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REVIEW ARTICLE

Integrative role of hormonal crosstalk in plant growth regulation- A review

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Abstract

Plant growth regulators (PGRs), like auxins, cytokinins, gibberellins, abscisic acid, brassinosteroids, ethylene, jasmonates and strigolactones, are central managers of plant development and stress adaptation. Their actions are controlled by biosynthesis, translocation and hormonal crosstalk, which jointly enable precise regulation of growth from embryogenesis to decline. Recent studies uncover intricate spatial and temporal control of Plant Growth Regulator (PGR) biosynthesis, for example, auxins in meristems via YUCCA Flavin-Containing Monooxygenase (YUCCA) enzymes, cytokinins in root tips via Isopentenyltransferase (IPT), Abscisic Acid (ABA) in vascular parenchyma under drought via cis-Epoxycarotenoid Dioxygenase (NCED) and ethylene via ACC synthase/oxidase under stress. Directed transport, like phloem-mediated gibberellin movement, polar auxin transport and xylem-dependent ABA redistribution, establishes hormonal gradients essential for developmental patterning. Crosstalk among hormones, including ABA-ethylene synergy and auxin-cytokinin antagonism integrates environmental and endogenous signals to fine-tune growth-defence trade-offs and stress toughness. Emerging paradigms, like scaffold-mediated signalling and hormonal hormesis, further highlight the originality of regulatory mechanisms that organize growth with adaptive responses. Comprehending these networks offers avenues for biotechnological applications, such as engineering crops with improved growth and stress tolerance. Future research should emphasise single -cell hormone mapping, synthetic hormone circuits and the roles of non-canonical PGRs in niche signalling.

Keywords: epigenetic regulation; hormonal crosstalk; hormonal homeostasis; plant development; plant hormones; signal transduction; stress adaptation

Introduction

Plant growth regulators (PGRs), also known as phytohormones, are small signalling molecules that regulate nearly every aspect of plant life, from cell division and differentiation to whole-plant responses to environmental stimuli (1, 2). They act at nano- to micromolar concentrations to coordinate critical developmental processes, including seed germination, root and shoot growth, flowering and senescence (3). Classical PGRs-including auxins, cytokinins, gibberellins (GAs), abscisic acid (ABA), ethylene, brassinosteroids (BRs), jasmonates (JAs) and strigolactones (SLs)-exhibit both independent and synergistic roles in integrating growth and stress responses (4, 5). Auxins drive cell elongation and apical dominance, cytokinins promote cell division and shoot initiation, GAs enhance stem elongation and seed germination, ABA regulates stomatal closure and dormancy, ethylene mediates fruit ripening and leaf abscission and BRs support cell expansion and stress tolerance (6, 7).

The regulation of PGRs relies on biosynthesis, transport and crosstalk (8, 9). Biosynthesis is spatially and temporally controlled: auxins are mainly synthesized in shoot apical meristems through YUCCA-mediated tryptophan-dependent pathways, cytokinins are produced in root tips via IPT activity and modified by cytochrome P450 enzymes, ABA is synthesized in vascular parenchyma and guard cells through NCED enzymes under drought and ethylene arises from methionine via the Yang cycle with key roles of ACS (Aminocyclopropane-1-Carboxylic Acid Synthase) and ACO (Aminocyclopropane-1-Carboxylic Acid Oxidase) enzymes (10–13).

Directed transport mechanisms establish hormonal gradients that determine developmental patterning (14, 15). Auxins are transported polarly via pin-formed (PIN-formed (pin) proteins) and ABCB (ATP-Binding Cassette subfamily B transporters) transporters. Cytokinins move as inactive glucosides in xylem sap and are reactivated in shoots. ABA is redistributed through ion-trapping mechanisms, SLs are transported acropetally to regulate shoot branching and BRs diffuse passively due to their lipophilic nature. These gradients orchestrate processes such as apical dominance, gravitropism and vascular differentiation. Hormonal crosstalk allows intersecting pathways to synergise or antagonise, fine-tuning plant responses (16, 17). Auxin-cytokinin antagonism regulates root-to-shoot allocation, ABA-ethylene interaction balances drought and submergence responses and GA-DELLA-JA interactions coordinate growth-defence trade-offs. Further regulatory layers, including hormonal hormesis, epigenetic mechanisms, enhance signal precision and plasticity and scaffold-mediated signalling (e.g., AUX/IAA proteins recruiting SCF “Skp1–Cullin–F-box protein complex“ ubiquitin ligases) (18–20). In spite of extensive studies, key research gaps remain (21, 22). The functions of non-canonical PGRs like karrikins and phytomelatonin, synthetic hormone circuits and single-cell hormone dynamics are not fully elucidated. Comprehending these gaps is crucial for leveraging hormonal networks in plant engineering to enhance growth and stress resilience (Table 1).

This review seeks to: Synthesise current knowledge on plant growth regulators (PGR) biosynthesis, transport and crosstalk; highlight emerging regulatory mechanisms, novel hormonal paradigms and discuss implications for engineering plants with maximum growth and stress tolerance. By combining these aspects, the review offers a comprehensive network for awareness of plant hormonal regulation.

Biosynthesis of plant growth regulators

The biosynthesis of PGRs is tightly controlled at both temporal and spatial levels, ensuring that hormone synthesis is precisely coordinated with environmental fluctuations and developmental programs. Auxins, especially indole-3-acetic acid (IAA), are primarily synthesised in young expanding leaves and shoot apical meristems by tryptophan-dependent pathways. Amid these, the indole-3-pyruvic acid (IPyA) pathway symbolises the predominant route, where YUCCA flavin monooxygenases catalyse the transformation of tryptophan-derived intermediates into indole-3-pyruvic acid, subsequently decarboxylated to form IAA (23). Substitute biosynthetic routes, such as the indole-3-acetamide (IAM) pathway, manage under specific physiological contexts, participating in auxin homeostasis (24). Cytokinins, adenine-derived phytohormones synthesised primarily in root apices, are produced via a rate-limiting reaction catalysed by adenosine phosphate-isopentenyltransferase (IPT), which transfers an isopentenyl group to ATP (Adenosine Triphosphate) or ADP (Adenosine Diphosphate) to generate isopentenyladenine (iP) nucleotides. These intermediates are hydroxylated by cytochrome P450 monooxygenases, particularly CYP735A (Cytochrome P450 CYP735A), to yield biologically active trans-zeatin forms (6). Activation is further refined by the LONELY GUY (LOG) LONELY GUY “(Cytokinin-activating enzyme)” enzyme family, which converts inactive nucleotide precursors into free-base cytokinins (25).

GAs, a class of diterpenoid hormones, are produced from geranylgeranyl diphosphate (GGDP) by sequential oxidation reactions happening in plastids and the endoplasmic reticulum. Enzymes like GA20-oxidase (GA200x) and GA3-oxidase (GA3ox) catalyse the creation of bioactive GA and GA, whereas GA2-oxidase (GA2ox) mediates their inactivation, thereby maintaining hormonal equilibrium (21). Environmental signals, such as temperature and light, modulate GA biosynthesis by regulating the expression of these key enzymes. Abscisic acid (ABA) biosynthesis is strongly induced under abiotic stresses, particularly drought conditions. In vascular tissues and guard cells, the enzyme 9-cis-epoxycarotenoid dioxygenase (NCED) cleaves carotenoid substrates such as violaxanthin to generate xanthoxin, which is subsequently converted into ABA in the cytosol (7). ABA homeostasis is dynamically maintained through catabolic processes, notably via CYP707A (Cytochrome P450 CYP707A)-mediated hydroxylation that produces inactive ABA derivatives (26).

Ethylene production proceeds through the Yang cycle, beginning with the conversion of methionine to S-adenosylmethionine (SAM). ACC synthase (ACS) catalyses the formation of 1-aminocyclopropane-1-carboxylic acid (ACC), which is then oxidised by ACC oxidase (ACO) to release ethylene gas. The expression of ACS and ACO genes is transcriptionally enhanced by hypoxia, mechanical stress and developmental signals, enabling rapid hormonal responses (8). Brassinosteroids (BRs) are produced from the sterol precursor campesterol via oxidation reactions and multiple hydroxylation. Critical enzymes like DET2 (a steroid 5x-reductase) and CPD (CYP90A1) “Inherent Photomorphogenesis and Dwarfism (Cytochrome P450 CYP90A1)” lead to the production of castasterone, the instant precursor of brassinolide, the highly active BR form (27).

Jasmonates (JAs) derive from a-linolenic acid released from chloroplast membranes. By means of the sequential actions of lipoxygenase (LOX), allene oxide cyclase (AOC) and allene oxide synthase (AOS), α-linolenic acid is transformed into jasmonic acid, which is further linked to isoleucine to form the bioactive communication molecule jasmonoyl-isoleucine (JA-Ile) (28). Strigolactones (SLs), carotenoid-obtained hormones, are synthesised via the sequential cleavage of ß-carotene facilitated by D27 (β-carotene isomerase), CCD7 (Carotenoid Cleavage Dioxygenase 7) and CCD8 (Carotenoid Cleavage Dioxygenase 8) enzymes. These biosynthetic steps create SL precursors that manage branches of shoot and root system architecture, especially under nutrient-limited conditions (29) (Table 2).

Table 1. Non-canonical plant growth regulators and their functions

Compound	Function	Potential application
Phytomelatonin	Antioxidant, circadian rhythm modulator	Abiotic stress tolerance
Karrikins	Promotes seed germination	Wildfire regeneration
Peptide hormones	Cell-cell communication	Developmental patterning

Table 2. Biosynthesis pathways of major plant growth regulators (PGRs)

Plant hormone	Main biosynthetic pathway	Site of synthesis	Key enzymes
Auxins (IAA)	Tryptophan → IPyA → IAA	Shoot apical meristems, young leaves	YUCCA
Cytokinins	Adenine → iP → trans-zeatin	Root tips	IPT, CYP735A, LOG
Gibberellins (GAs)	GGDP → GA1/GA4	Plastids and ER	GA20ox, GA3ox, GA2ox
Abscisic acid (ABA)	Carotenoids → Xanthoxin → ABA	Vascular tissue, guard cells	NCED, CYP707A
Ethylene	Methionine → SAM → ACC → Ethylene	All tissues (under stress)	ACS, ACO
Brassinosteroids (BRs)	Campesterol → Castasterone → Brassinolide	Growing tissues	DET2, CPD
Jasmonates (JAs)	α-Linolenic acid → JA → JA-Ile	Chloroplast membranes	LOX, AOS, AOC
Strigolactones (SLs)	β-Carotene → Carlactone → SLs	Roots under nutrient deficiency	D27, CCD7, CCD8

Translocation of plant growth regulators

The biological activity of PGRs depends not only on their biosynthesis but also on their precise spatial reallocation within plant tissues. Auxin transport is a well-characterised example of oriented hormone movement, occurring via a polar transport system facilitated by PIN-FORMED (PIN) PIN-FORMED proteins·efflux carriers and ABCB (ATP-Binding Cassette subfamily B) transporters. The asymmetric positioning of PIN proteins at the plasma membrane establishes directional auxin fluxes that are fundamental for tropic growth reactions and organogenesis (9). This orientation is dynamically managed by phosphorylation through PINOID (PINOID kinase) kinase, whereas auxin itself reinforces transport capacity through transcriptional feedback that modulates PIN expression (30). Cytokinins are primarily transported via the xylem as dormant riboside forms, such as trans-zeatin riboside, permitting long-distance signalling from roots to shoots. Upon appearance in target tissues, these ribosides are transformed into bioactive free bases by ß-glucosidases or members of the LONELY GUY (LOG) enzyme family. Cellular uptake and redistribution are promoted by specific membrane transporters, comprising equilibrative nucleoside transporters (ENTs) and purine permeases (PUPs), which ensure regulated intracellular cytokinin availability (10).

Gibberellins (GAs) display bidirectional transport via the phloem, enabling coordinated growth regulation between sink and source tissues. The identification of NPF3.1 (Nitrate Transporter 1/ Peptide Transporter Family 3.1), a member of the NITRATE TRANSPORTER 1/PEPTIDE TRANSPORTER FAMILY in Arabidopsis, has supplied molecular insight into GA mobility, especially in mediating root-to-shoot transport (31). This managed movement contributes to systemic control of stem lengthening and developmental timing. Abscisic acid (ABA) allocation relies on a pH-dependent ion-trapping mechanism that allows stress-responsive reallocation. Under alkaline xylem conditions, ABA predominantly exists in its neutralised form (ABA), facilitating its movement in the direction of guard cells. Membrane-localised transporters further refine this process: ABCG25 (ATP-Binding Cassette G25 transporter) functions mainly in ABA efflux, while ABCG40 (ATP-Binding Cassette G40 transporter) mediates cellular uptake, as a group maintaining hormonal homeostasis via stress responses (32).

Ethylene, as a gaseous hormone, spreads readily across membranes; nevertheless, its precursor 1-aminocyclopane-1-carboxylic acid (ACC) serves as a movable signal enabling spatial management. ACC transporters, such as LYSINE HISTIDINE TRANSPORTER 1, facilitate localised ACC movement, especially under hypoxic conditions where root-derived ACC improves ethylene biosynthesis in aerial tissues (33). Strigolactones (SLs) are transported acropetally through the xylem, coordinating root-to-shoot interaction. ABCG transporters like PDR1 (Pleiotropic Drug Resistance 1 transporter) in Petunia mediate SL secretion into the rhizosphere, as a result, regulating shoot branching and facilitating symbiotic interactions with arbuscular mycorrhizal fungi (34)(Table 3).

Crosstalk and plant growth regulators effect

Hormonal crosstalk illustrates a central regulatory layer through which plants combine developmental programs with environmental signals. A well-characterized example is auxin-cytokinin antagonism, which creates root-shoot balance. Auxin inhibits cytokinin biosynthesis in root tissues by decreasing IPT gene expression, while cytokinins reciprocally suppress auxin transport through repression of PIN transporter expression (13). This mutual inhibition generates a regulatory module that determines root-shoot allometry, with auxin promoting root system expansion and cytokinins enhancing shoot apical meristem activity and aerial growth (35).

Conversely to antagonistic interactions, abscisic acid and ethylene often play a synergistic role under stress conditions. During drought, ABA stimulates stomatal closure to limit water loss, whereas ethylene modulates ABA homeostasis by enhancing CYP707A3-mediated ABA catabolism, as a result preventing excessive and prolonged growth inhibition (14). Under flooding stress, ethylene signalling stabilises ABA80x transcripts, inhibits ABA accumulation and promotes hypoxia tolerance. This context-reliant coordination ensures flexible adaptation to differing environmental constraints.

Gibberellin (GA)-jasmonate (JA) crosstalk illustrates a growth-defence trade-off mechanism regulated by protein-protein interactions. DELLA proteins, major inhibitors of GA signalling, physically communicate with JAZ (jasmonate ZIM-domain) repressors to combine these pathways. Elevated GA levels enhance DELLA degradation, freeing JAZ proteins to reduce JA-dependent defence responses. In contrast, JA signalling stabilises DELLA proteins, as a result restraining growth and favouring defence under biotic stress (15). This bidirectional modulation allows plants to actively allocate resources between development and protection.

Synergistic interactions are also obvious between brassinosteroids (BRs) and auxin throughout cell elongation. BR signalling improves auxin transport capacity by increasing PIN expression, whereas auxin stimulates BR biosynthesis through transcriptional regulation of DWF4, a rate-limiting enzyme in BR production (27). This beneficial feedback loop amplifies elongation responses, especially in hypocotyl tissues. Strigolactone (SL)-auxin interaction further illustrates feedback management within hormonal networks. Auxin stimulates the expression of CCD7 and CCD8, key genes in SL biosynthesis, enhancing SL production. As a result, SLs restrict auxin transport by enhancing PIN1 internalisation from the plasma membrane, hence suppressing shoot branching (18). This circuit offers a mechanistic basis for apical dominance and adjustable architectural plasticity (35).

At an elevated regulatory level, mitogen-activated protein kinase (MAPK) cascades integrate multiple hormonal inputs. MPK6 and MPK3 phosphorylate transcription factors like MYC2 (MYC2 transcription factor) and ARF7 (Auxin Response Factor 7), allowing coordinated signalling between ABA, JA and auxin pathways

Table 3. Transport mechanisms of plant growth regulators

Hormone	Transport mechanism	Directionality	Main transporters
Auxin	Polar active transport	Apical to basal	PIN, ABCB
Cytokinin	As glucosides in xylem sap	Root to shoot	LOG, ENTs, PUPs
Gibberellins	Phloem transport	Bidirectional	NPF3.1
ABA	pH-dependent ion trapping	Systemic	ABCG25, ABCG40
Ethylene	Diffusion (ACC transport)	Local and systemic	LHT1
Strigolactones	Xylem-mediated acropetal	Root to shoot	ABCG (e.g., PDR1)

through stress responses (36). During such kinase-mediated nodes, plants translate diverse hormonal signals into consistent transcriptional outputs, supporting the concept that crosstalk plays a role as a dynamic signalling network instead of isolated linear pathways (Table 4).

Crosstalk and hormonal balance

Hormonal equilibrium indicates a dynamic and finely tuned state in which the comparative abundance, sensitivity and signalling intensity of PGRs collectively determine developmental and physiological results. Rather than acting independently, hormones play a role in quantitative balances that determine cell fate and tissue identity. A classical demonstration is the auxin-to-cytokinin ratio. In callus cultures, increased auxin levels activate Auxin Response Factor (ARF)-mediated transcriptional programs that enhance root initiation, while higher cytokinin concentrations promote shoot regeneration via Arabidopsis Response Regulator (ARR)-dependent signalling pathways (16). Above tissue culture systems, this ratio also regulates vascular patterning, with auxin promoting xylem differentiation and cytokinin promoting phloem advancement (37). Like ratio-dependent control highlights how hormonal balance plays a role as a developmental switch.

A comparable governing module operates within the gibberellin (GA)-DELLA-jasmonate (JA) axis, which aligns growth-defence trade-offs. DELLA proteins, adverse regulators of GA signalling, physically communicate with JAZ (Jasmonate ZIM-domain) inhibitors and modulate the activity of MYC2, a central JA-responsive transcription factor. When GA levels increase, DELLAs are targeted for degradation via the 265 proteasome, alleviating repression and allowing growth-related processes. Conversely, JA signalling stabilises DELLA proteins, restricting elongation growth and prioritising defence reactions under herbivore or pathogen attack (15, 38). This mutual modulation exemplifies how the balance between hormonal pathways decides resource allocation.

The physiological significance of hormonal balance becomes especially evident in mutant phenotypes. The abal mutant, lacking in ABA biosynthesis, reduces impaired stomatal closure and increased drought sensitivity, demonstrating the vital role of ABA in water stress adaptation (7). Likewise, ein2 mutants, defective in ethylene signalling, fail to display the trait triple response and show modified senescence patterns (39). In the case of max2, which is unresponsive to strigolactones, excessive shoot branching occurs because of disrupted repression of cytokinin activity, indicating a breakdown in architectural regulation (18). These genetic disruptions jointly underscore the requirement of systemic hormonal coordination (Table 5).

Hormonal homeostasis is therefore sustained via multilayered feedback mechanisms and cross-regulatory circuits. Like, auxin can promote its own biosynthesis via induction of YUCCA genes, whereas ABA can suppress GA biosynthetic pathways to modulate growth under different kinds of stress (40). Like interconnected feedback loops guarantee that hormonal signalling remains adaptable yet equilibrated, enabling crops to maintain developmental stability while responding flexibly to environmental challenges.

Scaffold of plant growth regulators

Scaffold proteins role as organisational platforms that spatially and chronologically assemble signalling components, as a result enhancing pathway specificity, signal fidelity and controlling efficiency. In auxin signalling, AUX/IAA proteins function as central scaffold elements by forming heterodimers with Auxin Response Factor (ARF) transcription factors. Upon auxin awareness, AUX/IAA proteins recruit the SCF^TIR1/AFB ubiquitin ligase complex, resulting in their polyubiquitination and subsequent degradation through the 265 proteasome. This breakdown releases ARFs to stimulate auxin-responsive gene expression (41). Structural analyses have shown that conserved degron motifs within Domain II of AUX/IAA proteins are vital for TIR1 binding, whereas phosphorylation events, such as those mediated by MPK14, can stabilise AUX/IAA proteins under different kinds of stress conditions, thereby fine-tuning auxin responsiveness (42).

In ethylene signalling, the scaffold kinase CTR1 (Constitutive Triple Response 1) serves as an essential regulatory node that physically connects ethylene receptors, such as ETR1 (Ethylene Response 1), to subsequent signalling modules. Under ethylene-free conditions, CTR1 phosphorylates EIN2 (Ethylene Insensitive 2), avoiding its proteolytic cleavage and nuclear translocation. Ethylene perception suppresses CTR1 activity, allowing EIN2 processing and stimulation of downstream transcriptional responses (39). Similar scaffold-based architecture operates in brassinosteroid (BR) signalling, where Brassinosteroid Signalling Kinases (BSKs) connect the membrane-localised receptor BRI1 with the BSU1 phosphatase. This interaction promotes dephosphorylation and enhances BZR1/BES1 transcription factors, as a result propagating BR-dependent gene expression (43).

Jasmonate (JA) signalling similarly depends on scaffold-mediated organisation. JAZ proteins role as repressory scaffolds that bind both MYC2 transcription factors and the COI1-JAZ co-receptor complex. The bioactive bind JA-Ile promotes COI1-JAZ interaction, triggering ubiquitination and breakdown of JAZ proteins and therefore releasing MYC2 to activate JA-responsive genes (28). Significantly, scaffold specificity restricts unintended

Table 4. Examples of hormonal crosstalk

Crosstalk type	Hormones involved	Physiological outcome	Reference
Antagonism	Auxin – Cytokinin	Root-shoot balance	(13)
Synergy	ABA – Ethylene	Submergence/drought balance	(14)
Integration	GA – JA – DELLA	Growth-defense balance	(15)
Synergy	BR – Auxin	Hypocotyl elongation	(27)
Antagonism	SL – Auxin	Branching inhibition	(18)

Table 5. Hormonal mutants and their phenotypes

Mutant	Deficiency/resistance	Phenotypic effect
aba1	ABA-deficient	Impaired stomatal closure; drought sensitivity
ein2	Ethylene-insensitive	No triple response;delayed senescence
max2	Strigolactone-insensitive	Excessive branching
cre1	Cytokinin receptor-deficient	Altered shoot meristem activity

signalling intersect; for example, JAZ12 (Jasmonate ZIM-domain protein 12) specifically recruits TOPLESS co-repressors to inhibit non-target transcriptional programs, as a result, minimising non-specific crosstalk (44). As a whole, these examples illustrate that scaffold proteins do not solely facilitate signal transmission but energetically shape signalling architecture. By organising sensor complexes, transcription factors, kinases and ubiquitination machinery into separate modules, scaffolds support both precision and flexibility, ensuring that hormonal responses remain context-dependent and firmly regulated (Table 6).

Is it crosstalk or hormonal balance?

The differentiation between hormonal crosstalk and hormonal balance lies mainly in the regulatory scale and the mechanistic scope. Crosstalk refers to direct molecular interactions between signalling pathways, typically taking place at the level of transcription factors, enzymes, or signalling precursors. A representative example is auxin-ethylene synergy through root hair development. Auxin stimulates ethylene biosynthesis by activating ACC synthase (ACS), as a result increasing ACC production, whereas ethylene reciprocally promotes auxin distribution via stabilisation of the PIN2 (PIN-FORMED 2) transporter (45). This bidirectional interaction functions locally within specific tissues, illustrating pathway-level integration. Conversely, hormonal balance represents a broader, system-wide balance in which relative hormone concentrations and biosynthetic functions are coordinated across the organism. Strigolactone-cytokinin antagonism in shoot branching demonstrates this concept. Strigolactones inhibit cytokinin biosynthesis by downregulating IPT gene expression, while cytokinins reciprocally reduce CCD7/8, key genes in strigolactone biosynthesis (11, 46). Instead of a single molecular interaction, this relationship indicates a quantitative adjustment of hormonal pools that decides overall plant architecture.

Significantly, crosstalk and hormonal balance do not act independently but coexist within integrated managing networks. Through seed dormancy, ABA-GA crosstalk includes direct molecular interactions between ABA-activated SnRK2 kinases and GA-regulated DELLA proteins, regulating transcriptional control of germination-related genes. At the same time, systemic hormonal balance assures a coordinated decrease in ABA levels and an increase in GA concentrations as germination proceeds (47). Comparable multilevel integration happens in light-regulated hypocotyl elongation, where restricted auxin-GA interactions are mediated by PIF (Phytochrome-interacting factor). transcription factors cross with broader brassinosteroid homeostasis to modulate growth reactions (48). In combination, these examples underscore that crosstalk supplies mechanistic specificity at the molecular level, while hormonal balance establishes quantitative integration at the whole-plant level. The integration of both dimensions enables plants to obtain precise yet responsive developmental regulation.

Crosstalk and hormonal hormesis

Hormesis, specified as a biphasic dose-response phenomenon in which minimal levels of a stimulus elicit advantageous adaptive effects while maximum levels become suppressive or damaging, represents a purified layer of hormonal regulation mediated by crosstalk among PGRs. This phenomenon demonstrates how quantitative variations in hormone concentration can shift physiological effects from growth enhancement to stress adaptation. Ethylene supplies a clear example of hormetic regulation. At low concentrations (about 0.1-1 ppm). Ethylene enhances root system development by promoting auxin biosynthesis and reinforcing polar auxin transport via stabilisation of the PIN2 transporter, as a result stimulating lateral root formation (45). On the other hand, elevated ethylene levels (>10 ppm) suppress primary root elongation via excessive activation of ERF1 (Ethylene Response Factor 1), which represses genes associated with cell expansion and growth (49). Thus, ethylene acts as a growth promoter or inhibitor depending on its concentration and signalling intensity.

Abscisic acid (ABA) likewise exhibits dose-dependent duality through drought responses. Sublethal ABA concentrations (0.1–1 µM) role as priming signals, such as RBOHF (Respiratory Burst Oxidase Homolog F) function and control reactive oxygen species (ROS) production. This moderate ROS surge activates antioxidant defence enzymes like catalase (CAT) and superoxide dismutase (SOD), promoting stress resilience (50). Nevertheless, excessive ABA accumulation (>10 µM) overwhelms antioxidant capacity, resulting in uncontrolled oxidative stress and cellular damage (51, 52). These different outcomes underscore the fine threshold between preventive signalling and toxicity.

Jasmonates (JAs) also follow a hormetic pattern. Moderate JA levels promote herbivore resistance through MYC2-dependent activation of defence genes, while sustained or elevated JA concentrations inhibit growth by stabilising DELLA proteins and reinforcing growth suppression (53). A similar concentration-sensitive response is noted with gibberellins (GAs). Optimal GA levels enhance seed germination via GID1 (GA-Insensitive Dwarf 1) receptor-mediated DELLA degradation, enabling cell expansion and meristem activation. In contrast, excessive GA disrupts root meristem maintenance, partly via hyperactivation of SCL3 (Scarecrow-Like 3), resulting in developmental imbalance (54). Together, these dose-dependent dynamics highlight the adjustable plasticity of hormonal networks. Hormesis allows plants to fine-tune signalling thresholds, switching between growth-enhancing and survival-oriented programs as stated by environmental intensity. This quantitative flexibility symbolises a crucial component of hormonal homeostasis and stress adaptability (Table 7).

Crosstalk and gene expression

Hormonal crosstalk eventually converges on transcriptional and epigenetic regulatory layers, where dynamic gene expression programs are confirmed and fine-tuned. At the transcriptional level, jasmonate (JA) and abscisic acid (ABA) signalling intersect via shared

Table 6. Scaffold proteins in PGR signaling

Hormone	Scaffold protein	Function	Effect on signaling
Auxin	AUX/IAA + SCF^TIR1	Targets repressors for degradation	Activates ARF transcription
Ethylene	CTR1	Links receptors to the MAPK cascade	Regulates EIN2 translocation
Brassinosteroid	BSK	Connects receptor to phosphatase	Activates BZR1/BES1
Jasmonate	JAZ	Binds MYC2 and COI1	Releases defense genes on degradation

Table 7. Hormonal hormesis: Biphasic responses

Hormone	Low dose effect	High dose effect	Biological outcome
Ethylene	Promotes root growth	Inhibits elongation (ERF1 activation)	Biphasic
ABA	Primes ROS defenses	Induces oxidative stress	Stress adaptation or damage
Jasmonates	Activates defense	Suppresses growth	Growth-defense trade-off
Gibberellins	Promotes germination	Disrupts meristem maintenance	Context-specific

transcription factors. MYC2 acts as a central integrator, binding to ABA-reactive elements (ABREs) within the enhancers of drought-inducible genes like RD29A (Responsive to Dehydration 29A) and COR15A (Cold-Regulated 15A), while simultaneously activating JA-responsive genes such as VSP2 (Vegetative Storage Protein 2) via cooperation with MYB transcription factors (55, 56). This dual governing capacity enables organised activation of defence and stress-adaptive pathways. Conversely, auxin-cytokinin antagonism is indicated in competitive enhancer occupancy: cytokinin-activated ARR1 (Arabidopsis Response Regulator 1) inhibits auxin-responsive genes such as SAUR19 (Small Auxin-Up RNA 19) and GH3.5 (Gretchen Hagen 3.5) by restricting ARF5 access to shared regulatory regions (57). Such transcription factor rivalry exemplifies how hormonal equilibrium is translated into gene expression outcomes.

Beyond transcription factor interactions, epigenetic mechanisms supply an additional regulatory dimension that maintains or modulates hormonal reactions. Auxin signalling boosts histone acetylation via HAC1 (Histone Acetyltransferase 1), promoting chromatin relaxation at PIN gene loci and facilitating maintained auxin transport capacity (58). In contrast, ABA signalling recruits HD2C (Histone Deacetylase 2C) to deacetylate histones at enhancers like HAB 1 (Hypersensitive to ABA 1), as a result attenuating ABA-responsive gene expression via chromatin condensation (59). DNA methylation dynamics further combine hormonal cues: JA induces the ROS1 (Repressor of Silencing 1) demethylase, which removes inhibitory methylation marks from defence-related genes like PDF1.2 (Plant Defensin 1.2), promoting pathogen resistance (60). These chromatin-level adjustments ensure that hormonal signalling outputs can be quickly activated yet temporarily controlled (Table 8).

Non-coding RNAs apply a further layer of post-transcriptional and transcriptional modulation during hormonal networks. Auxin-induced miR393 targets TIR1/AFB transcripts, reducing auxin sensitivity via feedback regulation (61). Likewise, the long non-coding RNA DRIR (Drought-Induced RNA) responds with the ABA-responsive transcription factor AREB1 to potentiate drought tolerance interactions (62). Through such RNA-mediated mechanisms, the plants achieve fine-scale control over signalling amplitude and duration. As a whole, the integration of transcription factor networks, chromatin remodelling, DNA methylation and non-coding RNA regulation demonstrates that hormonal crosstalk expands beyond simple pathway intersection. In place of this, it operates via multilayered regulatory architectures that grant precision, adaptability and memory to stress of plants and growth responses (Table 9).

Crosstalk as a mode of action

Hormonal crosstalk plays a central role as a mechanism facilitating plants to interpret and react to complex environmental cues. Through hypocotyl elongation, light-auxin-gibberellin (GA) integration demonstrates this principle. Phytochrome B (phyB) detects light signals and represses PIF4 (Phytochrome-Interacting Factor 4), a transcription factor that alternatively activates YUCCA8 and GA200x1. As a result, enhancing auxin and GA biosynthesis (48). Auxin further stabilises DELLA proteins, supplying a feedback restraint on GA reaction and ensuring balanced hypocotyl growth under variable light conditions (63). This coordination demonstrates how localised crosstalk transforms environmental information into accurate developmental outputs.

Brassinosteroid (BR)-ethylene interactions illustrate crosstalk in skotomorphogenesis. BR-activated BZR1 transcription factors suppress EIN3 (Ethylene Insensitive 3), whereas ethylene signalling strengthens EIN3 to counteract BR responses. This reciprocal regulation fine-tunes apical hook generation and hypocotyl curvature during etiolation (64). Similarly, cytokinin-strigolactone (SL) antagonism controls shoot branching: cytokinin-activated ARR1 induces the transcription factor BRC1 (Branching 1) to suppress axillary buds. .whereas SLs inhibit IPT expression to limit cytokinin biosynthesis, maintaining architectural balance (46).

Crosstalk also mediates adaptive responses under combined abiotic and biotic stresses. Drought and heat act synergistically to induce NAC019 (NAC Domain-Containing Protein 019), which integrates ABA and ROS signals to activate the heat shock protein HSP70, ensuring cellular protection under extreme conditions (17, 65). Likewise, salinity stress and pathogen challenge converge on WRKY53 (WRKY Transcription Factor 53), a transcription factor phosphorylated by JA-activated MAPK6, which enhances expression of defence genes such as PR1 (Pathogenesis-Related 1), coordinating tolerance to multiple stressors (66, 67).

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Table 8. Epigenetic regulation by plant hormones

Hormone	Epigenetic mechanism	Target gene/process	Effect
Auxin	Histone acetylation via HAC1	PIN loci	Enhances auxin transport
ABA	Histone deacetylation via HD2C	HAB1 promoter	Suppresses ABA signalling
Jasmonate	DNA demethylation via ROS1	PDF1.2	Activates pathogen defense

Table 9. Non-coding RNAs mediating hormonal crosstalk

ncRNA	Target/Interaction	Function in hormonal signalling
miR393	Targets TIR1/AFB mRNAs	Suppresses auxin signalling
lncRNA DRIR	Interacts with AREB1 (ABA)	Enhances drought tolerance

integrate growth and environmental inputs, linking restricted molecular interactions with systemic regulatory results. By coordinating hormone biosynthesis, signalling and transcriptional networks, crosstalk permits plants to achieve maximum growth, architecture and stress resilience concurrently (Table 10).

Conclusion

The organised biosynthesis, transport and crosstalk of PGRs form a combined regulatory network that supports plant development and environmental flexibility. Rather than acting as separate signals, cytokinins, auxins, abscisic acid (ABA), gibberellins, brassinosteroids (BRs), ethylene, jasmonates (JAs) and strigolactones (SLs) function within a dynamic system in which positionally controlled biosynthesis, vectorial transport and mutual pathway interactions together determine physiological consequences. Hormonal gradients established via mechanisms like PIN-mediated auxin transport and stress-induced ABA redistribution translate environmental inputs into coordinated growth reactions. A central understanding of this review is that hormonal crosstalk and systemic balance operate at supplementary scales. Local pathway interactions, like auxin-cytokinin inhibition in root-shoot patterning or ABA-ethylene coordination through stress-mediated precise molecular reactions, whereas broader regulatory modules, such as GA-DELLA-JA axis and SL-cytokinin feedback loops, preserve whole-plant balance. Scaffold-mediated signalling complexes, moreover, refine specificity, guaranteeing accurate transcriptional reprogramming in spite of fluctuating internal and external signals. Emerging concepts like hormesis and epigenetic modulation show additional layers of plasticity inside PGR networks, emphasising how quantitative thresholds and chromatin-level management fine-tune adaptability. Jointly, these mechanisms locate hormonal crosstalk not solely as pathway interaction but as a systems-level framework integrating growth and development, stress toughness and environmental sensing. Prospective research should emphasise deciphering hormone dynamics at single-cell clarity, engineering synthetic and controllable signalling circuits, elucidating the functions of non-canonical PGRs and translating network-based understandings into climate-resilient plant design. Enhancing from pathway-centred to network-oriented comprehension of plant growth regulators (PGR) regulation will be fundamental for developing precision breeding and bioengineering strategies capable of sustaining agricultural productivity within changing climatic conditions.

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Table 10. Hormonal crosstalk during combined environmental stress

Stress combination	Hormonal components	Key regulators	Integrated response
Drought + Heat	ABA + ROS	NAC019	Activates HSP70 expression
Salinity + Pathogens	JA + MAPK6	WRKY53	Induces PR1 gene expression

Authors' contributions

YSS, AFZA, AFAA and OHMA developed the concept and wrote the first draft. SAAS, MSA, TFF and MAO collected literature and contributed to writing. AAA, ZKK and AMN revised and edited the final manuscript. All authors read and approved the final manuscript.

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