated CDG1 and BSKs phosphorylate BSU1 (Tang et al., 2008; Kim et al., 2011). BSU1 then dephosphorylates BIN2, thereby inactivating BIN2 and ending its repression of BES1 and BZR1 (Kim et al., 2009; Ryu et al., 2010). BIN2 is further degraded by the 26S proteasome through ubiquitination mediated by the F-box protein KIB1 (Zhu et al., 2017). BES1 and BZR1 are finally activated by PP2As, which dephosphorylate them, enabling their nuclear accumulation and thus regulation of BRs-responsive genes (Tang et al., 2011). Interestingly, both BES1 and BZR1 can induce but also repress the expression of BRs-responsive genes, through binding to an E-box (CANNTG) or a BRs-responsive element (BRRE; CGTG(T/C)G), respectively (Sun et al., 2010; Yu et al., 2011). While gene induction involves cooperation of BES1/BZR1 with another TF, BES1/BZR1 likely form homodimers for gene repression and recruit in addition the transcriptional co-repressor TPL for reduced DNA accessibility of BRs-repressed genes (Oh et al., 2014b; Espinosa-Ruiz et al., 2017; Nolan et al., 2020).

Cytokinins

CKs are heavily involved in cell division and differentiation, and in this view, they notably control the development of vascular tissues and of female gametophytes (Wybouw and De Rybel, 2019; Argueso and Kieber, 2024). Structurally speaking, CKs are adenines with either an aromatic or an isoprenoid group as a side chain at their N^6 terminus. Among isoprenoid CKs, which are by far the most common and abundant CKs (Kamada-Nobusada and Sakakibara, 2009), *trans*-zeatin (Figure 1) is the most well-known bioactive compound.

Biosynthesis and transport

Three key enzymes are involved in the main biosynthetic pathway of *trans*-zeatin: IPTs, CYP735A, and LOGs (Osugi and Sakakibara, 2015; Márquez-López et al., 2019; Zhao et al., 2024b; Sakakibara, 2025). Rate-limiting IPTs use DMAPP mainly from the MEP pathway and adenosine tri/di/monophosphate to produce in plastids isopentenyladenosine-5′-tri/di/monophosphates, which are then hydroxylated in the ER by CYP735A. The resulting products are *trans*-zeatin nucleotides that must be dephosphorylated into *trans*-zeatin riboside 5′-monophosphate and then converted in the cytosol by LOG enzymes into *trans*-zeatin (Osugi and Sakakibara, 2015; Márquez-López et al., 2019; Zhao et al., 2024b; Sakakibara, 2025).

The biosynthesis of *trans*-zeatin, and more generally of *trans*-zeatin-type CKs, is mostly restricted to roots and especially root vascular tissues (Sakakibara, 2021; Zhao et al., 2024b). In contrast, isopentenyl-type CKs can be seemingly synthesized in both shoots and roots, likely with a greater production in shoots (Matsumoto-Kitano et al., 2008). Because of this differential production of CKs in various biosynthetic sites, an uneven distribution of CKs is observed between the phloem and the xylem, which rather contain isopentenyl-type CKs versus *trans*-zeatin-type CKs, respectively (Sakakibara, 2021). In this view, long-distance transport of CKs occurs in both directions through vascular bundles, with AtABCG14 as the main uncovered carrier. AtABCG14 indeed mediates xylem loading of *trans*-zeatin-type CKs, but also of isopentenyl-type CKs (Ko et al., 2014; Zhang et al., 2014a; Zhao et al., 2023). It is furthermore involved in phloem unloading of isopentenyl-type CKs in shoots, and in shoot-to-root transport of isopentenyl-type CKs (Zhao et al., 2021a, 2023). From the apoplast, CKs are imported into cells by AtAZG1-2 in roots, AtPUP1 in various tissues, AtPUP2 in flowers, and AtPUP14 in shoots and roots (Zürcher et al., 2016; Tessi et al., 2021, 2023; Zhang et al., 2023a). In root cells, import of CKs into the ER furthermore seems mediated by AtAZG2 (Tessi et al., 2021), with a suggested additional contribution of AtABCI19-21; the functions of the latter as importers/exporters must, however, still be uncovered (Kim et al., 2020).

Signalization

CKs signaling in plants (Figure 2D) consists of a phosphorylation relay with AHKs receptors, AHPs phosphotransfer proteins, and B-ARRs TFs as main components (Argueso and Kieber, 2024).

Without CKs, AHKs remain inactive, and unphosphorylated AHPs are thus restricted to the cytosol, whereas type B-ARRs

remain inactive in the nucleus: CKs signaling is thereby blocked (Argueso and Kieber, 2024).

When the levels of CKs rise, CKs bind to AHKs that undergo autophosphorylation at a conserved histidine residue (Nishimura et al., 2004); this could occur in the ER and/or at the PM (Romanov et al., 2018; Antoniadi et al., 2020; Kubiasová et al., 2020). This phosphate group is transferred within AHKs from the histidine residue to an aspartyl one, and transferred again to a conserved histidine residue in AHPs, which are phosphotransfer proteins (Suzuki et al., 1998; Tanaka et al., 2004; Hutchison et al., 2006). Once phosphorylated, AHPs move from the cytosol to the nucleus, where they phosphorylate ARRs (Suzuki et al., 1998; Tanaka et al., 2004; Hutchison et al., 2006). This may, however, be hampered by AHP6/PHP1, an AHP protein able to receive the phosphate group but unable to transfer it (Mähönen et al., 2006). AHPs, moreover, phosphorylate ARRs in the nucleus, but while type-B ARRs are functional TFs that bind to target (A/G)GAT motifs to regulate the expression of CK-responsive genes (Brenner et al., 2012), type-A ARRs lack the Myb-like DNA binding domain (D'Agostino et al., 2000; Tsai et al., 2012). Both PHP1 and type-A ARRs are thus considered as repressors of CK signaling, perhaps through phospho-competition with AHPs and type-B ARRs, respectively. Type-A ARRs are interestingly stabilized by CKs, and their transcription is upregulated by type-B ARRs, which creates a negative feedback loop (D'Agostino et al., 2000; To et al., 2008; Tsai et al., 2012).

Ethylene

The IUPAC name of ET is ethene, that is, it is the smallest unsaturated hydrocarbon molecule (C_2H_4; Figure 1). Despite its extremely small size, ET is an important regulator of growth in young leaves, defense in mature leaves, and senescence in old leaves (Dubois et al., 2018). More generally, ET represses leaf growth by inhibiting cell division and expansion (Dubois et al., 2018).

Biosynthesis and transport

The biosynthesis of ET in plants mainly occurs in the cytosol and starts from the amino acid methionine. This methionine is converted by SAMS to *S*-adenosyl-L-methionine, which is turned by ACS into 1-aminocyclopropane-1-carboxylic acid (ACC) and 5′-methylthioadenosine (Booker and DeLong, 2015; Pattyn et al., 2021). While the latter enters the Met/Yang cycle to be recycled back into methionine, ACC is oxidized by ACO, which produces ET (Booker and DeLong, 2015; Pattyn et al., 2021). Conversion of *S*-adenosyl-L-methionine to ACC is largely considered to be the rate-limiting step in ET biosynthesis; nonetheless, this is prone to debate with regard to the ACO-catalyzed step (Houben and Van De Poel, 2019).

The production of ET occurs in both roots and shoots, but a more precise mapping of ET and even of ACC inside plant tissues is nonetheless lacking, mostly due to analytical limitations (Vandenbussche et al., 2012). As for key biosynthetic sites, the expression patterns of *ACS* and *ACO* enzymes in the seedlings of *Arabidopsis thaliana* (L.) Heynh. seemingly differ, especially in shoots (Tsuchisaka and Theologis, 2004; Houben et al., 2026). *ACS* isoforms are globally well found in all plant tissues, with the exception of ACS9, which is unexpressed in seedlings (Tsuchisaka and Theologis, 2004). More precisely, *ACS8* is the single member expressed in the root tip, whereas only *ACS1* and *ACS5* are expressed in the hypocotyl, and *ACS1* is the sole isoform unexpressed in roots (Tsuchisaka and Theologis, 2004). It thus seems on that basis that almost all root and shoot tissues of Arabidopsis seedlings should be able to synthesize ACC, provided the presence of its precursor. In contrast, *ACO* isoforms are much more scarcely expressed, especially in shoots where only *ACO2* is expressed near the phloem (Houben et al., 2026). This implies that while expression patterns of *ACS* and *ACO* mostly coincide in Arabidopsis roots (Tsuchisaka and Theologis, 2004; Houben et al., 2026), this does not seem to be the case in shoots, where *ACO* expression seems very limited, at least under physiological conditions. *ACO* expression does not furthermore seem to be regulated by ET nor ACC, and therefore other cues are suggested to control this in specific contexts (Houben et al., 2026).

As for transport, ET freely diffuses through aqueous environments and lipid membranes as it is a gas (Zhang et al., 2023a). Unlike ET, ACC is not volatile and is known to be transported through vascular bundles, especially the xylem, and within cells (Zhang et al., 2023a). While there are thus likely transporters for ACC import and export, little is known about their existence, except for AtLHT1 and AtLHT2 (Shin et al., 2015; Choi et al., 2019). These two proteins are suggested to act as dual transporters of amino acids and ACC (Shin et al., 2015; Choi et al., 2019), but their precise localization in plant tissues remains to be determined in order to better understand their relevance.

Signalization

An overview of ET signaling is provided in Figure 2E. In the absence of ET and unlike other plant hormones, ET receptors are constitutively active and repress ET signaling by forming a signaling complex with the Raf-like kinase CTR1 (Huang et al., 2003). This physical association activates CTR1, of which the target is EIN2 (Ju et al., 2012). EIN2 phosphorylation by CTR1 prevents translocation of EIN2 to the nucleus and promotes its proteasomal degradation, which is mediated by the F-box proteins ETP1 and ETP2 (Qiao et al., 2009). Because of EIN2 repression, the regulation of ET-responsive genes is blocked as ET-responsive TFs (i.e., EIN3 and EILs) are recognized by the SCF^{EFB1-2} for proteasomal degradation (Guo and Ecker, 2003; Potuschak et al., 2003; Gagne et al., 2004; An et al., 2010; Wang et al., 2022b).

Under high concentrations of ET, the latter binds its AtEIN4, AtERS1-2, and AtETR1-2 ER-located receptors, resulting in their inactivation (Lacey and Binder, 2014). ET binding leads to the dissociation of its receptors with CTR1, resulting in CTR1 inactivation and the end of EIN2

phosphorylation (Bisson and Groth, 2011; Binder, 2020). The non-phosphorylated state of EIN2 facilitates the cleavage of its carboxyl-terminal part by an unknown protease (Ju et al., 2012; Qiao et al., 2012; Wen et al., 2012). The so-released EIN2-C represses the translation of EFB1-2 and thus prevents the degradation of EIN3/EILs in the nucleus (Li et al., 2015; Merchante et al., 2015). EIN2-C is also translocated from the ER to the nucleus as it contains a nuclear localization sequence (Ju et al., 2012; Qiao et al., 2012; Wen et al., 2012). In the nucleus, EIN2-C interacts with the histone-binding protein ENAP1 (Zhang et al., 2016). This association mediates histone acetylation and enhances DNA accessibility to EIN3, thus promoting transcriptional regulation of ET-responsive genes by EIN3 (Zhang et al., 2017). Finally, in addition to EIN2, ET also induces the translocation of CTR1 to the nucleus, where it interacts with EBF1-2 and promotes EIN3 stabilization by inhibiting its degradation (Park et al., 2023). CTR1 thus displays a dual role by negatively but also positively regulating ET signaling in the absence and presence of ET, respectively (Ju et al., 2012; Park et al., 2023). All in all, the presence of ET globally prevents the degradation of its responsive TFs, in addition to improving their access to ET-responsive genes.

Interestingly, ET-regulated transcriptional changes occur in two sequential phases. Indeed, EIN3/EILs bind as homodimers to the primary ethylene-responsive element (PERE, also known as EIN3 binding site; AYGWAYCT) (Kosugi, 2000; Yamasaki et al., 2005) to regulate the expression of primary ET-responsive genes, including ERFs, which are secondary ET-responsive TFs (Solano et al., 1998). EIN3/EILs turn on this way the expression of the ERFs, which bind to the GCC-box (AGCCGCC) of secondary ET-target genes (Solano et al., 1998; Bakshi et al., 2015).

Gibberellins

GAs form a wide group of tetracyclic diterpenoid acids with over 130 characterized compounds (MacMillan, 2001; Sponsel and Hedden, 2010). In plants, they promote cell division, elongation, and expansion in addition to inducing flowering and controlling seed germination (Shani et al., 2024). Of note, the most used nomenclature for GAs consists of a numbering system by order of discovery and structure characterization (Hedden and Sponsel, 2015).

Biosynthesis and transport

The biosynthesis of GAs starts with the plastidial MEP and/or cytosolic MVA pathways, where a GGPPS enzyme converts IPP into *trans*-geranylgeranyl diphosphate, which is then turned into *ent*-kaurene by CPS and KS through a two-step cyclization in plastids (Zi et al., 2014; Hedden, 2020). In the ER, kaurenoic acid is obtained from *ent*-kaurene by KO and is then hydroxylated sequentially three times by KAO to produce GA_{12}, the C20 precursor of all other GAs (Zi et al., 2014; Hedden, 2020). Mainly two families of enzymes, GA20Ox and GA3Ox, then convert in the cytosol GA_{12} into a wide range of (in)active GAs. While gibberellic acid (GA_3) is bioactive and doubtless the most famous among all GAs, it seems to be a minor compound in plants, where GA_1 and GA_4 (Figure 1) are the main bioactive GAs depending on plant species (Yamaguchi, 2008; Sponsel and Hedden, 2010).

Although GAs are biosynthesized in both roots and shoots, insights into their distribution are largely restricted to roots. GAs are known to accumulate especially in root endodermal elongating cells (Shani et al., 2013; Barker et al., 2021). More precisely, a longitudinal gradient of active GAs seems to occur within the root tip elongation zone, ranging from lowest in the late division zone to highest in the late elongation zone (Rizza et al., 2017). This gradient results from several factors according to modeling: shoot-to-root transport, expression of biosynthetic enzymes, and plasmodesmata (Kiradjiev et al., 2025). In a nutshell, GA_{12} is supposedly mostly delivered in roots from the shoots via the phloem, and is additionally locally produced in the root quiescent center. This GA_{12} is thought to largely diffuse from the quiescent center zone to the elongation zone, thanks to the inactivity of GA20Ox and GA3Ox in the quiescent center zone (Kiradjiev et al., 2025). Cell-to-cell transport of GAs for gradient establishment is, moreover, predicted to be more critically mediated via plasmodesmata rather than NPF carriers (Kiradjiev et al., 2025). Interestingly, these NPF carriers mediate dual transport of ABA and GAs: AtNPF2.12-13 are involved in GA_{12} import for shoot-to-root translocation (Binenbaum et al., 2023), AtNPF3 imports ABA and GA into root endodermal cells (Tal et al., 2016), where they are imported by AtNPF2.14 into vacuoles (Binenbaum et al., 2023). AtSWEET13-14 are also suggested to mediate cell import of GAs in vascular tissues of roots and shoots, as well as in flowers (Kanno et al., 2016). Exporters of GAs remain, in contrast, undeciphered up to date. Besides, while shoot-to-root translocation of the inactive GA_{12} is mostly discussed, transport of GA_{12} from roots to shoots also occurs (Regnault et al., 2015; Camut et al., 2019).

Signalization

GAs signalization (Figure 2F) relies on the proteasomal degradation of DELLA proteins upon GAs sensing by the nucleus-located GID1 receptor (Shani et al., 2024). DELLA proteins are indeed key transcriptional regulators of GAs signaling through direct protein–protein interactions (Van De Velde et al., 2017; Shani et al., 2024).

In the absence of GAs, their GID1 receptor is inactive, and GAs signaling is repressed by DELLA proteins. The presence of DELLA proteins positively and negatively affects the expression of many genes as they function as transcriptional co-activators of many TFs, notably GAF1 and FLC, but also as co-repressors for other TFs, like WRKY75 (Fukazawa et al., 2015, 2021; Li et al., 2016b; Zhang et al., 2018a). This repression by DELLAs likely involves the binding of DELLA to both a TF and H2A for complex stabilization (Huang et al., 2023a). Furthermore, DELLA proteins can also sequestrate and subsequently induce the degradation of some TFs, as shown for PIFs (Li et al., 2016a). DELLAs can additionally

interact with chromatin-remodeling complexes, thereby likely affecting DNA accessibility (Sarnowska et al., 2013; Park et al., 2017). Through these numerous protein–protein interactions, DELLA proteins are thus able to upregulate but also to downregulate target genes.

Upon binding of GAs, however, GID1 undergoes conformational changes (Ueguchi-Tanaka et al., 2005; Nakajima et al., 2006; Griffiths et al., 2007; Shimada et al., 2008). This creates a hydrophobic surface allowing the binding of DELLA proteins to GID1, therefore promoting the formation of a stable GA-GID1-DELLA complex (Murase et al., 2008; Shimada et al., 2008). A conformational change in DELLA proteins seems to occur upon GID1 binding, which triggers the recognition of DELLA proteins by SLY1 (also known as GID2) (Dill et al., 2004; Griffiths et al., 2007; Ariizumi et al., 2011; Xiang et al., 2018). As SLY1 is an F-box protein part of an SCF complex along with ASK1-4/ASK11 and CUL1, its interaction with DELLA proteins results in the polyubiquitination and subsequent degradation of DELLA proteins by the 26S proteasome (McGinnis et al., 2003; Dill et al., 2004; Fu et al., 2004; Ariizumi et al., 2011; Islam et al., 2025). This ends the repression of GAs signaling, as the degradation of DELLA proteins upon GA perception cancels their previous interactions and results in the dissociation of their transcriptional complexes.

Jasmonates

JAs are lipid-derived hormones that are most well-known in plant defense, notably in the fight against necrotrophic pathogens and insects (Acosta and Farmer, 2010).

Biosynthesis and transport

JAs are part of the oxylipin family as they are oxidized derivatives of glycolipids (Wasternack and Feussner, 2018). More precisely, JAs derive from the hydrolysis of plastidial galactolipids into linolenic acid (C18:3) and hexadecatrienoic acid (C16:3) (Acosta and Farmer, 2010). These polyunsaturated fatty acids are oxidized by LOX enzymes into hydroperoxides, which are turned into (dinor-)12-oxo-phytodienoic acid after the successive action of AOS and AOC. (Dinor-)12-oxo-phytodienoic is then transported from plastids to peroxisomes for conversion into jasmonic acid by OPR3, and two to three rounds of β-oxidation (Acosta and Farmer, 2010). In an alternative route and to a much lesser extent, dinor-12-oxo-phytodienoic acid is turned in peroxisomes into tetranor-oxophytodienoic acid, then 4,5-didehydro-jasmonic acid, which is exported to the cytosol for OPR2-mediated jasmonic acid production (Chini et al., 2018). Jasmonic acid can then be metabolized into a wide range of (in)active derivatives (Wasternack and Strnad, 2016). Of note, while jasmonic acid is by far the most well-known among JAs, it is not bioactive *per se* and instead, its conjugated form to isoleucine (jasmonoyl-isoleucine; JA-Ile; Figure 1) is the most bioactive; this conjugation is predominantly catalyzed by JAR1 (Staswick and Tiryaki, 2004; Fonseca et al., 2009; Wasternack and Strnad, 2016). Furthermore, in Arabidopsis notably, the fatty acyl chains of galactolipids can also be directly oxidized by the LOX2 isoform and further processed by AOS and AOC: this produces esterified jasmonates like the so-called arabidopsides (Glauser et al., 2009). Whether or not hydrolysis of these esterified jasmonates constitutes an alternative biosynthesis pathway for the production of (dinor-)12-oxo-phytodienoic acid remains to be clarified (Genva et al., 2019).

Enzymes responsible for the biosynthesis of JAs are predominantly found in shoots (Zimmermann et al., 2004). LOXs, AOS, and AOC1-2 are indeed broadly expressed in all shoot tissues of Arabidopsis (Stenzel et al., 2003, 2012; Chauvin et al., 2016). In contrast, OPR3 is mainly expressed in leaf veins, especially in phloem cells (Li et al., 2013). All shoot tissues should thus be able to synthesize (dinor-)12-oxo-phytodienoic acid, whereas production of jasmonic acid and hence of its derivatives seems likely restricted to vascular sites. Consistent with this, levels of 12-oxo-phytodienoic acid, jasmonic acid, and JA-Ile are higher in primary veins than in surrounding tissues (Morin et al., 2023). In roots, expression levels of biosynthetic enzymes are weak, and hence, JA content is much lower than in shoots (Glauser et al., 2008; Grebner et al., 2013). More precise localization of JA-Ile within the root apical meristem of Arabidopsis is pinpointed in the division zone, particularly in its epidermis, ground tissues, and vascular tissues (Larrieu et al., 2015). Strikingly, despite low expression levels of other biosynthetic enzymes in roots, JAR1 seems relatively highly active in roots (Acosta et al., 2013). This is likely explained by the fact that, in addition to root-derived JAs, JA-Ile precursors seem to be translocated from shoots to roots via the phloem, especially 12-oxo-phytodienoic acid (Schulze et al., 2019). Up to date nonetheless, transporters involved in this shoot-to-root transport remain unknown, although AtJAT3 and AtJAT4 are likely candidates (Li et al., 2020d). In another context, leaf-to-leaf translocation of JAs is mediated by several carriers: AtJAT1 is responsible for cell export of jasmonic acid, whereas AtJAT3 and AtJAT4 are cell importers of JAs with a higher affinity for jasmonic acid than for JA-Ile (Li et al., 2017a, 2020d). AtJAT1 furthermore mediates the entry of JA-Ile in the nucleus (Li et al., 2017a). Besides, several carriers mediate the intracellular transport of 12-oxo-phytodienoic acid, and the volatile methyl ester of jasmonic acid can diffuse as a gas in plant cells (Zhang et al., 2023a).

Signalization

JA-Ile signaling (Figure 2G) is in many ways similar to that of auxins (Pérez and Goossens, 2013; Williams et al., 2019).

Without JA-Ile, its COI1 nucleus-located receptor remains inactive, whereas JAZ proteins rule and repress its signaling by interacting with JA-Ile-responsive TFs (Chini et al., 2009). These consist of members from the MYC and MYB families, with MYC2 considered as the master regulator of JA transcriptional responses (Chini et al., 2016). In particular, the binding of JAZ proteins with MYCs triggers conformational changes in MYCs, resulting in competition between JAZ

proteins and their transcriptional mediator complex for interaction with MYCs (Zhang et al., 2015; Zhai et al., 2020). JAZ proteins also repress gene transcription by reducing DNA accessibility for TFs. This is achieved through the recruitment of TPL, TPRs, and PcG proteins, which must be mediated for most JAZ proteins by NINJA and by ECAP proteins (Pauwels et al., 2010; Shyu et al., 2012; Li et al., 2020a, 2021a; Zhou et al., 2023c).

Upon high JA-Ile levels, JA-Ile binds to its nucleus-localized COI1 receptor. As for auxins with TIR1/AFBs, JA-Ile does not induce conformational change in its nucleus-localized COI1 receptor and rather brings together COI1 and JAZ proteins (Katsir et al., 2008; Yan et al., 2009; Sheard et al., 2010). Because COI1 is an F-box protein recognizing JAZ proteins, their physical interaction triggers the degradation of the latter (Thines et al., 2007). Together with RBX1 and E2, the SCF^{COI1} indeed mediates the polyubiquitination of JAZ proteins and thus marks them for proteasomal degradation (Chini et al., 2007; Thines et al., 2007). As JAZ proteins repress JA-Ile signaling (Chini et al., 2007), this allows the expression of JA-responsive genes to be regulated. Indeed, the degradation of JAZ proteins enables the release of JA-responsive TFs (Chini et al., 2016). Along with their co-activator Mediator for MYCs (Zhai et al., 2020), these TFs promote the transcription of JA-responsive genes via their binding sites. The latter consists of a (T/)G-box (CA(C/T)GT(G/T)) for MYCs and of an MYB-core binding site ((C/T)NGTT(G/T)) for MYBs (Prouse and Campbell, 2012; Kazan and Manners, 2013).

Salicylates

SA (Figure 1) is a phenolic plant hormone largely associated with the so-called growth-immunity tradeoff, as it generally suppresses growth and activates immunity (Van Butselaar and Van Den Ackerveken, 2020). Additionally, SA is a key player in the induction of systemic acquired resistance (Hartmann and Zeier, 2019).

Biosynthesis and transport

SA production can occur via two biosynthetic routes, that is, the ICS versus the PAL pathways. In the ICS pathway, chorismate is converted into isochorismate by the plastidial AtICS1-2 (Lefevere et al., 2020; Peng et al., 2021; Ullah et al., 2023). In the cytosol, AtPBS3 then turns isochorismate into isochorismoyl-9-glutamate (Rekhter et al., 2019; Torrens-Spence et al., 2019), which is broken down into SA. This process can occur spontaneously but is enhanced by AtEPS1 (Torrens-Spence et al., 2019). While the ICS pathway was the first fully identified for SA production and is by far the most important SA biosynthetic route in Arabidopsis (Garcion et al., 2008), it seems to be relevant exclusively in plant species belonging to the Brassicales order (Hong et al., 2025). Although minor in Arabidopsis, the PAL pathway seems much more conserved for SA biosynthesis in plants (Ullah et al., 2023; Wu et al., 2023). In this route, phenylalanine is converted by cytoplasmic PALs into *trans*-cinnamic acid, which is exported to peroxisomes for β-oxidation. There, *trans*-cinnamic acid is turned successively into cinnamoyl-coenzyme A, benzoylacetyl-coenzyme A, and finally benzoyl-coenzyme A after sequential action of OSD1/CNLs, AIM1/CHD, and KATs (Xu et al., 2017; Kotera et al., 2023; Wang et al., 2024d; 2024f; Zhu et al., 2025). OSD2/BEBT-mediated addition of benzyl alcohol to benzoyl-coenzyme A produces benzylbenzoate, which is then oxidized in the ER by OSD3/BBH into benzyl salicylate (Liu et al., 2025; Zhu et al., 2025; Wang et al., 2025a; Ma et al., 2025b). Finally, hydrolysis of benzyl salicylate by BSE/OSD4 leads to SA formation in the cytosol (Liu et al., 2025; Zhu et al., 2025; Wang et al., 2025a; Ma et al., 2025b).

Generally speaking, SA seems to be mostly produced in shoots, as best observed in rice (*Oryza sativa* L.) (Lebeis et al., 2015; Wang et al., 2024f). Other than that, insights on SA production sites and distribution are scarce. This is complexified by the fact that not only do biosynthetic pathways differ between plant species, but also that basal SA levels widely vary depending on plant species. Now that the two routes for SA production in plants are elucidated, there is a need to better apprehend their distribution and localization in plant species, organs, and tissues. Similarly, little data on SA transport is available. The inactive methyl ester of SA is known to undergo long-distance transport in the phloem for leaf-to-leaf translocation (Park et al., 2007), and given its volatile nature, it is also an airborne signal (Gong et al., 2023). Long-distance transport of SA is otherwise suggested to be perhaps rather limited, and instead, *N*-hydroxypipecolic acid is proposed to act as a mobile signal inducing SA biosynthesis in distant leaf tissues, at least in the context of systemic acquired resistance (Hartmann and Zeier, 2019). Within cells, SA is exported from chloroplasts to the cytosol through the EDS5 transporter (Serrano et al., 2013); this is the only unveiled SA transporter up to date, with no carrier identified yet in the involvement of long-distance or cell-to-cell transport of (methyl ester) SA.

Signalization

Unlike other plant hormones, SA can bind a multitude of proteins, collectively known as SA-binding proteins, including metabolic enzymes, redox regulators, and transcriptional regulators (reviewed in (Pokotylo et al., 2019; Ding et al., 2023)). For instance, more than 100 proteins have been reported in Arabidopsis as candidates for binding SA (Manohar et al., 2015). How SA binds its numerous targets is an outstanding question, which has only very recently started to be addressed. An even more important yet little explored question is how SA perception by SA-binding proteins affects them and induces downstream changes in plant growth and immunity (Pokotylo et al., 2019, 2020; Wang et al., 2020c). Here, we focus on SA perception and downstream signaling by NPRs (Figure 2H), which are responsible for SA-dependent transcriptional regulation (Chen et al., 2021; Zhou et al., 2023b). These NPRs all display distinct SA-binding affinities (Fu et al., 2012; Wu et al., 2012; Manohar et al., 2015; Castelló et al., 2018), with the SA-binding site of NPR4

recently characterized (Wang et al., 2020c). As NPRs lack a DNA-binding domain, they regulate gene expression by acting as transcriptional cofactors of SA-responsive TFs, known as TGAs (Zhang et al., 1999, 2006; Després et al., 2000; Zhou et al., 2000; Tomaž et al., 2022). However, while NPR1 and NPR2 appear to be positive regulators of SA signaling, NPR3 and NPR4 seem to repress it (Zhang et al., 2006; Castelló et al., 2018; Ding et al., 2018). The current understanding of this discrepancy is explained below.

In the absence of SA, the nucleus-localized NPR3 and NPR4 interact with TGAs to sequestrate them, thus preventing SA transcriptional responses (Ding et al., 2018). NPR1 is moreover targeted by SCF^{HOS15} for proteasomal degradation in the nucleus (Spoel et al., 2009; Skelly et al., 2019; Shen et al., 2020), and this may be mediated through the possible function of NPR3 and NPR4 as adapters for CUL3 (Fu et al., 2012). In contrast to NPR1, NPR3 and NPR4 are protected from proteasomal degradation by UBP12 and UBP13 (Zhou et al., 2023a).

Upon higher levels of SA, however, SA binds its receptors, resulting in the inactivation of NPR3-4 and the activation of NPR1 (Ding et al., 2018; Kumar et al., 2022). High concentrations of SA, moreover, induce the monomerization of NPR1, which is translocated from the cytosol to the nucleus (Kinkema et al., 2000; Mou et al., 2003). Once in the nucleus, NPR1 is ubiquitinated by CUL3 to further enhance its activity as a transcriptional coactivator (Skelly et al., 2019). NPR1 interacts with TGAs, which regulate transcription of SA-responsive genes by binding to the TGACG motif (Zhang et al., 1999; Zhou et al., 2000; Thibaud-Nissen et al., 2006), to enhance their transcriptional activity and hence promote SA responses (Després et al., 2000; Zhou et al., 2000; Skelly et al., 2019; Kumar et al., 2022). Upregulation of gene expression is also promoted by increasing DNA accessibility through the addition of HACs to the NPR1-TGA complex (Jin et al., 2018).

Strigolactones

SLs are carotenoid derivatives that are the most recent addition to the hub of plant hormones (Gomez-Roldan et al., 2008; Umehara et al., 2008). Besides their functions as seed germination inducers in parasitic plants and as chemical messengers between plants and arbuscular mycorrhizal fungi, SLs control various endogenous plant processes and most famously shoot branching (Aquino et al., 2021; Dun et al., 2023).

Biosynthesis and transport

The precursor of SLs is β-carotene, which is obtained from DMAPP and IPP. More specifically, *trans*-β-carotene is transformed into 9-*cis*-β-carotene and then into the first SL-like compound—carlactone—by the subsequent action of three plastid-localized enzymes, that is, D27, CCD7, and CCD8 (Auldridge et al., 2006; Lin et al., 2009; Alder et al., 2012; Abe et al., 2014). MAX1, suggested to localize in the ER, then converts carlactone into carlactonoic acid (Abe et al., 2014; Zhang et al., 2014d), and from there, the biosynthetic pathway of SLs seems to diverge into the so-called canonical SLs and non-canonical SLs. Canonical SLs possess a tricyclic lactone structure (called the ABC ring system) connected to a butenolide D-ring via an enol-ether bridge (Nomura et al., 2024). They seem to be produced by various cytochrome P450 monooxygenases and are further divided into strigol-type SLs and orobanchol-type SLs, depending on the orientation of the C-ring (Yoneyama and Brewer, 2021). Non-canonical SLs lack the A-, B-, and/or C-ring but still display the characteristic D-ring attached to the enol–ether group. They are thought to derive from methylation of carlactonoic acid into the bioactive methyl carlactonoate (Abe et al., 2014; Yoneyama and Brewer, 2021). Up to date, more than 30 SLs have been characterized with a plant species-dependent structural diversity, the most common being orobanchol (Figure 1; Nomura et al., 2024).

While SLs are synthesized in both roots and shoots, their levels are much lower in shoots than in roots; this globally seems consistent with expression levels of biosynthetic enzymes (Kameoka and Kyozuka, 2018). Enzymes responsible for carlactone production (D27, CCD7, and CCD8) are mostly expressed in the vascular tissues, and the expression levels of CCD7 and CCD8 are higher in roots, contrary to D27 (Booker et al., 2004; Auldridge et al., 2006; Lin et al., 2009; Waters et al., 2012). Similarly, MAX1 is also expressed in the shoot and root vasculature (Booker et al., 2005). A more precise localization of all these enzymes is, however, necessary to determine whether the expression pattern of MAX1 fully overlaps with that of CCD7, CCD8, and D27. It indeed seems that in roots, AtMAX1 is rather expressed in the meristem differentiation zone, and AtMAX4 (corresponding to OsCCD8) is instead strongly expressed in the root tip (Sorefan et al., 2003; Booker et al., 2005), thus suggesting short-distance transport of SLs. Furthermore, although shoot-derived SLs are generally sufficient for proper shoot development (Kameoka and Kyozuka, 2018), root-derived SLs are also known to be translocated in a root-to-shoot manner (Kohlen et al., 2011). Which SLs are transported and how this transport occurs is not fully understood yet. In *Petunia hybrida* and *Petunia axillaris*, an ABCG transporter known as PDR1 is able to export SLs from root cells (Kretzschmar et al., 2012; Sasse et al., 2015). In petunia lateral roots, PDR1 is suggested to enable exudation of SLs (Kretzschmar et al., 2012; Sasse et al., 2015), similarly to SbSLT1-2 in *Sorghum bicolor* (L.) Moench (Shi et al., 2025). In the root tip, PDR1 also seems involved in the cortical transport of SLs for root-to-shoot transport (Kretzschmar et al., 2012; Sasse et al., 2015). The possible additional role of the xylem in long-distance transport is still under debate (Kohlen et al., 2011; Kameoka and Kyozuka, 2018). Other transporters of SLs, whether intracellular or intercellular, remain to be discovered.

Signalization

Research on the signaling of SLs (Figure 2I) has revealed many similarities with GAs signaling (Wallner et al., 2016).

Core components consist of the receptor D14, the transcriptional regulators SMXL6-8 (known as D53 in rice), and the F-box protein MAX2 (known as D3 in rice) (Bennett et al., 2016).

In the absence of SLs, their D14 receptor is inactive, and SMXL6-8 represses SLs signaling by interacting with TPR2, which reduces DNA accessibility through histone deacetylases (Wang et al., 2015; Ma et al., 2017). They also interact with TFs like TB1 and IPA1, thereby sequestrating them and preventing their transcriptional activity (Song et al., 2017; Wang et al., 2020b). Finally and most interestingly, SMXL6-8/D53 can directly bind DNA and thus also acts as a TF, notably downregulating this way its own expression and forming a negative feedback loop (Wang et al., 2020b).

Under high levels of SLs, however, perception of SLs activates their D14 receptor and promotes the formation of a D14-SMXL6-8 complex, triggering recognition of the latter by MAX2 (Zhou et al., 2013; Bennett et al., 2016). The SCF^{MAX2} mediates polyubiquitination of SMXL6-8, resulting in the proteasomal degradation of SMXL6-8, and this enables signaling of SLs (Tal et al., 2022; Liu et al., 2023b).

A puzzling element in this signaling resides in the perception of SLs. There is indeed currently little insight into which SL is the ligand of D14, except that the D-ring seems to be a structural requirement for bioactivity (Dun et al., 2023). Furthermore, D14 not only perceives but also hydrolyzes SLs: the receptor thus destroys its ligand (Hamiaux et al., 2012; Chevalier et al., 2014; Yao et al., 2016). Up to date and despite intense research on this topic (Hamiaux et al., 2012; Nakamura et al., 2013; Zhao et al., 2013, 2015; De Saint Germain et al., 2016; Yao et al., 2016; Carlsson et al., 2018; Shabek et al., 2018; Seto et al., 2019; Arellano-Saab et al., 2023; Hu et al., 2024), whether SL hydrolysis by D14 is a requirement for its activation or not has not been consensually answered yet (Bürger and Chory, 2020; Machin et al., 2020).

THE MOLECULAR INTERPLAY BETWEEN PLANT HORMONES

While plant hormones were discovered and investigated individually at first, their networks later appeared to be in fact largely interconnected (Robert-Seilaniantz et al., 2011; Vanstraelen and Benková, 2012). Rather than acting on their own, plant hormones work indeed together and can negatively and/or positively impact each other. For instance, GA and ABA antagonistically regulate seed germination, while SA–JA antagonism and JA–ET protagonism drive plant immunity (Robert-Seilaniantz et al., 2011; Shu et al., 2016; Verma et al., 2016). In this context, one question is critical: how do plant hormonal pathways crosstalk at a molecular level? Accumulating evidence underlines the complexity behind this question as plant hormones are strongly suggested to interact with each other at multiple levels and via numerous mechanisms. While we cannot highlight exhaustively all hormonal interactions described in the literature, we emphasize using case studies the many ways plant hormones cross-regulate each other directly, but also in more intricate manners involving intermediary proteins. Generally speaking, hormones largely crosstalk through direct transcriptional regulation and direct protein–protein interactions. In the first case, this enables them to regulate the expression of key proteins for their respective production, localization, and sensing in specific plant tissues and cells. In the second case, they affect the whole cascade downstream of the targeted protein, which can be either promoted or repressed upon binding. Such interactions overall have a wide range of outcomes and can result in the modification of gene expression or in protein activation, repression, stabilization, or degradation, through binding and/or post-translational modifications. The same protein can moreover be regulated both at transcriptional and post-translational levels; this is notably the case of PIN proteins that are targeted by many hormones (Semeradova et al., 2020). As auxin production is indeed extremely local, their export into auxin-sensing cells is critical and rate-limiting (Petrášek et al., 2006; Brumos et al., 2018; Blakeslee et al., 2019), resulting in PINs being a staple for hormones to interfere in auxin polar gradients (Semeradova et al., 2020).

Globally, the fact that plant hormones are able to control their respective spatial accumulation, distribution, and action through a multi-leveled intricate crosstalk deeply affects plant physiology, as discussed below.

Crosstalk through direct transcriptional regulation

Plant hormones can (in)activate each other by acting through their responsive TFs on the transcription of genes involved in the biosynthesis, transport, and signaling of other plant hormones. This is illustrated here mainly with auxins-CKs crosstalk, which is critical notably for the specification of many cell types (Figure 3; Chandler and Werr, 2015).

Notably, CKs can exert transcriptional control over the biosynthesis of auxins. In response to light, CKs promote in aerial tissues the biosynthesis of auxins through the upregulation of *TAA1* by ARR1 in concert with ARR12 (Yan et al., 2017). This protagonism *in fine* enables cotyledon opening and expansion, seemingly through auxin-mediated gene induction of *SAUR50* and *SAUR65* (Sun et al., 2016; Yan et al., 2017). Promotion of *TAA1* expression by ARR1, interestingly, also occurs in collaboration with SPT during gynoecium development of Arabidopsis (Figure 3A; Reyes-Olalde et al., 2017). On the contrary, CKs prevent local auxin biosynthesis during shoot regeneration by repressing the expression of *YUC1 and YUC4*; this is mediated by ARR1, ARR10, and ARR12, perhaps in collaboration with another TF (Meng et al., 2017). In this case, antagonism between CKs and auxins is critical for proper *WUS* expression and thus determination of the shoot meristem (Meng et al., 2017). This repression of auxin biosynthesis remains, however, very local: outside this *WUS*-expressing region with high CKs activity, auxin signaling is strong and prevents CKs accumulation through the ARF3-mediated inhibition of *IPT5* (Cheng et al., 2012).

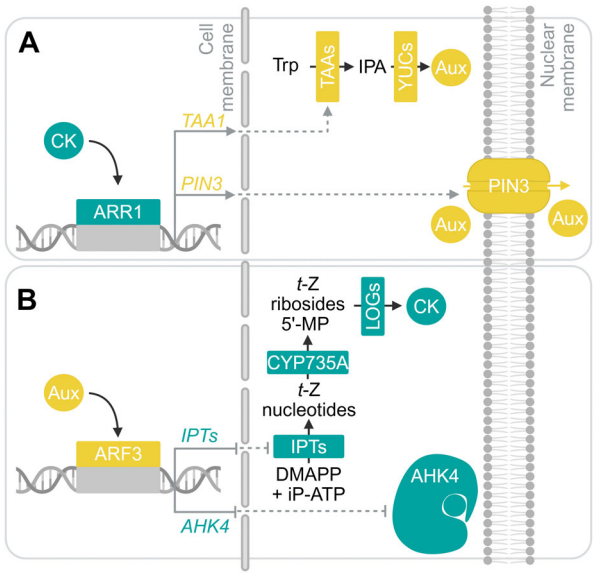

Figure 3. The mediation of plant hormonal crosstalk by control of the transcriptional regulation of hormonal biosynthesis, transport, and signaling, illustrated with case studies on auxins-cytokinins crosstalk

(A) During gynoecium development, cytokinins activate auxins through ARR1-driven gene upregulation of *TAA1* and *PIN3*, that is, auxin-related biosynthetic enzyme and transporter (Reyes-Olalde et al., 2017). **(B)** During floral determinacy, auxins antagonize cytokinins through ARF3, which directly represses the expression of *IPTs* and *AHK4*, that is, cytokinin-related biosynthetic enzymes and receptor (Zhang et al., 2018b). Full gray arrows indicate the induction of gene transcription, and dotted gray arrows showcase the resulting protein. Abbreviations: AHK4, ARABIDOPSIS HIS KINASE4; ARF3, AUXIN RESPONSE FACTOR3; ARR1, ARABIDOPSIS RESPONSE REGULATOR1; Aux, auxins; CK, cytokinins; CYP735A, CYTOCHROME P450 FAMILY 735 SUBFAMILY A; DMAPP, dimethylallylpyrophosphate; IPA, indole-3-pyruvic acid; iP-ATP, isopentenyl-adenosine triphosphate; IPT, ISOPENTENYL TRANSFERASE; LOG, LONELY GUY; MP, monophosphate; PIN3, PIN-FORMED3; TAA, TRYPTOPHAN AMINOTRANSFERASE; Trp, tryptophan; *t*-Z, *trans*-zeatin. Created with BioRender: Genva, M. (2026) https://BioRender.com/padstme.

This two-way negative crosstalk thus affects both auxin and CKs biosynthesis, resulting in a spatial distribution of auxins and CKs within shoot meristems for regeneration (Cheng et al., 2012; Meng et al., 2017). Repression of CKs biosynthesis by auxins also occurs during early flower development, with ARF3 (in)directly inhibiting the expression of several *IPTs* and *LOGs* genes (Figure 3B; Zhang et al., 2018b). This likely ends *WUS* expression, which is normally induced by CKs, and hence floral meristem determinacy is enabled (Zhang et al., 2018b). Evidently, transcriptional regulation of hormonal biosynthesis is not limited to auxins-CKs crosstalk, and plenty of studies illustrate this occurrence for other hormones, resulting in their antagonism/protagonism depending on contexts and plant species. For instance, the biosynthesis of GAs can be regulated during various processes by BRs, ET, and JAs, through the binding of BES1, EIN3, and MYC2, respectively, to the promoter regions of several GAs oxidases (Unterholzner et al., 2015; Wang et al., 2024a). JAs are also able to enhance ET levels during apple (*Malus domestica* Borkh.) fruit ripening by promoting the transcription of *ACS1* and *ACO* via MYC2 (Li et al., 2017b).

Multiple studies indicate that auxin transport is also transcriptionally regulated by CKs, positively or negatively depending on contexts (Růžička et al., 2009; Zhang et al., 2013; Šimášková et al., 2015; Cucinotta et al., 2016; Street et al., 2016). In some cases, this regulation is mediated by B-ARRs, as for ARR1, which enhances *PIN3* expression during gynoecium development (Figure 3A; Reyes-Olalde et al., 2017). Combined with ARR1-mediated promotion of *TAA1* expression, this suggests an accumulation of auxins that is relocalized from the medial domain to the repla and valves of the gynoecium, likely enabling cell specification (Larsson et al., 2014; Reyes-Olalde et al., 2017). In contrast, ARR1 and ARR12 repress *LAX2* expression in root tips, thereby preventing auxin influx in provascular cells and blocking specification of the quiescent center (Zhang et al., 2013). Additionally, CKs promote the expression of *PIN1* and *PIN7* in roots through CK-responsive TFs known as CRFs (Šimášková et al., 2015). In response to CKs, CRFs also positively regulate the expression of *PIN1* in the placenta: this is thought to enable the establishment of an auxin gradient, which is essential for ovule development (Cucinotta et al., 2016). Besides CKs, multiple hormones also seem to regulate *PINs* expression in various contexts: BRs through BES1/BZR1 (Lee et al., 2015; Li et al., 2020e), GAs via DELLAs (Hu et al., 2022b), and ABA likely via ABI4 (Shkolnik-Inbar and Bar-Zvi, 2010); ET, JA-Ile, and SLs may also control the transcription of *PINs,* although precise mechanisms and TFs remain unknown (Lewis et al., 2011; Pandya-Kumar et al., 2014; Jang et al., 2019). Overall, auxin transporters thus appear to be a prime transcriptional target for other hormones to crosstalk with auxins. Importantly, as auxin transport is perhaps best known among plant hormones, insights into its hormone-mediated transcriptional regulation are far more numerous than for other hormonal transporters.

Transcriptional crosstalk also occurs for hormonal signaling elements, as for ARF3, which represses *AHK4* expression and thereby blocks CKs signaling, in addition to CKs biosynthesis, for determinacy of floral meristems (Figure 3B; Zhang et al., 2018b). On the contrary, type-B ARRs can notably bind promoter regions of *TIR1* and *AFB2* (Xie et al., 2018), although biological relevance and occurrence must still be investigated.

The cross-regulation between auxins and CKs is thus largely mediated at a transcriptional level, resulting in either antagonism or synergism depending on the context. Globally speaking, this illustrates how one plant hormone can activate or repress another plant hormone through transcriptional regulation of its biosynthesis and/or transport and/or signaling, and how this is context-dependent.

Crosstalk through direct protein–protein interactions

Besides transcriptional regulation, the importance of direct protein–protein interactions on the cross-regulation of plant hormones has been increasingly highlighted in recent years

(Altmann et al., 2020; Sun et al., 2020). Most notably and as presented in Table 1, a great number of them occur within plant hormonal signaling networks, that is, a signaling protein associated with one hormone interacts with a signaling protein associated with another hormone. As a means to visualize the interactome formed by hormonal signaling proteins, the data shown in Table 1 was used to build a simplified conceptual scheme in Figure 4. Although the data comes from different plant species and conditions (Table 1), and may therefore not fully reflect *in vivo* reality, Figure 4 emphasizes the complex crosstalk mediated by interactions between signaling proteins from various hormonal networks: It is not simply one hormonal signaling pathway influencing another one through one interaction, but all of them conversing together at multiple levels. Such an interplay between these proteins results in a positive or negative outcome, thereby activating or suppressing hormonal signaling hubs and ultimately leading to specific physiological changes as discussed below.

Transcriptional repressors are major crosstalk hubs

The signaling of auxins, GAs, JA-Ile, and SLs relies on their responsive transcriptional repressors, which consist of Aux/IAAs, DELLAs, JAZs, and SMXL6-8, respectively (Tiwari et al., 2001; Chini et al., 2007; Murase et al., 2008; Wang et al., 2015). As explained above, these proteins cannot regulate gene expression alone as they generally lack a DNA-binding domain. Instead, they control gene transcription by interacting with TFs. The range of TFs with which they interact was initially thought to be largely limited to TFs responding to their corresponding hormones, for example, Aux/IAAs-ARFs and JAZs-MYC2 (Tiwari et al., 2001; Zhang et al., 2015). However, these transcriptional repressors are now emerging as staple junctions across hormonal signaling networks, as they have been discovered to interact, in fact, with many other hormone-responsive TFs (Table 1). As the most extreme example, DELLA proteins interact with many hormonal TFs and mediate this way the crosstalk of GAs with auxins, BRs, CKs, ET, JAs, and SLs, in various plant species and processes (Hou et al., 2010; An et al., 2012, 2024; Yang et al., 2012; Bai et al., 2012; Wild et al., 2012; Gallego-Bartolomé et al., 2012; Li et al., 2012; Wild and Achard, 2013; Oh et al., 2014a; La Rosa et al., 2014; Marín-de La Rosa et al., 2015; Hu et al., 2018, 2022b). Similarly, JAZ proteins can bind and thereby repress TFs from ABA, auxin, BR, and ET signaling pathways (Table 1). Meanwhile, OsD53 and AtSMXL6-8 are also able to interact and thereby activate AtBES1 and OsBZR1, respectively (Fang et al., 2020; Hu et al., 2020).

Furthermore, transcriptional repressors can also interact with each other, for example, DELLA proteins can bind and thereby repress JAZ proteins (Hou et al., 2010; Wild et al., 2012; Xu et al., 2025a). Because JAZ9 is also able to repress RGA in Arabidopsis (Yang et al., 2012), this suggests that DELLA and JAZ proteins can mutually repress each other and (in)activate JA-Ile/GAs signaling, respectively, as a means to either induce plant growth or defense (Hou et al., 2010; Wild et al., 2012; Yang et al., 2012). In other plants, D53 has also been highlighted to physically interact with both DELLA and JAZ proteins, resulting in OsSLR1 activation and NaJAZb repression, respectively (Li et al., 2020c; Sun et al., 2024).

In this context, one question is outstanding: how does one transcriptional repressor interact with so many proteins? The answer lies in protein (sub)domains. To illustrate this, we take advantage of Arabidopsis DELLA proteins (GAI, RGA, RGL1-3) that interact with a wide range of proteins: the GAs receptor GID1, the F-box protein SLY1, GAs-responsive TFs like PIFs, chromatin remodelers, other hormonal TFs, and other hormonal transcriptional repressors (Alabadí and Sun, 2025). As an example, DELLA proteins interact with GID1 in the presence of bioactive GAs. GAs binding indeed creates a hydrophobic surface on the amino-terminal extension (N-Ex) domain of GID1, which can then be bound by the DELLA domain of DELLA proteins (Murase et al., 2008; Shimada et al., 2008). This binding with GID1 stabilizes DELLA proteins, which further interact with GID1 through their GRAS domain (Hirano et al., 2010). Following formation of the GAs-GID1-DELLA complex, SLY1 recognizes and interacts with the GRAS domain of DELLA proteins (Dill et al., 2004; Hirano et al., 2010). The SCF^{SLY1} subsequently mediates the ubiquitination of the DELLA domain, resulting in DELLA protein degradation.

The GRAS domain of DELLA proteins is also essential for interacting with various domains of TFs, for example, DNA-binding domains. For instance, the bHLH DNA-recognition domain of PIF4 interacts with the leucine heptad repeats (LHR1 and LHR2) within the GRAS domain of RGA; interaction with RGA thus prevents PIF4 from binding DNA, and this *in fine* impedes hypocotyl cell elongation (De Lucas et al., 2008). Similarly, RGA and GAI interact with a region of EIN3 associated with DNA binding: this inhibition of EIN3 downregulates *HLS1* expression, providing a mechanism by which DELLA proteins interfere with apical hook development (An et al., 2012). Which motif(s) of RGA and GAI exactly interact with the DNA-binding domain of EIN3 remains to be verified, although it is likely part of the GRAS domain and may perhaps correspond to LHR1 (An et al., 2012). In contrast, RGA does not seem to block the DNA-binding domain of BZR1, but instead interacts through LHR1 with the BIN2 phosphorylation domain of BZR1 (Gallego-Bartolomé et al., 2012; Li et al., 2012). RGA seemingly forms an inactive complex with BZR1 to repress root and hypocotyl cell elongation, although the precise mechanism for inhibition of BZR1 transcriptional activity remains unclear (Gallego-Bartolomé et al., 2012; Li et al., 2012). This association between RGA and BZR1, moreover, seems to be stabilized through the additional binding of RGA to histone H2A (Huang et al., 2023a). As this also extends to other TFs interacting with the LHR1 domain of DELLA proteins, DELLA proteins are suggested to form in many instances a tripartite complex at the target chromatin, binding on one side to a TF through LHR1 and binding on the other side to H2A through PFYRE, both being subdomains of the GRAS domain (Huang et al., 2023a).

Table 1. Direct protein–protein interactions within hormonal signaling networks

Hormonal crosstalk		Protein–protein interaction					
Primary hub	Target hub	Primary protein	Effect	Target protein	Plant species	Development stage/Stress condition	References
ABA	BR	ABI1,2	-----|	BIN2	*At*	n.s.	Wang et al. (2018b)
ABA	CK	ABI5	n.d.	A-ARR4-6	*At*	Seed germination	Wang et al. (2011)
ABA	CK	SnRK2s	----->	A-ARR5	*At*	Drought	Huang et al. (2018)
ABA	JA	ABI1	n.d.	JAZ1	*At*	n.s.	Sun et al. (2020)
ABA	JA	PYL4	n.d.	JAZ1	*At*	n.s.	Sun et al. (2020)
ABA	JA	PYL6	-----|	MYC2	*At*	Cotyledon expansion and greening	Aleman et al. (2016)
Aux	ABA	ARF10,16	----->	ABI5	*At*	Seed germination	Mei et al. (2023)
Aux	ABA	TMK1,4	----->	ABI2	*At*	Cotyledon greening, primary root growth	Yang et al. (2021), Li et al. (2021b)
BR	ABA	BES1	-----|	ABI5	*At*	Seed germination	Zhao et al. (2019)
BR	ABA	BIN2	----->	ABI5	*At*	Seed germination	Hu and Yu (2014)
BR	ABA	BIN2	----->	SnRK2.2,2.3	*At*	Primary root growth	Cai et al. (2014)
BR	Aux	BIN2	-----|	ARF2	*At*	Hypocotyl elongation	Vert et al. (2008)
BR	Aux	BIN2	----->	ARF7,19	*At*	Lateral root development	Cho et al. (2014)
BR	Aux	BZR1	----->	ARF6	*At*	Hypocotyl elongation	Oh et al. (2014a)
BR	CK	BZR1	----->	B-ARR1	*At*	Ovule initiation	Zu et al. (2022)
BR	JA	BIN2/GSK2	-----|	JAZ1/JAZ4	*Gh,At/Os*	Defense/n.s.	He et al. (2020), Song et al. (2021a)
BR	JA	BZR2	----->	MYC2	*Ppy*	Bud dormancy release	Wang et al. (2024b)
BR	SA	BIN2	-----|	TGA4	*At*	Defense	Kim et al. (2022)
BR	SA	BIN2	----->	TGA3	*At*	Defense	Han et al. (2022)
CK	SA	B-ARR2	----->	TGA3	*At*	Defense	Choi et al. (2010)
ET	Aux	ERF108	----->	ARF7	*Gh*	Fiber secondary cell wall biosynthesis	Wang et al. (2023d)
ET	Aux	ERF72	-----|	ARF6	*At*	Hypocotyl elongation	Liu et al. (2018)
ET	BR	ERF72	-----|	BZR1	*At*	Hypocotyl elongation	Liu et al. (2018)
ET	BR	EIN3	----->	BES1, BZR1	*At*	Apical hook development, hypocotyl elongation	Zhao et al. (2021b), Wang et al. (2023a)
ET	CK	EIN3	----->	ARR1	*At*	Root growth	Yan et al. (2017)
ET	CK	ETR1	----->	AHP1,2,3,5	*At*	Root apical meristem size	Scharein and Groth (2011), Zdarska et al. (2019)
ET	JA	EIN3	-----|	MYC2	*At*	Defense	Song et al. (2014)
GA	Aux	RGA	-----|	ARF6	*At*	Hypocotyl elongation	Oh et al. (2014a)
GA	Aux	DELLA/RGL1	-----|	ARF7	*Sl/Pt*	Fruit initiation/cambial development	Hu et al. (2018, 2022b)
GA	BR	GAI, RGA	-----|	BZR1	*At*	Root and hypocotyl elongation	Bai et al. (2012), Gallego-Bartolomé et al. (2012), Li et al. (2012)
GA	CK	GAI, RGA	----->	ARR1,10,12	*At*	Root meristem size, photomorphogenesis, and cell specification	Marín-de La Rosa et al. (2015), Yan et al. (2017), Cai et al. (2025)
GA	CK	GID1	-----|	ARR1,10,12	*At*	Cell specification	Cai et al. (2025)
GA	CK	SLY1	-----|	ARR1,10,12	*At*	Cell specification	Cai et al. (2025)
GA	ET	GAI,RGA	-----|	EIN3	*At*	Apical hook development	An et al. (2012)
GA	JA	GAI1	-----|	JAZ2.2	*Me*	Defense	Xu et al. (2025a)
GA	JA	RGA, RGL3	-----|	JAZ1	*At*	n.s.	Hou et al. (2010), Wild et al. (2012)
GA	JA	RGL3	-----|	JAZ8	*At*	n.s.	Wild et al. (2012)

Continued

Table 1. Continued

Hormonal crosstalk		Protein–protein interaction					
Primary hub	Target hub	Primary protein	Effect	Target protein	Plant species	Development stage/Stress condition	References
GA	JA	RGA	-----|	MYC2	*At*	Sesquiterpene biosynthesis	Hong et al. (2012)
GA	SL	RGL2a	-----|	SMXL8	*Md*	Anthocyanin biosynthesis	An et al. (2024)
JA-Ile	ABA	JAZ1	-----|	ABF3	*At*	Salinity	Zhang et al. (2024)
JA-Ile	ABA	JAZ1,5,8	-----|	ABI3	*At*	Seed germination	Pan et al. (2020)
JA-Ile	ABA	JAZ1-2	-----|	ABI4	*Md*	Cold	An et al. (2022)
JA-Ile	ABA	JAZ1/JAZ3	-----|	ABI5	*Ta*/*At*	Seed germination	Ju et al. (2019)
JA-Ile	Aux	JAZ1,9	-----|	ARF10,16	*At*	Seed germination	Mei et al. (2023)
JA-Ile	BR	JAZ1-2	-----|	BIM1	*Md*	Cold	An et al. (2023)
JA-Ile	BR	MYC2-4,2-6/ MYC2	-----|	BZR1	*Zm*/*At*	Internode elongation/apical hook development	Zhang et al. (2023b), Wang et al. (2024e)
JA-Ile	ET	JAZ1	-----|	EIN3, EIL1	*At*	Root development, defense	Zhu et al. (2011)
JA-Ile	ET	MYC2,3,4	-----|	EIN3	*At*	Apical hook development	Song et al. (2014)
JA-Ile	ET	MYC2	-----|	ERF2	*Md*	Fruit ripening	Li et al. (2017b)
JA-Ile	GA	JAZ9	-----|	RGA	*At*	n.s.	Yang et al. (2012)
SA	ET	NPR1	-----|	EIN3	*At*	Apical hook development	Huang et al. (2020)
SA	GA	NPR1	-----|	GID1	*At*	Growth	Yu et al. (2022b)
SA	JA	NPR1	-----|	MYC2,3,4	*At*	Defense	Nomoto et al. (2021)
SA	JA	NPR3,4	-----|	JAZ1	*At*	Defense	Liu et al. (2016)
SL	Aux	D3	-----|	IAA17	*Gh*	Fiber elongation	Tian et al. (2024)
SL	BR	MAX2/D3	-----|	BES1/BZR1	*At*/*Os*	Shoot branching/tillering	Wang et al. (2013), Fang et al. (2020)
SL	BR	SMXL6-8/D53	----->	BES1/BZR1	*At*/*Os*	Shoot branching/tillering	Fang et al. (2020), Hu et al. (2020)
SL	GA	D14	-----|	SLR1	*Os*	Nitrogen limitation, tillering	Nakamura et al. (2013), Sun et al. (2023)
SL	GA	D53/ SMXL7-8	----->	SLR1	*Os*/*Gh*	Nitrogen limitation/fiber elongation	Sun et al. (2023, 2024)
SL	JA	D53	-----|	JAZb	*Na*	Defense	Li et al. (2020c)

Standards arrows indicate a positive effect of the primary protein on the target protein, flat-ended arrows indicate a negative effect, n.d. and n.s. correspond to nondetermined effect and non-specific conditions, respectively. For plant species, *At* corresponds to *Arabidopsis thaliana, Gh* to *Gossypium hirsutum* (cotton), *Me* to *Manihot esculenta* (cassava), *Md* to *Malus* × *domestica* (apple), *Na* to *Nicotiana attenuata* (wild tobacco), *Os* to *Oryza sativa* (rice), *PPy* to *Pyrus pyrifolia* (pear), *Pt* to *Populus tomentosa* (poplar), *Sl* to *Solanum lycopersicum* (tomato), *Ta* to *Triticum aestivum* (wheat), and *Zm* to *Zea mays* (maize). Abbreviations: ABA, abscisic acid; Aux, auxins; BR, brassinosteroids; CK, cytokinins; ET, ethylene; GA, gibberellins; JA-Ile, jasmonoyl-isoleucine; SA, salicylates; SL, strigolactones.

Interestingly, the LHR1 motif within the GRAS domain of RGA is also required, in addition to the DELLA domain, for its interaction with the NT and Jas domains of JAZ1 (Hou et al., 2010). As this Jas domain is responsible for JAZs interaction with MYCs (Zhang et al., 2015), the interaction of RGA/RGL3 with JAZs thereby antagonizes JAZs binding to MYC2 (Hou et al., 2010; Wild and Achard, 2013). In Arabidopsis and in cassava (*Manihot esculenta* Crantz), DELLA proteins thereby promote the expression of MYC2-activated defense genes, thus *in fine* modulating plant defense (Wild and Achard, 2013; Xu et al., 2025a). In contrast, the direct interaction between RGA and the N terminus of MYC2 negatively affects the MYC2-mediated upregulation of *TPS21* and *TPS11* expression, which encode enzymes involved in sesquiterpene biosynthesis (Hong et al., 2012). In this case, DELLA proteins thus block MYC2 signaling to prevent sesquiterpene biosynthesis (Hong et al., 2012).

DELLA proteins are not exclusively transcriptional repressors and can also act as transcriptional co-activators for some hormonal-responsive TFs, most notably ARR1 (Marín-de La Rosa et al., 2015). In this case, the LHR1 motif of GAI is not required for interaction with ARR1; it is instead the VHIID motif, in addition to another motif in the GRAS domain, which is critical for interaction with the glutamine-rich region of ARR1 (Marín-de La Rosa et al., 2015). Thus, GAI binding to ARR1 does not block its DNA-binding domain and even promotes its transcriptional activity at target promoters, thereby positively affecting root meristem maintenance and skotomorphogenesis (Marín-de La Rosa et al., 2015). The enhanced activity of ARR1 upon interaction with GAI may be explained by the recruitment of other transcriptional co-activators by GAI (Marín-de La Rosa et al., 2015); MED15 is suggested for this purpose (Hernández-García et al., 2024).

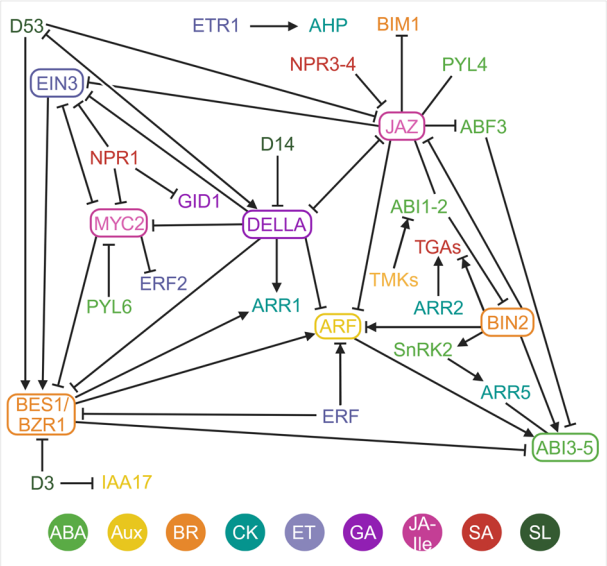

Figure 4. The global interactome formed by hormonal signaling proteins from numerous plant species during various processes
Details on the data used to build this interactome are provided in Table 1. Simple lines indicate protein interactions for which the effect has not been investigated yet, standard arrows indicate a positive effect of the primary protein on the target protein, flat-ended arrows indicate a negative effect, two-ended arrows indicate a double-sided interaction, while a combination of standard arrow and flat end on the same side indicates that the effect is protein-dependent. Depicted interactions only concern proteins from different hormonal networks. Major crosstalk hubs, that is, proteins interacting with at least five other proteins, are highlighted with colored frames. Abbreviations: ABA, abscisic acid; Aux, auxins; BR, brassinosteroids; CK, cytokinins; ET, ethylene; GA, gibberellins; JA-Ile, jasmonoyl-isoleucine; SA, salicylates; SL, strigolactones. Full protein names are provided in Table S1. Created with BioRender: Genva, M. (2026) https://BioRender.com/tptygho.

While we do not further detail such protein domains for other transcriptional repressors like JAZ proteins, explanations are largely similar: each of them contains conserved domains that display different functions critical for interacting with a wide range of proteins, including TFs. Surprisingly, whereas the interaction networks of DELLA and JAZ proteins are well characterized, very few studies have explored AtSMXL6-8/OsD53 and Aux/IAA proteins as possible nodes in hormonal cross-regulation. This represents a promising avenue of research.

Transcription factors associate to form (in)active signaling complexes

Beyond associations between transcriptional repressors and TFs, many hormone-responsive TFs also interact with one another to regulate gene transcription together (Table 1). Whether they cooperate or repress each other depends on the TFs and contexts considered. Seed dormancy is, for instance, maintained most notably through ABA signaling, which auxins promote through ARF10/16-mediated enhancement of ABI5 transcriptional activity (Mei et al., 2023). During seed germination, however, BES1 interacts with the bZIP domain of ABI5, which confers its DNA-binding ability (Zhao et al., 2019). This represses ABI5-mediated activation of target genes, like *EM6*, and thereby contributes to end seed dormancy (Zhao et al., 2019). In another instance, apical hook development is negatively regulated by JAs through the binding of MYC2 to BZR1 and to EIN3 (Song et al., 2014; Zhang et al., 2014b, 2023b). Interaction between MYC2 and BZR1 does not involve their respective DNA-binding domains and rather seems to disrupt BZR1 interaction with PIFs, although PIFs and MYC2 do not bind the same domain in BZR1 (Zhang et al., 2023b). As a result of MYC2-BZR1 competitive binding and thus BZR1-PIFs dissociation, the expression of *WAG2* is not upregulated anymore, and apical hook development is hindered (Zhang et al., 2023b). Additionally, MYC2 binding inhibits the DNA-binding ability of EIN3, supposedly through interaction with its DNA-binding domain, and thereby prevents *HLS1* expression (Song et al., 2014; Zhang et al., 2014b). On the contrary, *HLS1* expression is positively regulated by the binding of BZR1 to EIN3, enabling apical hook development; their association also promotes hypocotyl cell elongation by *PREs* gene upregulation (Zhao et al., 2021b). BZR1 further promotes hypocotyl cell elongation by also forming a signaling complex with ARF6 to induce the expression of *SAUR15,* notably (Oh et al., 2014a). This illustrates how different contexts require multiple diverse hormonal TF interactions to mediate appropriate responses.

While TF repression is quite logically explained through interaction with DNA-binding domains in most cases, the promotion of transcriptional activity is often less clear. The positive interaction between ARF16 and ABI5, for instance, is not mediated through their DNA-binding domains (Mei et al., 2023). Instead, the middle region of ARF16 (amino acids 357-544) and the amino acid residues 165–220 of ABI5 are required for interaction (Mei et al., 2023). Similarly, it is not the DNA-binding domain but again the middle domain of ARF6, as well as its C-terminal domain, which are responsible for its interaction with BZR1 (Oh et al., 2014a). However, more detailed insights into what precisely enhances their transcriptional activities, for example, possibly the recruitment of additional regulators, are currently lacking.

Phosphatases and kinases are major key players in crosstalk by regulating protein activity

ABA, auxins, BRs, and CKs largely rely on phosphorylation cascades for signal relaying from receptors to TFs, and in fact, these hormonal pathways also crosstalk with each other through this means. For instance, phosphorylation of ABI2 by TMK1 and TMK4 mediates the interplay between ABA signaling and auxin extracellular signaling, notably in the context of cotyledon greening (Yang et al., 2021; Li et al., 2021b). Most interestingly, it must be noted that this interaction seems to mediate both auxins-ABA synergism and antagonism, depending on whether ABI2 phosphorylation is performed by TMK1 or TMK4, respectively (Yang et al., 2021; Li et al., 2021b). TMK4-mediated phosphorylation of ABI2 indeed enhances its phosphatase activity, resulting in higher

inhibition of ABA signaling and thereby promoting plant growth (Li et al., 2021b). In contrast, phosphorylation of ABI2 by TMK1 represses its phosphatase activity, contributing this way to higher SnRK2s activation (Yang et al., 2021). This may be explained by the differential ABI2 phosphorylation sites of TMK1 versus TMK4: while TMK1 phosphorylates ABI2 at Thr-321, TMK4 targets ABI2 at Ser-139, Ser-140, and Ser-266 (Yang et al., 2021; Li et al., 2021b). Further investigation is required to better decipher this intriguing phenomenon.

Meanwhile, ABA is able to antagonize CKs signaling during drought by promoting the stabilization of ARR5, through ARR5 phosphorylation by SnRK2s likely at Ser-21, Ser-33, Ser-72, and Ser-117 (Huang et al., 2018). In contrast, ABA rather activates BRs signaling through the ABI1- and ABI2-mediated dephosphorylation of BIN2: this inhibits the kinase activity of BIN2 and thus prevents it from inactivating BES1 (Wang et al., 2018b). While ABA activates BR signaling by antagonizing BIN2 in that case, BIN2, on the contrary, promotes ABA signaling during seed dormancy, by stabilizing ABI5 through phosphorylation at different sites (Thr-35, Ser-36, Ser-41, Ser-368, and Ser-372) (Hu and Yu, 2014). BIN2 also mediates ABA–BRs antagonism in primary root growth inhibition. In this context, indeed, BIN2 phosphorylates SnRK2.3 on Thr-180, thereby increasing its kinase activity and promoting ABA signaling (Cai et al., 2014).

Phosphorylation does, however, not mediate crosstalk between ABA, auxins, BRs, and CKs only. Most notably, BRs crosstalk through BIN2 with the SA and JA-Ile pathways in the context of pathogen defense. Arabidopsis defense against the bacterial pathogen *Pseudomonas syringae* pv. *tomato* DC3000 indeed seems to be modulated by BIN2, which phosphorylates TGA3 and/or TGA4 (Han et al., 2022; Kim et al., 2022). Phosphorylated TGAs interact differently with NPR1, thereby likely affecting expression of defense genes (Han et al., 2022; Kim et al., 2022); because these studies nonetheless display opposite results for similar experiments, further investigation is required to clarify these interactions. Additionally, Arabidopsis defense against the soil-borne fungus *Verticillium dahliae* is negatively regulated by BIN2, which interacts with the ZIM domain of JAZ1 (Song et al., 2021a). BIN2 indeed phosphorylates JAZ1 at Thr-196, and this promotes JAZ1 degradation through an unknown mechanism (Song et al., 2021a). JAZ1 degradation is thought to globally activate the JA-Ile pathway through MYC2 release; this would antagonize SA-mediated defense and thereby prevent adequate responses to the pathogen, although this remains to be confirmed (Song et al., 2021a). Furthermore, ABI1 has also been reported to interact with JAZ1 (Sun et al., 2020), but whether ABI1 is able to dephosphorylate JAZ1 or not remains to be investigated. The outcome and the biological relevance of their interaction also remain to be deciphered.

Hormonal receptors interact with various signaling proteins from other hormonal hubs

Various hormonal receptors also interact with signaling components from other hormonal hubs: this includes PYL4 with JAZ1, PYL6 with MYC2, ETR1 with AHPs, GID1 with B-ARRs, D14 with SLR1, and most remarkably NPRs with EIN3, GID1, MYC2-4, and JAZ1 (Table 1). As an example, the binding of NPR3-4 to SA favors their interaction with JAZ1 during the hypersensitive response (Liu et al., 2016). This seemingly triggers JAZ1 proteasomal degradation, possibly through NPR3-4 acting as substrate adapters for CUL3, and thereby globally activates the JA-Ile pathway (Liu et al., 2016). SA signaling also mediates apical hook development, which is blocked by the interaction of the BTB/POZ domain of NPR1 with the DNA-binding domain of EIN3 (Huang et al., 2020). This effectively prevents EIN3 from binding its target promoters, and *HLS1* expression is hence downregulated, illustrating how SA antagonizes ET signaling in this case (Huang et al., 2020). In another instance, ET signaling cooperates with CKs signaling to control the size of the root apical meristem, through the interaction of ETR1 with several AHPs (Zdarska et al., 2019). As ETR1 is known to autophosphorylate on its His residue, ETR1 may perhaps activate AHPs by phosphorylating them, though this remains to be confirmed (Zdarska et al., 2019).

In a unique example, SA receptors other than NPRs can also mediate crosstalk between SA and other hormonal networks (Manohar et al., 2017). SA is indeed able to bind to multiple SA-binding proteins, including certain PP2Cs that are involved in ABA signaling (Manohar et al., 2015, 2017). This outstanding example outlines another way for SA to mediate the growth-defense tradeoff in Arabidopsis, and further emphasizes the need for future research to investigate SA-binding proteins other than NPRs.

F-box proteins keep more than one hormonal protein in check

SLY1 and AtMAX2/OsD3 are F-box proteins that recognize for proteasomal degradation DELLA and AtSMXL6-8/OsD53, respectively (Zhou et al., 2013; Islam et al., 2025). Recent studies, however, highlighted that SLY1 additionally triggers the degradation of ARR1, ARR10, and ARR12 (Cai et al., 2025), while AtBES1/OsBZR1 are also targeted for degradation by AtMAX2/OsD3 (Wang et al., 2013; Fang et al., 2020). In Arabidopsis, indeed, SLs promote shoot branching through BES1, which is degraded following interaction of its PEST domain with MAX2 (Wang et al., 2013). In rice, this shoot branching rather corresponds to tillering, which interestingly also relies on the interaction between these proteins (Fang et al., 2020).

Interactions between signaling proteins and biosynthetic enzymes and/or transporters also mediate hormonal crosstalk

Although physical interactions are most described to occur between hormonal signaling proteins, they are not restricted to signaling networks only. Some signaling proteins can indeed interact with biosynthetic enzymes and transporters to modulate their activity. For instance, ABI1 interacts with ACS6 and dephosphorylates its C-terminal fragment: this

undermines ACS6 activity and thus limits ET production during ozone stress (Ludwików et al., 2014). Auxin transport is also post-translationally targeted by several hormonal hubs like BRs (Lu et al., 2022). PIN1 is indeed targeted for direct phosphorylation by BIN2: This supposedly affects PIN1 polarity, which is of crucial importance for auxin transport (Lu et al., 2022). Furthermore, transporter–transporter interactions may also occur, as for the CKs-carrier AZG1 that stabilizes PIN1 in Arabidopsis root cells (Tessi et al., 2023).

Crosstalk through signaling-node rewiring

While direct transcriptional regulation and direct protein–protein interactions are two major means for cross-regulation between hormones, they do not constitute the full arsenal for plant hormones to crosstalk and are, in fact, rather simple mechanisms. There is indeed in many instances a signaling cascade that relays crosstalk between different hormonal networks.

In the simplest scenario, an intermediary protein mediates crosstalk between two hormonal proteins, like the TF WRKY57 with JAZ4 and IAA29. JAZ4 and IAA29 indeed regulate leaf senescence antagonistically by competitive binding to the zinc-finger domain of WRKY57, which modulates the expression of *SEN4* and *SAG12* notably (Jiang et al., 2014). Here, WRKY57 thus mediates crosstalk between auxins and JA-Ile during leaf senescence (Jiang et al., 2014). In the same vein, the histone acyltransferase HLS1 serves as a junction between ET and ABA signaling pathways during germination (Guo et al., 2023). HLS1 promotes ABA signaling through histone acetylation of *ABI3* and *ABI5* loci; however, ET counteracts this through EIN2, which interacts with HLS1 in the nucleus and thereby inactivates it (Guo et al., 2023).

In more complex scenarios, there is a whole downstream cascade from one hormone to another. This cascade sometimes includes transcriptional and non-transcriptional crosstalk. For instance, SA can, through NPR1, likely promote the expression of *ABAPT11*, a gene encoding an enzyme involved in protein deacylation, which targets BSKs (Liu et al., 2023a). ABAPT11-mediated deacylation of BSKs disrupts the function of BSKs: SA thereby inhibits BRs signaling to prevent plant growth (Liu et al., 2023a). This example of a post-translational modification—other than phosphorylation and polyubiquitination—regulating protein activity is far from being an exception in hormonal networks, including in crosstalk (Hill, 2015; Blanco-Touriñán et al., 2020). In other cases, cascades consist of multiple protein–protein interactions. Root architecture during salt stress is, for instance, regulated by ABA, which alters auxin transport by inducing PIN2 phosphorylation at Ser-258 through a PYLs–PP2A–PID cascade (Li et al., 2020b). The phosphorylation status of PIN2 is indeed antagonistically regulated by PP2As phosphatases and the PID kinase, which interact with each other (Li et al., 2020b). In the absence of ABA, PYLs interact with the catalytic C subunits of PP2As, and this promotes the activity of PP2As, seemingly resulting in a lower PID-mediated phosphorylation of PIN2. (Li et al., 2020b). ABA binding to PYLs, on the contrary, inhibits PP2As activity, and in turn, PIN2 phosphorylation increases, disrupting *in fine* auxin polar transport in roots (Li et al., 2020b). Most interestingly, SA also regulates root development by modulating PIN2 phosphorylation via PP2As (Tan et al., 2020). SA is indeed able to bind the A subunits of PP2As: This represses their activity and results in an increase in PIN2 phosphorylation, which seems likely mediated by PID and other kinases (Tan et al., 2020). Thus, ABA and SA signaling appear to both converge toward PP2As in order to crosstalk with auxins for the regulation of root development (Li et al., 2020b; Tan et al., 2020). Besides, SLs also modulate auxin transport during root development and shoot branching; the precise mechanism has nonetheless not been fully deciphered yet. What is understood for now is that SLs seem to control through D14 and MAX2 the localization of PIN1 and PIN2 at the PM, and that this is related to the clathrin-dependent endocytosis of PINs (Shinohara et al., 2013; Pandya-Kumar et al., 2014; Zhang et al., 2020b). Further insights on this phenomenon are awaited to further unveil the mechanisms behind the effect of SLs on PINs localization.

Getting the bigger picture of plant hormonal crosstalk

The regulation of one physiological event by a multitude of hormonal networks

With this multitude of molecular crosstalk mechanisms, it is evident that the signaling networks of plant hormones do not simply form nine individual parallel lines but rather constitute a vast spiderweb, with numerous intersections at various levels. The occurrence of crosstalk is, however, highly context-specific: Not all interactions always arise in all tissues, whether they are transcriptional or physical. On the contrary, crosstalk generally displays a temporal and spatial specificity leading to unique physiological outcomes. This is notably exemplified with the mediation of cell type specification by auxins and CKs: They act synergistically for gynoecium development, but rather antagonistically during shoot meristem regeneration (Cheng et al., 2012; Meng et al., 2017; Reyes-Olalde et al., 2017). It is moreover becoming increasingly evident that in many cases, it is not simply two hormones crosstalking but multiple hormones dictating together the fate of an event (Figure 5).

Seed dormancy, for instance, is largely enabled by ABA signaling through ABI3/5, which represses GAs at a transcriptional level (Shu et al., 2016). Multiple hormonal proteins furthermore directly interact with ABI3/5 to reinforce or downregulate ABA signaling, as a means to either prevent or induce seed germination (Figure 5A; Hu and Yu, 2014; Ju et al., 2019; Zhao et al., 2019; Pan et al., 2020; Mei et al., 2023). More precisely, auxins promote the transcriptional activity of ABI5 through ARF10 and ARF16 (Mei et al., 2023), whereas JA-Ile induces the degradation of JAZ proteins, and thereby ends JAZ-mediated repression of both ARF10/16 and ABI3/5 (Ju et al., 2019; Pan et al., 2020; Mei et al., 2023). On the contrary, BRs facilitate seed germination by repressing ABI5, both through BES1 activation and BIN2 inhibition (Hu and Yu, 2014; Zhao et al., 2019).

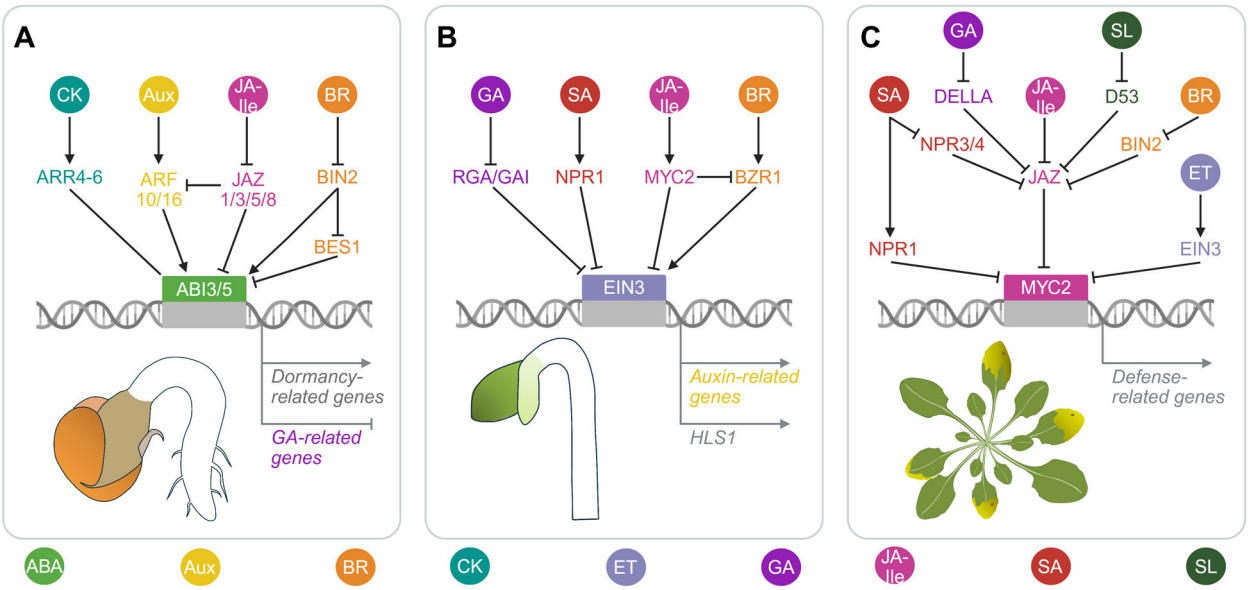

Figure 5. The context-specificity of plant hormonal crosstalk

(A) Seed dormancy is primarily controlled by ABI3/5 through transcriptional regulation of dormancy-related genes and of GA-related genes. The action of ABI3/5 is, moreover, promoted or repressed by direct interaction with various hormonal proteins. **(B)** Apical hook development is mostly induced by EIN3, which induces the expression of *HLS1* and of auxin-related genes. Multiple hormonal proteins modulate the action of EIN3 through (in)direct physical interactions. **(C)** Defense against pathogens is mainly activated by MYC2, which is stimulated or repressed through direct interactions with MYC2 or JAZ proteins. Standard arrows indicate a positive effect, flat-ended arrows indicate a negative effect, and a simple line indicates protein interactions for which the effect has not been investigated yet. Abbreviations: ABA, abscisic acid; ABI3/5, ABA INSENSITIVE3/5; ARR4-6, ARABIDOPSIS RESPONSE REGULATOR4-6; ARF10/16, AUXIN RESPONSE FACTOR10/16; Aux, auxins; BES1, BRI1-EMS SUPPRESSOR1; BIN2, BRASSINOSTEROID INSENSITIVE2; BR, brassinosteroids; BZR1, BRASSINAZOLE-RESISTANT1; CK, cytokinins; D53, DWARF53; EIN3, ETHYLENE INSENSITIVE3; ET, ethylene; GA, gibberellins; GAI, GIBBERELLIN INSENSITIVE; JA-Ile, jasmonoyl-isoleucine; JAZ, JASMONATE-ZIM DOMAIN; MYC2, MYELOCYTOMA2; NPR, NON PATHOGENESIS RELATED; RGA, REPRESSOR OF GA; SA, salicylic acid; SL, strigolactones. Created with BioRender: Genva, M. (2026) https://BioRender.com/fqvobz8.

In contrast, ET is central for the induction of apical hook development because it activates EIN3 that works in concert with PIFs to promote the expression of *HLS1* and auxin-related genes, which *in fine* enable hook formation (Wang et al., 2023b); this is positive crosstalk from ET to auxins at a transcriptional level. Nonetheless, many other hormonal networks additionally directly interact with EIN3 to promote apical hook development, as for BRs and GAs via BZR1 and DELLA degradation, or to prevent it as for SA and JA-Ile through NPR1 and MYC2 (Figure 5B; An et al., 2012; Song et al., 2014; Zhang et al., 2014b, 2023b; Huang et al., 2020; Zhao et al., 2021b).

In a last example, JA-Ile mediates defense against numerous pathogens through MYC2, and other hormonal proteins can moreover modulate MYC2-driven defense in a direct or indirect manner (Figure 5C). Direct modulation involves direct interaction with MYC2, such as for NPR1 and EIN3, which thereby repress MYC2 (Song et al., 2014; Nomoto et al., 2021). Indirect regulation of MYC2 consists of protein interaction with JAZ proteins, which are responsible for MYC2 sequestration. This applies to DELLAs, D53, and BIN2 that repress JAZ proteins and thereby promote JA-Ile signaling; upon increasing levels of their respective bioactive hormones (GAs, SLs, BRs), they are inhibited, and thus JA-Ile-mediated defense is impaired by JAZ proteins (Hou et al., 2010; Wild and Achard, 2013; Liu et al., 2016; Li et al., 2020c; Song et al., 2021a).

All in all, this highlights that the same event is under the control of multiple hormones, which crosstalk through both transcriptional regulation and protein interactions; these mechanisms are moreover largely context-specific.

Evolution of crosstalk mechanisms

Many protein–protein interactions have been studied in specific plant species during particular processes (Table 1), making it difficult to assess whether they are frequently encountered or not. In some cases, though, independent studies have revealed similar protein–protein interactions in various situations (Table 1), suggesting that the crosstalk mechanism may be relatively conserved among plant species and/or processes. For instance, physical interactions between BIN2 and JAZ proteins have been described in three plant species (Arabidopsis, rice, and cotton (*Gossypium hirsutum* L.)) (He et al., 2020; Song et al., 2021a), as for JAZ and DELLA proteins in Arabidopsis and cassava (Hou et al., 2010; Wild and Achard, 2013; Xu et al., 2025a). The physical repression of several ARF TFs by DELLA proteins has also been highlighted in three plant species (Arabidopsis, tomato (*Solanum lycopersicum* L.), and poplar (*Populus tomentosa* Carrière)) (Oh et al., 2014a; Hu et al., 2018, 2022b). Similarly, physical interactions have been observed for BES1/BZR1 with AtMAX2/OsD3 as well as with AtSMXL6-8/OsD53 in Arabidopsis and rice (Wang et al., 2013; Fang et al., 2020;

Sun et al., 2023). Furthermore, some of these protein–protein interactions seem to occur in several plant species in similar processes (Table 1). It would thus seem in this view that some protein–protein interactions may be conserved to some extent among various plant species in alike contexts, likely in link with key conserved amino acid residues in both proteins.

How and why protein–protein interactions are conserved or not among plant species will hopefully be revealed in the coming years by phylogenetic studies. Such studies have indeed already disclosed evolutionary changes in individual pathways, in particular for JAs between vascular plants (Tracheophyta) and non-vascular plants like bryophytes (Chini et al., 2023). In contrast to Arabidopsis, the common liverwort (*Marchantia polymorpha* L.) was indeed shown not to produce the bioactive JA-Ile (Monte et al., 2018). Instead, it is not JA-Ile but dinor-12-oxophytodienoic acid that is the bioactive jasmonate for COI1-JAZ interaction in *Marchantia* (Monte et al., 2018; Kaji et al., 2024). Contrary to jasmonic acid with JA-Ile, the conjugation of dinor-12-oxophytodienoic acid to isoleucine has not been observed in *Marchantia*, although it occurs with other amino acids (Liang et al., 2024). In addition to using a different COI1 ligand, *Marchantia* displays a simpler hormonal network with only one JAZ protein; for comparison, Arabidopsis displays 13 *JAZ* genes (Monte et al., 2019). Similarly, there is only one Aux/IAA transcriptional repressor in the auxin signaling of *Marchantia* (Flores-Sandoval et al., 2015). With a better understanding of individual pathways in *Marchantia* and other species, it may become possible to decipher crosstalk between these hormonal pathways, and this could offer exciting insights into the evolution behind hormonal cross-regulation (Monte, 2023; Komatsu et al., 2025).

CURRENT AND FUTURE DIRECTIONS IN PLANT HORMONAL RESEARCH

Major progress and breakthroughs in plant hormonal control have led the research community into the dissection of hormonal pathways in order to better understand how they regulate plant growth, development, and stress responses. But while filling gaps on both individual hormonal pathways and crosstalk in various situations is crucial, these are far from being the only stakes in the field of plant hormones, and many questions still need to be fully addressed to get a holistic view of plant hormonal control. Important aspects remain to be elucidated, including the mechanisms by which plant hormonal pathways are activated under specific conditions, and the ways by which these plant hormones interact with other signaling messengers *in planta*. In addition, the influence of non-plant organisms capable of producing and/or manipulating plant hormones adds an extra layer of complexity that is not yet fully understood. Equally crucial is the need to translate this expanding molecular knowledge into practical applications. Addressing these aspects will be essential to advance the field for a better integrated understanding of plant hormonal control, as discussed below through current insights and applied perspectives.

From sensing to hormonal signaling

With most articles addressing how plant hormones drive changes in plants, research is mainly focused on the consequences of plant hormonal signaling. A crucial piece is therefore missing to fully contextualize plant hormonal control, that is, the primary triggers of plant hormonal signaling. Plant hormonal networks must indeed be activated before they can drive downstream changes. This is even more important as each situation requires specific responses from plants, and thus the (in)activation of plant hormonal pathways is context-dependent. In this view, it is highly relevant to investigate what happens between the sensing of a specific condition and the resulting hormonal signaling.

A situation in which plant hormonal triggering is relatively well understood is the transition from seed dormancy to germination, which is regulated by light, notably (Yang et al., 2020). In the absence of red light indeed, seed germination is blocked at the endosperm level as cytosolic phytochromes B — a family of apoproteins perceiving red/far-red-light — remain in an inactive signaling state. This enables light-responsive TFs known as PIFs/PILs to accumulate in the nucleus and globally maintain a high ABA/GA balance through transcriptional regulation (Yang et al., 2020). This dominance of ABA over GA maintains the seed in a dormant state as ABA positively regulates storage proteins and lipid reserves (Finkelstein et al., 2008). In the presence of red light, however, phytochromes B become activated and translocate from the cytosol to the nucleus, where they interact with PIFs/PILs (Yang et al., 2020). This results in opposite transcriptional changes that globally enable the switch between ABA and GAs, and this change in hormonal dominance ultimately controls, in concert with other plant hormones (Wang et al., 2011; Ju et al., 2019; Zhao et al., 2019; Pan et al., 2020; Guo et al., 2023; Mei et al., 2023), the transition from seed dormancy to germination (Shu et al., 2016; Yang et al., 2020; Li et al., 2022). Here, the signaling link between light sensing by phytochromes B and hormonal triggering thus consists of the PIFs/PILs TFs, which notably promote ABA biosynthesis and signaling and suppress GA biosynthesis and signaling.

In many cases, however, the cascade from sensing to inducing plant hormonal signaling remains incompletely uncovered. For some environmental stresses, this may be explained by the fact that the mechanisms by which plants sense these stresses are still unknown or were only very recently uncovered, as for salt stress and aluminum toxicity, notably (Jiang et al., 2019; Ding et al., 2024; Xu et al., 2025b). This now opens the door to exciting novel studies expected to disclose the signaling cascade between condition sensing and hormonal triggering. Unveiling such a link is of crucial importance to better understand both the causes and consequences of plant hormonal signaling.

Plant hormones across space and time

Even within the same plant species, plant hormonal signaling displays a spatio-temporal variability that depends on external conditions, developmental stages, and plant tissues, notably. For instance, Arabidopsis seems to display at an adult vegetative stage a much higher expression of *JAZ1* in roots than in shoots, contrary to *JAZ9* (Jin and Zhu, 2017). This spatio-temporality is now increasingly well-illustrated thanks to progress in mass spectrometry imaging and the emergence of numerous hormonal direct/indirect biosensors (Chen et al., 2024). For instance, GPS1 and its improved version, GPS2, are able to bind GA4, and hence, they are direct GA4 biosensors that notably revealed GA4 dynamics during the hypocotyl growth of Arabidopsis (Griffiths et al., 2024). Their use highlighted that cell length in etiolated hypocotyls is correlated with an accumulation gradient of GA_4, which is mainly generated via the light-dependent patterned expression of *GA20ox1* (Griffiths et al., 2024). In contrast, qmRGA is an indirect GA_4 biosensor as it is a modified fluorescent DELLA protein designed to be inactive in signaling but still degraded upon GID1 perception of GAs (Shi et al., 2024). The ongoing combined development of biosensors and mass spectrometry imaging is expected to yield novel insights into the spatiotemporal regulation of plant hormones, and even perhaps of their crosstalk.

Plant hormones, but not solely

While plant hormones are well established as major chemical messengers, they are part of a wide signaling network that also includes calcium, reactive oxygen species (ROS), nitric oxide, peptides, and microRNAs, notably. In this view, plant hormonal crosstalk extends beyond hormone–hormone interactions to include these additional signaling components.

Notably, hormone peptides are small signaling peptides that play critical roles in nutrient acquisition and in the maintenance of stem cells (de Bang et al., 2017; Selby and Jones, 2023). Since the discovery of systemin in local and systemic defense signaling (Pearce et al., 1991; Zhang et al., 2020a), many more hormone peptides have been described, many of which belong to the CLE family (Hirakawa and Sawa, 2019). Among them, CLE19 represses BRs signaling by triggering a cascade for BIN2 activation during pollen exine patterning, whereas CLE25 induces ABA signaling to trigger stomatal closure in shoots during drought (Takahashi et al., 2018; Wang et al., 2025b). Stomatal closure is indeed a key ABA-mediated response to drought (Chen et al., 2020). Drought must, however, be primarily sensed in roots while stomatal closure occurs in shoots: a signal moving from roots to shoots thus must exist to trigger this response. In Arabidopsis, the CLE25 peptide was recently unveiled to fulfill this role (Takahashi et al., 2018). Upon drought, CLE25 is produced in roots and translocated to shoots, where it induces ABA accumulation by upregulating the *NCED3* biosynthetic gene, notably, possibly via interaction with BAM receptors (Takahashi et al., 2018). Interestingly, ABA subsequently relies on ROS signaling to induce stomatal closure (Mustilli et al., 2002). Activation of PYR/PYLs receptors by ABA indeed results in PP2Cs inhibition and thereby SnRK2 release (Chen et al., 2020). Among these, SnRK2.6/OST1 interacts with and phosphorylates RBOHF, likely activating this way ROS production (Sirichandra et al., 2009), though this remains to be fully demonstrated. In a nutshell, stomatal closure is thus mediated during drought by a signaling cascade at least consisting of CLE25, ABA, SnRK2.6, and ROS (Mustilli et al., 2002; Sirichandra et al., 2009; Takahashi et al., 2018).

The interplay between ROS and auxins also mediates root architecture changes during xerobranching. Soil air gaps indeed trigger the accumulation of ROS in elongating root cells, and this induces in the nucleus the multimerization of IAA3 through the formation of disulfide bonds between its cysteine residues (Roy et al., 2025). Multimerization enhances the repressive function of IAA3 via increased interaction with TPL, thereby blocking lateral root development (Roy et al., 2025). Interestingly, ROS-dependent auxin signaling also suppresses lateral root formation in rice to avoid heavy metal stress (Wang et al., 2023c), suggesting a conserved mechanism.

During seed germination, NO also modulates hormonal signaling by reducing ABA levels to break dormancy (Albertos et al., 2015; Zhao et al., 2024a). NO is indeed rapidly produced upon seed imbibition, and this triggers *S*-nitrosylation of both ABI5 and MYB30 (Albertos et al., 2015; Zhao et al., 2024a). This facilitates the degradation of ABI5 via interaction with E3 ligases, such as KEG and CUL4 (Albertos et al., 2015). In contrast, *S*-nitrosylation of MYB30 enhances its transcriptional activity by relieving PYL4-mediated repression of MYB30, thereby promoting expression of *CYP707A2* and thus ABA catabolism (Zhao et al., 2024a). NO thus promotes seed germination of Arabidopsis through inhibition of ABA signaling (Albertos et al., 2015; Zhao et al., 2024a).

In addition to classical chemical messengers, other compounds emerge as important signaling molecules, such as karrikins. Karrikins are smoke-derived compounds integrating a butenolide moiety, which are hence structurally relatively close to SLs (Flematti et al., 2015). Most interestingly, the signaling of karrikins relies on MAX2, that is, the same F-box protein involved in SLs signaling; yet, karrikins and SLs do not always regulate the same processes in plants (Nelson et al., 2011). More specifically, in Arabidopsis, karrikins do not control shoot branching, contrary to SLs; nonetheless, karrikins stimulate seed germination much more than SLs (Nelson, 2021). How MAX2 differentially mediates SLs versus karrikin signaling will hopefully be unveiled, as well as how this connects to their differential use in plants.

This underlines the importance of integrating hormonal crosstalk with other signaling networks to fully understand how plants coordinate responses to environmental cues.

Plant hormones outside of plants

Because hormones are central signaling components in plants, they also mediate interactions between plants and non-plant organisms, some of which are remarkably able to manipulate hormonal networks to promote infection or symbiosis.

One of the most well-known examples is *Pseudomonas syringae* pv. tomato DC3000, a bacterial pathogen that induces JA signaling by secreting in plant cells a JA-Ile mimic called coronatine (Zheng et al., 2012; Lee et al., 2013). By binding COI1, coronatine triggers JAZ degradation and ends MYC2 sequestration (Katsir et al., 2008; Yan et al., 2009; Zheng et al., 2012). MYC2 then upregulates NAC TFs that act on the expression of *SAGT1* and *ICS1* in order to respectively induce SA storage and block SA biosynthesis (Zheng et al., 2012). As DC3000 is SA-sensitive, suppression of SA signaling via coronatine-mediated COI1 activation enhances its virulence. More recently, DC3000 was also shown to manipulate ABA signaling via the AvrE effector (Hu et al., 2022a). AvrE is indeed able to interact with TOPP phosphatases, resulting in elevated SnRK2 phosphorylation and activation of ABA signaling (Hu et al., 2022a). This induces *in fine* stomatal closure, thereby creating a favorable aqueous environment for DC3000 (Hu et al., 2022a).

Although some fungi also appear to hijack plant hormonal control, underlying mechanisms remain less clear. For instance, infection by *Botrytis cinerea* induces a massive production of IAA-aspartate conjugates in plant cells, and this is curiously suggested to promote disease development through the transcriptional regulation of fungal virulence genes (González-Lamothe et al., 2012). While multiple pathogenic but also beneficial fungi are known to produce auxins and CKs, the functions of such fungi-derived hormones remain poorly explored (Chanclud and Morel, 2016). Strikingly enough, CKs are synthesized by many mycorrhizal, (hemi)biotrophic and even saprophytic fungi, but very few necrotrophic fungi seem to produce them (Chanclud and Morel, 2016). Whether fungi-derived cytokinins are perceived and used by fungi themselves, for instance, to support growth, and/or how they also affect plants during plant–fungus interactions, remains an open question. In this view, investigating if fungi are able to perceive their own versions of plant hormones, and/or if these fungal hormones are secreted into plant cells in order to contribute to virulence/symbiosis, will be critical.

Plant–parasitic nematodes also heavily rely on exploiting plant hormonal pathways (Gheysen and Mitchum, 2019; Jhu et al., 2025). To facilitate cell expansion and/or fusion for the creation of their feeding sites, nematodes are able to upregulate auxin biosynthesis and to alter auxin transport (Gheysen and Mitchum, 2019). While effectors such as 19C07 and 10A07 from *Heterodera schachtii* contribute to this (Lee et al., 2011; Hewezi et al., 2015), scarce data are otherwise available to explain how nematodes act on the auxin pathway. Notably, conjugated forms of auxins have been found in several nematode species, but whether this contributes or not to plant manipulation remains to be deciphered (Gheysen and Mitchum, 2019). Plant–parasitic nematodes must additionally suppress host defense, which they trigger during their establishment, and that is also mediated by plant hormones (Gheysen and Mitchum, 2019). In this view, the effector 2G02 from *Meloidogyne javanica* strongly attenuates host cell death, likely by interfering with JA-Ile signaling (Song et al., 2021b), while *M. incognita* secretes an ICS-1 effector that disrupts SA biosynthesis (Qin et al., 2022).

Globally, plant hormones are critical players in the mediation of interactions between plants and non-plant organisms, whether they are pathogenic or beneficial. Yet, the molecular mechanisms by which these non-plant organisms manipulate plant hormones to their benefit, and notably the consequences of hormone production by non-plant organisms, remain incompletely understood.

Toward applied perspectives

Ultimately, fundamental research on plant hormonal control delivers valuable insights for improving crop production, notably through breeding. This was reflected during the Green Revolution with the development of semi-dwarf rice and wheat (*Triticum aestivum* L.) varieties, which achieved higher yields through improved grain resource allocation and lodging resistance (Robil et al., 2025; Xiao et al., 2025). While no molecular background for these particular traits existed in the 1960s, they were later explained by major defects in GA biosynthesis/signaling (Peng et al., 1999; Sasaki et al., 2002).

Developing new semi-dwarf varieties is still envisioned today, notably to address hybrid maize (*Zea mays* L.) lodging exacerbated by high planting density (Robil et al., 2025; Xiao et al., 2025). Contrary to rice and wheat, though, direct knockout of GAs-related genes is not a viable solution in maize due to severe developmental defects, so future strategies are rather suggested to target GAs indirectly (Xiao et al., 2025). Nonetheless, GAs disruption also results in a reduced nutrient use efficiency, and while this side-effect was previously counteracted by large fertilizer applications, this seems unsustainable in the long run (Robil et al., 2025; Xiao et al., 2025). A proposed alternative is in this view to work on BRs, which also control plant height without affecting nutrient use efficiency (Yang et al., 2024; Xiao et al., 2025). Potential side effects of BRs should, however, be thoroughly investigated, and a comprehensive understanding of their pathway is still lacking in many cereal crops (Yang et al., 2024). Finally, while cereal crops were most discussed here, it is evident that crop-specific approaches are necessary for breeding. In rapeseed (*Brassica napus* L.) for instance, yield improvement may be achieved through an increase in seed weight, caused by a mutation in an enzyme degrading CKs (Schwarz et al., 2020).

The achievement of high agricultural yields is also largely conditioned by the concentration and the availability of nutrients in soils (White and Brown, 2010). Among them, primary macronutrients — especially nitrogen and phosphorus — represent critical constraints for plant growth and thus yield: this issue is currently managed with the application of chemical fertilizers (White and Brown, 2010). The excessive use of fertilizers is nonetheless problematic at many levels, with biodiversity loss, eutrophication, environmental pollution, limited production resources, and so on (White and Brown, 2010; Oldroyd and Leyser, 2020). Alternatives to

fertilizers must thus be explored for a more sustainable yet still productive agriculture. Because plant hormones modulate not only nutrient acquisition but also responses to nutrient levels (Jia et al., 2022), we can perhaps leverage them to improve nutrient use efficiency and thereby reduce fertilizer application (Li, 2024). Nitrate, for instance, is transported by OsNRT1.1B, which was recently also discovered to bind ABA (Ma et al., 2025a). This results in the release of the OsNLP4 TF from OsSPX4, and hence in OsNPL4-activated ABA transcriptional responses (Ma et al., 2025a). It would be interesting to further explore if NTR1.1B may be a target for crop engineering. Auxins may also be targeted for this purpose as they affect nutrient use efficiency in multiple ways (Li, 2024). A concrete example is highlighted with OsDNR1, an amino transferase that negatively affects nutrient use efficiency by competing for indole-3-pyruvate with auxin biosynthetic enzymes YUCs/YUCCAs (Zhang et al., 2021). In the rice variety *indica*, *OsDNR1* expression is lower compared to *japonica*, and auxin content is hence higher: OsARFs are hence more active in promoting nitrogen metabolism and grain yield, and repressing tillering (Zhang et al., 2021). Interestingly, OsWRKY23 directly promotes the expression of *OsDNR1,* and thus, *indica* plants with lower OsWRKY23 levels also present enhanced nitrate use efficiency and grain yield under low nitrate conditions (Zhang et al., 2025). Similarly, OsRNR10 is an F-box protein that stabilizes OsDNR1 through its monoubiquitination, and hence, nitrate use efficiency and grain yield are lower in *japonica* plants, which express *OsRNR10* more than *indica* plants (Huang et al., 2023b). Practically, *japonica* and *indica* rice varieties display alleles of *DNR1*, *WRKY23*, and *RNR10* that differ in their promoter sequences: this thereby likely explains the differential expression of these genes in *indica* versus *japonica* rice varieties (Zhang et al., 2021, 2025; Huang et al., 2023b). As the variant alleles of *indica* are more beneficial under low nitrate conditions, their introgression from *indica* to *japonica* would improve their nitrate use efficiency and grain yield; because introgression is nonetheless time-consuming, current genome editing tools are preferred for their fast generation of novel alleles (Tao et al., 2025). This illustrates how transitioning to an agriculture using less nitrogen fertilizers may be facilitated by using auxin-accumulating crops.

With the total global food demand roughly expected to double from 2010 to 2050, enhancing production sustainability is more critical than ever (Van Dijk et al., 2021). Climate change poses significant additional challenges as it will drastically affect crop production through more frequent extreme events, altered seasonality, and increased pathogen pressure (Rezaei et al., 2023). In this view, the idea of breeding stress-resistant crops has widely emerged, with plant hormones largely suggested as key targets (Zhang et al., 2018c; Gupta et al., 2020; Rivero et al., 2022). Between these stress-resistant and high-yielding bred crops, however, a major question persists: how tolerant to stress are high-yielding crops, and how high-yielding are stress-tolerant crops? One cannot simply put aside that hormones do not simply mediate plant growth or stress responses, and thus that manipulating one hormonal pathway will likely have repercussions on both aspects. Moreover, plants rarely encounter a single stress, and combined stresses deeply modulate hormonal networks and responses. Low phosphorus, for instance, significantly impacts plant defense against herbivores through the JA-Ile pathway (Khan et al., 2016), whereas low soil levels of nitrate induce ABA signaling and thereby enable better drought tolerance (Ma et al., 2025a).

Beyond crop breeding, a number of studies have investigated the potential of plant hormones as chemical primers to enhance pathogen and abiotic stress resistance (Sako et al., 2021; Hönig et al., 2023). Post-application metabolism and side effects remain poorly explored, and the low stability of plant hormones also constitutes a hurdle for agricultural applications. In this context, the rational design of hormonal agonists — synthetic compounds mimicking the effects of endogenous bioactive hormones — may represent a promising alternative. For instance, *N*-coronafacyl *ent*-coronamic acid O-phenyloxime selectively enhances COI1–JAZ9/10 interactions, thereby enhancing fungal resistance without growth defects (Takaoka et al., 2018). Even more selective, a stereoisomer of coronatine only induces COI1-JAZ9 interaction (Hayashi et al., 2023). The ABA agonist opabactin, meanwhile, displays a 10-fold stronger activity than ABA itself, and its application significantly increases PYL2-mediated drought tolerance in various crops (Vaidya et al., 2019). A key feature and challenge of these agonist lies in their receptor specificity, which relates to their application purpose. Agonists should indeed be able to trigger responses in a wide range of monocot and dicot crops (Bono et al., 2025). Receptor specificity, however, also drives agonist effects on plants, and this poses the question of developing broad versus narrow-spectrum agonists. In other words, do we wish to develop, for instance, an ABA agonist that almost exclusively acts on drought resistance, or do we rather prefer an agonist that has a range of action as close as possible to ABA? Further work is needed to assess growth tradeoffs related to this issue and thus to refine receptor specificity.

In some parts of the world, though, yield improvement alone is insufficient. In sub-Saharan Africa, especially, massive losses in crop yields are caused by highly adapted plant–parasitic weeds like *Striga* spp., and efficient methods for their management are lacking (Jamil et al., 2021). As these weeds germinate in response to SLs (Nelson, 2021), current developments for their sustainable management mostly exploit SLs. In sorghum, for instance, knocking out SL carriers that mediate SL export outside roots effectively reduces *Striga* parasitism by lowering SL exudation (Shi et al., 2025). Besides, the idea of a "suicide germination" with SLs agonists has emerged as a promising control option (Jamil et al., 2021). This has led to the development of agonists like sphynolactone-7 and 4a, which induce weed germination as efficiently as the synthetic GR24, without triggering typical SL responses in non-parasitic plants and with little effect on

mycorrhizal fungi (Uraguchi et al., 2018; Wang et al., 2022a). While field trials with various crops and parasitic weeds are needed to validate the efficiency of such agonists, this illustrates how this strategy could significantly enhance crop production in infested regions.

In a nutshell, plant hormonal networks represent a powerful and versatile toolkit that we could manipulate in many ways to improve crop production, and thereby meet the food demand of a rapidly growing global population despite climate change. Rather than relying on a single approach, integrating both genetic and chemical hormonal strategies is likely to yield the most impactful results. Importantly, though, improving crop yield and stress tolerance also extends beyond prospects centered around plant hormones. Beneficial microorganisms, in particular, hold great promise for enhancing nutrient acquisition and defense (Compant et al., 2025; Copeland et al., 2025). These approaches, based on hormonal tools versus microbial partnerships, should not be seen as competing strategies, but rather as complementary solutions to tackle agricultural challenges. Because hormones also mediate plant–microbe interactions (Eichmann et al., 2021), understanding how hormonal modifications through breeding and/or agonist application influence the plant microbiome will be essential. This reinforces the importance of fundamental research, which directs the development of future applied solutions. Ultimately, deciphering the multilayered regulatory roles of plant hormones provides not only deeper insights into plant biology but also a conceptual and practical framework for shaping the future of agriculture. As we face unprecedented environmental and societal challenges, harnessing hormonal networks will be central to reinventing an agriculture that is more productive, resilient, and sustainable.

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CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

AUTHOR CONTRIBUTIONS

L.V., M-L.F., and M.G. conceptualized the manuscript. L.V. wrote the original draft. L.V., M.G., and M-L.F. revised and edited the manuscript. L.V. and M.G. prepared the figures. All authors read and approved the content of the manuscript.

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article: http://onlinelibrary.wiley.com/doi/10.1111/jipb.70238/suppinfo

Table S1. Abbreviated names of proteins and peptides mentioned in the main text and their associated full names, ordered alphabetically