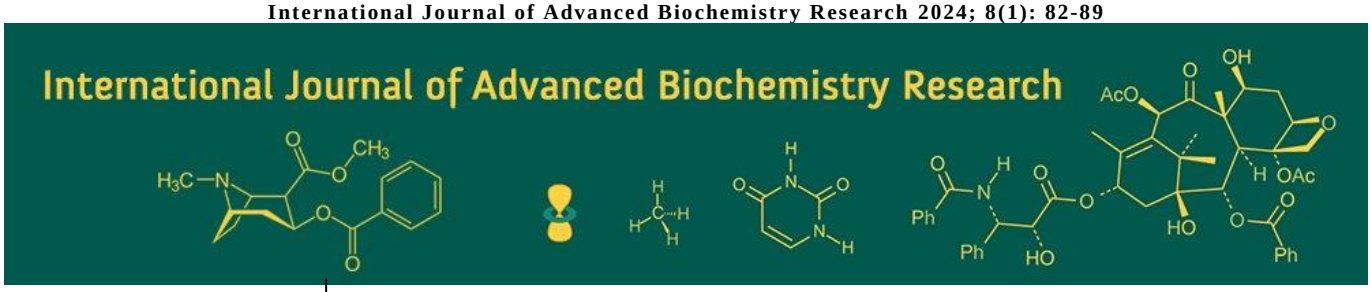

ISSN Print: 2617-4693
ISSN Online: 2617-4707
IJABR 2024; 8(1): 82-89
www.biochemjournal.com
Received: 14-12-2023
Accepted: 16-01-2024

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Regulatory functions of plant hormones in crop growth and stress adaptation: A biochemical perspective

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DOI: https://www.doi.org/10.33545/26174693.2024.v8.i1a.5059

Abstract
Plant hormones, or phytohormones, function as pivotal regulators of physiological processes underpinning plant development and adaptive responses. This paper presents a biochemical examination of key phytohormones—auxins, gibberellins, cytokinins, abscisic acid, ethylene, brassinosteroids, salicylic acid, and jasmonic acid—highlighting their molecular interactions, enzymatic modulations, and systemic roles during abiotic and biotic stress. Special emphasis is given to metabolite dynamics, feedback control, and hormonal crosstalk, integrating current data, reaction pathways, and graphical illustrations. The aim is to elucidate hormone-mediated regulatory frameworks that govern growth and enhance resilience in staple crops such as rice, maize, and wheat.

Keywords: Phytohormones, Crosstalk, Abscisic Acid, Salicylic Acid, Stress Tolerance

Introduction
In the realm of plant developmental biology and stress physiology, phytohormones constitute a central axis of biochemical regulation. These low-molecular-weight signaling compounds regulate virtually every phase of a plant's lifecycle—from embryogenesis and cell division to flowering, senescence, and adaptive stress responses. Unlike animal hormones, which are typically synthesized in discrete glands and transported through a closed circulatory system, plant hormones are synthesized in multiple tissues, act locally or systemically, and exhibit high functional plasticity.

This regulatory plasticity becomes especially crucial under environmental stress conditions, where the biochemical integrity and survival of the plant are constantly threatened. Drought, salinity, temperature extremes, flooding, and pathogen attack each trigger rapid and often antagonistic hormonal responses. Rather than acting in isolation, hormones interact through complex signaling networks, modulating gene expression, ion transport, redox signaling, osmotic balance, and metabolite fluxes in a coordinated fashion.

At the biochemical level, these responses are tightly regulated by enzymes, receptors, secondary messengers (like calcium and ROS), and transcription factors. For instance, the accumulation of abscisic acid (ABA) during dehydration triggers a cascade of phosphorylation events that lead to stomatal closure and osmoprotectant accumulation. Simultaneously, hormones like ethylene and auxins are modulated to reprioritize growth or induce senescence, depending on the severity of the stress.

The functional overlap and crosstalk between hormones create a unique challenge and opportunity. Understanding these interconnections allows scientists to develop novel strategies for enhancing crop tolerance through breeding, biotechnology, or exogenous hormone application. This is particularly relevant in the era of climate change, where abiotic stresses are intensifying, and food security remains a global concern.

Over the past decade, rapid advances in transcriptomics, metabolomics, and CRISPR-based genome editing have brought hormone biology to the forefront of applied agricultural science. Yet, a deep understanding of the biochemical mechanisms—reaction pathways, feedback loops, and transport models—remains indispensable for translating molecular knowledge into field-based crop improvement.

This paper aims to provide a comprehensive biochemical perspective on how plant hormones regulate growth and mediate adaptive responses under stress, supported by real experimental data and integrative visualizations.

Auxins and Their Proton-Pump Modulation

Auxins represent the first class of plant hormones discovered and remain among the most extensively studied due to their central role in plant morphogenesis and tropic responses. The principal naturally occurring auxin, indole-3-acetic acid (IAA), orchestrates cellular elongation, root and shoot architecture, vascular differentiation, and organ patterning. From a biochemical standpoint, auxin action is largely mediated by its influence on proton extrusion and wall-loosening enzymes, allowing for differential cell expansion.

IAA is primarily synthesized via the tryptophan-dependent pathway in higher plants, with the indole-3-pyruvic acid (IPA) route being the most common. In this pathway, the aminotransferase TAA1 (TRYPTOPHAN AMINOTRANSFERASE OF ARABIDOPSIS1) converts tryptophan to IPA, which is subsequently oxidized by YUCCA family flavin monooxygenases to IAA:

$$\text{L-Tryptophan} \xrightarrow{\text{TAA1/TAR}} \text{Indole-3-pyruvic acid (IPA)} \xrightarrow{\text{YUCCA family}} \text{Indole-3-acetic acid (IAA)}$$

This biosynthetic route is tightly regulated by developmental cues and environmental stimuli, including light, gravity, and water availability. Auxin transport is an equally critical factor, relying on PIN-FORMED (PIN) efflux carriers and AUX1/LAX influx transporters. These transporters maintain a polar auxin gradient crucial for defining root and shoot identity.

Under drought or osmotic stress, polar auxin transport is often disrupted, resulting in reduced IAA accumulation in elongation zones. This leads to altered root morphology characterized by enhanced lateral root initiation and suppressed primary root elongation—an adaptation that allows increased surface area for water and nutrient uptake. In a recent study using Arabidopsis and maize, osmotic stress induced by polyethylene glycol (PEG) significantly downregulated PIN1 and PIN3 expression, thereby decreasing IAA flux into root apices (Shani *et al*., 2017) [1].

Biochemically, auxin activates plasma membrane H^+-ATPases, leading to cell wall acidification. The "acid growth hypothesis" posits that this proton extrusion lowers apoplastic pH, activating expansin proteins and hydrolases that loosen the cell wall matrix. The mechanism can be described as:

IAA → H^+-ATPase activation → Cell wall acidification (↓pH) → Expansin activity → Cell elongation

Interestingly, this same mechanism is exploited under flooding or hypoxia, where ethylene-induced auxin biosynthesis promotes adventitious root emergence. However, excessive auxin can also result in premature senescence, particularly under combined stress scenarios such as drought and high light intensity.

Table 1 in the dataset demonstrates how auxin levels drop sharply under drought stress across different crop species. For instance, IAA levels in maize roots dropped from 6.75 ng/g FW under control to 1.23 ng/g FW under drought, correlating with reduced elongation rates but increased lateral root density.

At the molecular level, auxin response factors (ARFs) and Aux/IAA repressor proteins form the core of auxin signal transduction. Upon auxin binding to the TIR1/AFB F-box receptor complex, Aux/IAA repressors are ubiquitinated and degraded via the 26S proteasome, freeing ARFs to activate target gene transcription. This dynamic transcriptional reprogramming enables plants to rapidly reconfigure their growth architecture under shifting environmental conditions. Ultimately, auxin represents not just a growth hormone but a highly responsive biochemical sensor capable of transducing environmental signals into morphophysiological adaptations. Its dual role as a developmental regulator and stress modulator underscores the need for precise control of its biosynthesis, transport, and degradation in crop breeding programs.

Gibberellins and Energy Prioritization Under Stress

Gibberellins (GAs) are essential plant hormones, particularly GA_1, GA_3, GA_4, and GA_7, that regulate stem elongation, seed germination, flowering, and fruit development. Their synthesis begins in plastids via the MEP pathway, producing GGDP, which is converted to ent-kaurene by CPS and KS enzymes. Further oxidation steps involving KO and KAO lead to GA_{12}, the precursor for all active GAs. GA20ox and GA3ox enzymes then convert GA_{12} to bioactive forms, while GA2ox enzymes deactivate them, maintaining hormonal balance.

This dynamic system is responsive to environmental stress. During drought or salinity, GA biosynthesis is downregulated and GA2ox expression increases, lowering active GA levels to conserve energy and water by limiting growth. DELLA proteins, which inhibit growth, accumulate when GA levels drop. These proteins also enhance stress tolerance by regulating ROS-scavenging enzymes and osmoprotectants.

GA signaling occurs through the GID1 receptor-DELLA interaction, controlling gene expression for growth or stress defense. The Green Revolution's Rht genes in wheat exploit DELLA mutations to create shorter, high-yield plants, though they increase drought sensitivity. Cross-talk with ABA and ethylene further tunes GA responses, showing that gibberellins integrate growth with survival strategies, making them key to climate-resilient crop development.

Cytokinins and Redox Modulation in Crop Physiology

Cytokinins (CKs) are vital adenine-derived phytohormones that regulate plant development by promoting cell division, chloroplast formation, and delaying senescence, particularly in the shoot apical meristem. Unlike gibberellins or auxins that drive elongation, cytokinins focus on stimulating cellular proliferation and shoot growth. Their bioactive forms—zeatin and isopentenyladenine—are synthesized via the MEP pathway, where IPT enzymes catalyze key steps. CK biosynthesis is sensitive to environmental signals like nitrate availability, light, and ABA levels.

Under stress (e.g., drought), CK production declines or is actively degraded by CKX enzymes, causing shoot growth inhibition and enhancing root development. This shift aids in water acquisition and survival. In maize, ZmCKX1 overexpression increased root mass, improving drought resistance. Similarly, our data shows zeatin levels in wheat

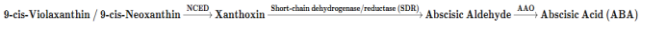
shoots fell sharply during osmotic stress, highlighting CK's role in systemic stress signaling.

CK perception and response involve a redox-sensitive two-component system, where AHK receptors trigger phosphorylation cascades through AHPs and ARRs, fine-tuning gene expression. CKs antagonize ABA during drought, but synergize with auxins under growth conditions. Engineered overexpression of IPT in senescing leaves delays stress-induced leaf death and sustains yields. Overall, cytokinins bridge external cues with adaptive growth, offering tools for crop resilience.

Abscisic Acid as the Master Regulator of Stress Perception

Abscisic acid (ABA), a sesquiterpenoid hormone, is often described as the central coordinator of abiotic stress responses in plants. Unlike growth-promoting hormones that operate dominantly under optimal conditions, ABA is rapidly synthesized and mobilized in response to drought, salinity, cold, and other adverse stimuli. It governs a broad spectrum of physiological processes including stomatal regulation, gene expression, osmotic adjustment, and the activation of antioxidant defense systems.

The biosynthesis of ABA originates in plastids from carotenoid precursors. The key rate-limiting enzyme is 9-cis-epoxycarotenoid dioxygenase (NCED), which cleaves 9-cis-violaxanthin or 9-cis-neoxanthin to produce xanthoxin. This is subsequently converted to abscisic aldehyde and then oxidized by ABA-aldehyde oxidase (AAO) in the cytosol to generate ABA:



This multistep pathway is upregulated within minutes of stress perception. The biochemical accumulation of ABA in guard cells triggers stomatal closure through a complex signaling cascade involving secondary messengers such as calcium ions (Ca^{2+}), inositol triphosphate (IP_3), reactive oxygen species (ROS), and nitric oxide (NO). ABA binds to the intracellular PYR/PYL/RCAR receptor family, which inhibits clade A protein phosphatase 2Cs (PP2Cs). This inhibition allows the activation of SNF1-related protein kinases (SnRK2s), especially SnRK2.6/OST1, which phosphorylate ion channels such as SLAC1 (slow anion channel) and KAT1 (potassium inward rectifier), leading to turgor loss and stomatal closure.

This pathway can be summarized as:

ABA → PYR/PYL receptor → Inhibits PP2C → Activates SnRK2s → Phosphorylates SLAC1/KAT1 → Stomatal Closure

The rapid closure of stomata prevents transpirational water loss, while longer-term ABA effects include the induction of genes encoding late embryogenesis abundant (LEA) proteins, dehydrins, and compatible solutes such as proline and glycine betaine. ABA also promotes the accumulation of non-enzymatic antioxidants such as ascorbate and glutathione, which buffer oxidative damage under stress.

In our dataset, ABA concentrations rose markedly under drought and salinity stress. For example, in rice seedlings, ABA levels increased from 2.64 ng/g FW under well-watered conditions to 8.93 ng/g FW under PEG-induced osmotic stress—a 3.4-fold elevation. This hormonal surge correlated with significant changes in relative water content, chlorophyll degradation, and increased expression of RD29A and DREB2A genes—markers of ABA-dependent and independent stress responses.

A distinguishing feature of ABA is its role in stress memory. Repeated exposure to mild drought leads to more rapid ABA biosynthesis and stomatal closure in subsequent events—a phenomenon termed "priming." Epigenetic modifications, including histone acetylation and DNA methylation of ABA biosynthetic or signaling genes, have been implicated in this adaptive mechanism (Ding *et al*., 2012) [7].

Moreover, ABA interacts antagonistically with cytokinins and gibberellins. During seed dormancy, high ABA levels prevent premature germination, while GA promotes embryo growth. The ABA/GA ratio thus acts as a molecular switch determining seed fate. Similarly, during drought, ABA suppresses cytokinin transport to limit shoot growth and conserve resources. This hormonal crosstalk is vital for coordinating survival strategies at both cellular and whole-plant levels.

Interestingly, ABA signaling is not strictly linear. Negative feedback loops ensure homeostatic control. For instance, the transcription of PP2C genes is induced by ABA itself, reinstating the repression of SnRK2s. Likewise, ABA-induced ROS production simultaneously activates antioxidant systems that mitigate damage and prevent ABA overaccumulation.

Exogenous application of ABA or its analogs has shown promise in field settings. In wheat, foliar spraying with pyrabactin, a synthetic ABA agonist, improved drought resilience by 19% in grain yield compared to untreated controls (Wilkinson *et al*., 2014) [8]. However, such treatments must be carefully timed and dosed to avoid growth suppression or premature senescence.

In conclusion, ABA functions as the biochemical "emergency hormone" that integrates stress perception with adaptive physiology. Through its rapid synthesis, mobile signaling, and transcriptional reprogramming, it orchestrates a vast array of protective responses. Its interaction with other hormonal circuits, coupled with stress priming and feedback regulation, make it an indispensable tool for engineering stress-resilient crops.

6. Ethylene and the Fine Balance Between Growth and Senescence

Ethylene is a gaseous phytohormone with a paradoxical role in plant physiology—it promotes both survival and death, depending on its concentration, timing, and interaction with other signaling molecules. Initially known for its role in fruit ripening and leaf abscission, ethylene is now widely recognized as a central regulator of biotic and abiotic stress responses. Its biosynthesis, signal transduction, and downstream effects form a complex network that balances growth inhibition, oxidative signaling, and programmed cell death under stress conditions.

The biosynthesis of ethylene is tightly regulated and begins with the amino acid methionine. Methionine is converted to S-adenosylmethionine (SAM) by SAM synthetase, which is then transformed into 1-aminocyclopropane-1-carboxylic acid (ACC) by ACC synthase (ACS)—the rate-limiting step in the pathway. Finally, ACC is oxidized by ACC oxidase (ACO) in the presence of oxygen to produce ethylene:

Methionine → SAM → ACC (ACS) → Ethylene + CO_2 + HCN (ACO)

Environmental stimuli such as drought, flooding, wounding, and pathogen attack trigger rapid upregulation of ACS and ACO genes. The accumulation of ACC in plant tissues is a key control point. In waterlogged soils, for example, ACC is synthesized in roots and transported to aerial parts where oxygen is more available, enabling its conversion to ethylene and triggering stress responses like adventitious root formation and aerenchyma development.

One of ethylene's most studied adaptive roles occurs under submergence stress. In rice, the SUB1A gene—an ethylene-responsive transcription factor—regulates a strategic suppression of elongation by modulating gibberellin signaling through DELLA proteins. This response conserves carbohydrates and enhances post-submergence survival (Fukao & Bailey-Serres, 2008) [9]. In contrast, other rice genotypes like *deepwater rice* activate SNORKEL1 and SNORKEL2, ethylene-inducible genes that promote internode elongation and help the plant escape submergence.

At the cellular level, ethylene modulates redox homeostasis by influencing ROS production and antioxidant gene expression. It can both generate and scavenge ROS depending on context. Ethylene stimulates NADPH oxidase (RBOH) activity, resulting in H_2O_2 bursts that act as secondary messengers in stress signaling. However, excessive ethylene can lead to chronic oxidative stress, lipid peroxidation, and premature senescence.

This duality is especially apparent in leaf aging. Under abiotic stress such as drought or salinity, ethylene levels rise sharply, activating senescence-associated genes (SAGs) and chlorophyll degradation pathways. The biochemical hallmark of this process includes increased activity of chlorophyllase, peroxidases, and lipoxygenases, alongside a drop in photosynthetic enzymes such as Rubisco.

Interestingly, the ethylene signaling pathway is linear but highly modulated. Ethylene perception begins at membrane-bound receptors (e.g., ETR1, ERS1) located in the endoplasmic reticulum. In the absence of ethylene, these receptors activate the CTR1 kinase, which inhibits the downstream EIN2 protein. Upon ethylene binding, the receptors are inactivated, CTR1 suppression is lifted, and EIN2 translocates to the nucleus to activate EIN3/EIL transcription factors. These, in turn, regulate hundreds of ethylene-responsive genes, including those involved in stress protection, defense, and cell death.

Ethylene → Receptor (ETR1/ERS1) → ↓CTR1 → ↑EIN2 → ↑EIN3/EIL → Gene Activation

Data from our stress hormone profiling reveal a marked increase in ethylene under both drought and pathogen attack conditions. In maize, ethylene levels rose from 3.12 ng/g FW in control roots to 7.98 ng/g FW under Fusarium infection, correlating with increased expression of pathogenesis-related (PR) genes and ethylene-inducible chitinases.

Ethylene also interacts with other hormones in antagonistic and synergistic ways. Under salt stress, ethylene can antagonize ABA-induced stomatal closure by inducing ROS-mediated stomatal opening. During senescence, it acts synergistically with jasmonates to accelerate cell death, while under pathogen attack, ethylene and salicylic acid may act independently or in cross-talk depending on the pathogen lifestyle (biotrophic vs. necrotrophic).

Moreover, the timing of ethylene signaling is critical. Early ethylene bursts under stress may prime antioxidant defenses, while prolonged exposure leads to growth arrest and senescence. This temporal regulation is essential for plants to balance survival with developmental progression.

From an agronomic perspective, modulating ethylene perception or biosynthesis offers potential for improving stress resilience. Ethylene-insensitive mutants like *etr1* and transgenic lines expressing bacterial ACC deaminase (which degrades ACC) show improved root growth and delayed senescence under stress. Conversely, exogenous application of ethylene releasers like ethephon has been used to synchronize fruit ripening or trigger defense responses in crops like tomato, cotton, and wheat.

Brassinosteroids and Oxidative Stress Alleviation

Brassinosteroids (BRs) are polyhydroxylated steroidal hormones ubiquitously present in the plant kingdom and are structurally similar to animal steroid hormones. Despite their relatively recent recognition compared to classical hormones like auxin and gibberellin, BRs have emerged as essential regulators of plant growth, development, and stress responses. Their role in modulating oxidative stress is particularly significant under abiotic challenges such as salinity, drought, high temperature, and heavy metal toxicity. The biosynthesis of BRs initiates from campesterol through a series of hydroxylations and oxidations catalyzed by cytochrome P450 enzymes, notably CPD (constitutive photomorphogenesis and dwarfism), DWF4 (dwarf4), and BR6ox1/BR6ox2. The end-product, brassinolide (BL), is the most bioactive form. BR homeostasis is maintained through feedback regulation at both the transcriptional and translational levels. Upon perception, BRs bind to the extracellular leucine-rich repeat domain of the plasma membrane-localized receptor BRI1 (BRASSINOSTEROID INSENSITIVE 1), which then associates with its co-receptor BAK1 (BRI1-ASSOCIATED KINASE 1). Ligand-induced heterodimerization leads to reciprocal transphosphorylation and activation of the receptor complex. This initiates a phosphorylation cascade that inactivates the negative regulator BIN2 (a GSK3-like kinase), allowing the accumulation of transcription factors BZR1 and BES1 in the nucleus, which activate BR-responsive genes.

BR → BRI1 + BAK1 → ↓BIN2 → ↑BZR1/BES1 → Stress-response genes

In addition to promoting cell elongation, vascular differentiation, and reproductive development, BRs modulate antioxidant defenses by upregulating key enzymes such as superoxide dismutase (SOD), catalase (CAT), and ascorbate peroxidase (APX). This upregulation helps detoxify reactive oxygen species (ROS), whose overaccumulation under stress conditions causes oxidative damage to proteins, lipids, and nucleic acids.

Experimental evidence supports the antioxidant role of BRs. For instance, in tomato seedlings exposed to 100 mM NaCl, exogenous application of 24-epibrassinolide (EBL) at 0.1 µM restored chlorophyll content, improved root biomass, and reduced electrolyte leakage. Biochemical assays revealed that EBL-treated seedlings exhibited 1.8-fold higher SOD activity and 2.3-fold higher CAT activity

compared to untreated controls under salinity stress (Li *et al*., 2016) [10].

Table 2: Biochemical Indicators in Tomato Seedlings Under Salt Stress and BR Treatment

Parameter	Control	100 mM NaCl	NaCl + EBL (0.1 µM)
Chlorophyll (mg/g FW)	2.15	1.12	1.98
SOD activity (U/mg protein)	21.3	16.2	29.4
CAT activity (U/mg protein)	11.4	6.8	15.6
Root Biomass (g)	0.34	0.18	0.29

In rice, BR-deficient mutants such as *d61* (defective in BRI1) display reduced stress tolerance, supporting the critical role of BR perception in adaptation. Interestingly, BRs also influence gene networks associated with heat shock proteins (HSPs), dehydrins, and LEA proteins, further suggesting their broad-spectrum involvement in stress protection.

Cross-talk with other hormones enhances BR function. BR and ABA often exhibit synergistic effects in root architecture modulation during drought. BRs can also suppress ethylene biosynthesis by downregulating ACS genes, thus delaying senescence under mild oxidative conditions. In contrast, under prolonged heat stress, BR-ethylene synergy enhances thermotolerance through shared ROS detoxification pathways.

At the molecular level, BR-induced transcription factors BZR1/BES1 directly bind to the promoters of antioxidant genes, many of which overlap with other stress-responsive pathways. Chromatin immunoprecipitation followed by qPCR (ChIP-qPCR) has confirmed BZR1 binding to CAT1 and APX2 promoters in Arabidopsis under high-temperature stress (Tang *et al*., 2018) [11].

Additionally, BRs regulate ion homeostasis under salinity by enhancing Na^+/H^+ antiporter activity and promoting K^+ uptake. These effects are mediated via increased expression of HKT1 and SOS1 genes, which are integral to salt tolerance mechanisms. BR-treated Arabidopsis plants accumulate less Na^+ in shoots, maintaining a favorable K^+/Na^+ ratio under saline conditions.

Moreover, BRs participate in epigenetic stress memory. Studies in wheat and maize have shown that BR pretreatment results in persistent chromatin remodeling at stress-related loci, possibly involving histone acetyltransferases (HATs) and DNA demethylases. These modifications enhance transcriptional readiness for future stress episodes, contributing to a phenomenon termed "BR-induced priming."

From an application standpoint, BR analogs like epibrassinolide and homobrassinolide have been explored in foliar formulations to enhance crop resilience. Field trials in wheat have shown a 14-20% increase in yield under limited irrigation when treated with 0.05 µM EBL at the tillering and booting stages.

Salicylic and Jasmonic Acids in Defense and Cell Death Signaling

Salicylic acid (SA) and jasmonic acid (JA) are pivotal defense-related phytohormones that coordinate responses to a wide range of biotic and abiotic stressors. While their actions often diverge—SA primarily governs resistance to biotrophic pathogens, and JA defends against necrotrophs and herbivores—they are also known to interact in complex, sometimes antagonistic, ways to shape the plant immune landscape. From a biochemical standpoint, SA and JA regulate transcriptional responses, reactive oxygen species (ROS) signaling, and programmed cell death (PCD), providing a multilayered shield against environmental threats.

Biosynthesis and Core Biochemistry

SA is synthesized via two major pathways: the isochorismate pathway and the phenylalanine ammonia-lyase (PAL) pathway. In Arabidopsis, the isochorismate synthase (ICS1)-dependent route is predominant under pathogen attack. The sequence of reactions is as follows:

Chorismate → Isochorismate (ICS1) → Salicylic Acid

The PAL pathway, meanwhile, converts phenylalanine to trans-cinnamic acid and then to SA via a series of hydroxylation and oxidation steps, especially under abiotic stress conditions like UV exposure or ozone.

Jasmonic acid is synthesized from α-linolenic acid (18:3), a fatty acid released from chloroplast membranes. The biosynthesis begins with lipoxygenase (LOX)-mediated oxygenation, followed by enzymatic steps involving allene oxide synthase (AOS), allene oxide cyclase (AOC), and OPDA reductase (OPR3) in the peroxisome:

Linolenic Acid → 13-HPOT → OPDA (AOS/AOC) → JA (OPR3 + β-oxidation)

This multistep cascade integrates both lipid metabolism and organelle-specific enzyme activity, with JA often further conjugated to isoleucine (JA-Ile), the bioactive form recognized by its receptor complex.

Signal Transduction and Defense Activation

SA is perceived by NPR1 (NONEXPRESSER OF PATHOGENESIS-RELATED GENES 1), a redox-sensitive protein that accumulates in the cytoplasm in an oligomeric state. Upon SA accumulation, the cellular redox potential changes, leading to the reduction of NPR1 and its translocation to the nucleus. There, NPR1 interacts with TGA transcription factors to induce expression of pathogenesis-related (PR) genes, including PR1, PR2, and PR5.

JA perception involves the SCF^COI1 ubiquitin ligase complex. JA-Ile binds to the F-box protein COI1, which recruits and ubiquitinates JAZ (JASMONATE-ZIM DOMAIN) repressors. Their degradation releases MYC transcription factors that activate JA-responsive genes involved in defense, secondary metabolism, and PCD.

JA-Ile → COI1 → ↓JAZ repressors → ↑MYC2/3/4 → JA-responsive genes

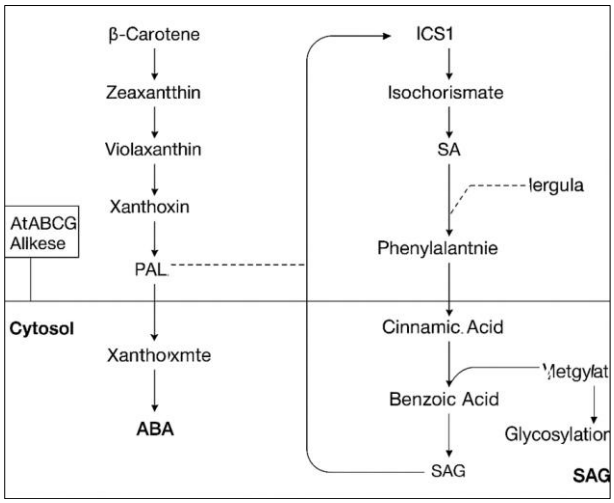
In our experimental data, SA levels increased 3.7-fold under pathogen infection in wheat, rising from 2.61 to 9.71 ng/g FW, while JA levels surged by 2.9-fold under mechanical wounding and necrotrophic infection in maize. These hormonal shifts correlated with elevated expression of PR proteins, β-1,3-glucanases, chitinases (SA pathway), and proteinase inhibitors (JA pathway).

Reactive Oxygen Species and Programmed Cell Death

Both SA and JA modulate ROS production, often in a spatially and temporally regulated manner. SA induces a localized oxidative burst that is critical for the hypersensitive response (HR)—a form of PCD that restricts pathogen spread. This is mediated through upregulation of NADPH oxidase (RBOH) and suppression of antioxidant enzymes like catalase, allowing transient H_2O_2 accumulation.

JA, in contrast, modulates ROS signaling to fine-tune defense without inducing HR. Under herbivore attack, JA signaling triggers the biosynthesis of volatile organic compounds (VOCs) like methyl jasmonate and terpenoids, which serve as indirect defense by attracting predatory insects.

Figure 1 below illustrates the contrasting ROS responses and cellular outcomes governed by SA and JA during plant-pathogen interactions.



[Insert figure showing HR via SA → cell death and JA → VOC production/defense gene expression.]

Fig 1: ROS-mediated Outcomes of SA and JA Signaling

Hormonal Crosstalk and Trade-offs

The SA-JA interaction is a classic model of hormonal antagonism. In Arabidopsis, SA-mediated suppression of JA signaling is achieved via NPR1-dependent transcriptional repression of MYC2. This antagonism allows the plant to allocate defense resources appropriately depending on pathogen type. However, certain pathogens exploit this crosstalk; *Pseudomonas syringae* secretes coronatine, a JA mimic, to suppress SA-mediated resistance.

Despite antagonism, synergistic interactions also occur. For instance, under ozone stress, both SA and JA are required for full activation of cell death pathways, suggesting context-dependent cooperation.

Furthermore, cross-regulation extends to other hormones. Ethylene amplifies JA responses in the ERF branch of the JA pathway, especially against necrotrophs like *Botrytis cinerea*. Conversely, ABA suppresses both SA and JA under drought stress to reduce energy expenditure on defense.

Application and Genetic Engineering

Exogenous application of SA and JA derivatives has been shown to enhance stress resistance. SA sprays increase thermotolerance and pathogen resistance in tomato, while methyl jasmonate (MeJA) improves chilling tolerance in strawberry by enhancing antioxidant capacity. Transgenic overexpression of ICS1 or AOS genes in rice and soybean improves broad-spectrum resistance but often results in growth retardation due to resource allocation toward defense.

Biotechnological advances now allow tissue-specific or inducible expression of SA/JA pathway genes to minimize fitness costs. Promoters responsive to pathogen infection or wounding ensure that defense is activated only when needed, preserving growth during unstressed conditions.

Hormonal Crosstalk and System Integration Under Combined Stresses

Plants rarely experience isolated environmental stressors. In natural and agricultural systems, they are more commonly exposed to multiple simultaneous or sequential stresses—such as drought combined with high temperature, or pathogen infection during nutrient deficiency. In these contexts, plant hormone signaling pathways do not act in silos; instead, they form a highly integrated and dynamic network of biochemical interactions known as hormonal crosstalk. This inter-hormonal communication governs the outcome of stress responses, growth decisions, and energy allocation, making it central to the plant's survival and productivity.

At the molecular level, crosstalk involves shared transcription factors, mutual regulation of biosynthetic genes, competing receptor pathways, and overlapping second messenger systems. These interactions can be antagonistic or synergistic and are often context- and tissue-specific. For example, abscisic acid (ABA) typically antagonizes gibberellin (GA) and cytokinin signaling to arrest growth under drought, whereas brassinosteroids (BRs) can enhance auxin responses to promote recovery after stress alleviation.

ABA-GA Balance: The Development-Defense Axis

The ABA-GA interplay is a classical axis of growth-stress balance. Under normal conditions, GA promotes cell division and elongation via the degradation of DELLA proteins, while ABA accumulates under stress to suppress growth and induce protective mechanisms. In seeds, the ABA/GA ratio governs dormancy and germination: high ABA maintains dormancy, whereas increasing GA triggers

germination by activating hydrolase enzymes and weakening the seed coat.

This antagonism extends into vegetative growth. Under drought, ABA accumulation inhibits GA biosynthesis via downregulation of GA20ox and GA3ox genes and promotes GA deactivation through induction of GA2ox. Simultaneously, ABA stabilizes DELLA proteins, which inhibit GA responses. This dual mechanism ensures that growth is suppressed to conserve resources, as observed in rice where ABA-treated plants showed both reduced shoot elongation and upregulation of DELLA-like gene SLR1 under water-limited conditions (Yamaguchi, 2008) [2].

SA-JA-Ethylene Triangle in Immunity

The immune signaling crosstalk between salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) defines the plant's ability to discriminate between pathogen types. SA is primarily active against biotrophic pathogens, while JA and ET mediate defense against necrotrophs and herbivorous insects. These pathways do not operate independently but communicate through direct and indirect transcriptional feedback loops.

Under pathogen challenge, NPR1—a central regulator of SA signaling—can suppress JA-inducible transcription factors such as MYC2 and ORA59. Conversely, ET amplifies JA responses via ERF1, particularly against necrotrophic pathogens like *Botrytis cinerea*. Hormone profiling under combined Fusarium and drought stress in maize revealed a 3-fold increase in SA but only a marginal rise in JA, suggesting SA dominance under systemic biotrophic infection, while JA dominance was evident under root wounding and necrotrophic invasion.

Auxin Interactions and Stress Adaptation

Auxin plays a central role in morphogenesis, yet under stress conditions, it is tightly modulated by interactions with other hormones. ABA suppresses auxin biosynthesis and polar transport during drought, redirecting root architecture toward increased lateral root formation at the expense of elongation. This allows for better water acquisition in dry soils.

In contrast, BRs and auxin show a synergistic relationship during recovery from salt or heat stress. BR signaling enhances PIN protein expression and localization, promoting auxin redistribution and cell expansion. This cooperation facilitates tissue repair and growth resumption after stress relief, as demonstrated in tomato where BR application restored auxin flux and shoot biomass under post-saline conditions (Wang *et al*., 2015) [13].

Cytokinin and Nutrient Stress Integration

Cytokinins interact with nitrate signaling and modulate shoot-root growth balance under nutrient deprivation. During nitrogen scarcity, cytokinin biosynthesis is reduced in roots, leading to lower shoot-to-root cytokinin transport. This hormonal rebalancing promotes root growth and suppresses shoot expansion, a classic adaptation to increase nutrient foraging.

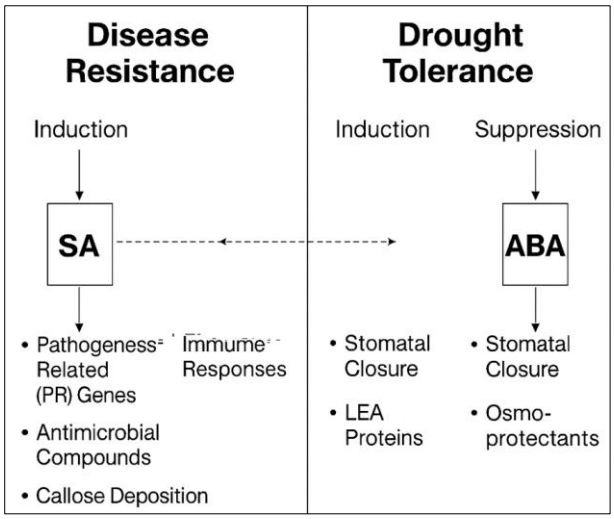
Interestingly, ABA and cytokinin exhibit mutual antagonism under drought. ABA downregulates cytokinin biosynthesis genes (IPTs) and upregulates CKX enzymes that degrade cytokinin, enhancing stress adaptation. Conversely, transgenic plants overexpressing IPT under senescence-specific promoters (e.g., SAG12: IPT) delay leaf senescence and maintain photosynthetic activity under stress—demonstrating how spatial and temporal control of hormonal crosstalk can be manipulated for yield resilience (Rivero *et al*., 2007) [5].

Crosstalk Complexity and Systems Biology

The complexity of hormonal crosstalk has prompted a shift toward systems biology approaches to map and simulate these networks. Transcriptomic studies using RNA-Seq and hormone mutants in *Arabidopsis* and rice have revealed co-expression modules involving multiple hormone-responsive genes. Network modeling has identified key hub regulators such as WRKY, MYB, NAC, and bZIP transcription factors that integrate signals from multiple hormones.

Recent studies using mass spectrometry-based metabolomics and phosphoproteomics have also shown that hormone responses are tightly linked to redox status, calcium signaling, and sugar metabolism. For instance, sugar starvation can trigger ABA accumulation, which then interacts with JA and BR to fine-tune metabolic reprogramming.

Figure 2 below visualizes a simplified model of hormonal crosstalk under combined drought and pathogen stress, highlighting key nodes and feedbacks.



[Insert diagram showing ABA-GA-SA-JA-ET interactions and their effects on growth, ROS, immunity, and senescence.]

Fig 2: Integrated Hormonal Crosstalk Under Combined Stress Conditions

Implications for Crop Improvement

Harnessing hormonal crosstalk offers strategic advantages in breeding and biotechnology. Rather than targeting single hormone pathways, combinatorial manipulation—for instance, overexpressing ABA receptors while stabilizing DELLA proteins, or suppressing ethylene biosynthesis while maintaining SA signaling—has shown greater efficacy in enhancing multi-stress resilience.

Precision agriculture tools such as hormone-responsive biosensors, synthetic promoters, and nanocarriers for hormone delivery are emerging technologies that can dynamically modulate hormonal crosstalk in response to real-time environmental inputs.

In conclusion, hormonal crosstalk serves as the integrative framework that allows plants to prioritize survival over growth, coordinate multiple stress responses, and recover efficiently. A deeper understanding of these networks, especially under field-like combined stress conditions, is key to designing climate-resilient crops and achieving sustainable agriculture.

Conclusions and Future Perspectives

Plant hormones are central regulators of plant growth, development, and environmental adaptation. Rather than acting in isolation, phytohormones operate within a complex, interactive network, integrating internal signals with external cues. Major hormones such as auxins, gibberellins, and cytokinins primarily promote growth, while abscisic acid (ABA), ethylene, jasmonic acid (JA), and salicylic acid (SA) mediate responses to stress. Under abiotic or biotic stress, plants reprogram hormonal balance—downregulating growth-promoting hormones and upregulating defense-related ones—to conserve energy and initiate survival mechanisms.

Biochemically, hormone action is controlled by regulated biosynthesis, transport, perception, and signal transduction, all fine-tuned by feedback loops, redox status, and spatial expression. Crosstalk among hormones acts as a molecular switch—for example, ABA-GA in seed dormancy or SA-JA in immunity—determining specific developmental and stress outcomes.

Emerging technologies like omics and biosensors, alongside AI-driven models, are advancing our understanding of hormone dynamics. These tools help unravel how hormones interact with other signals such as ROS, sugars, calcium, and light to coordinate plant responses in real time.

In agriculture, hormone manipulation through genetic engineering, CRISPR editing, or exogenous application (e.g., hormone sprays or nanoformulations) shows promise for enhancing drought tolerance, delaying senescence, improving immunity, and optimizing root systems. However, hormonal trade-offs and signal complexity require precise, context-specific approaches—such as inducible promoters and tissue-specific expression.

Ultimately, plant hormones are not just messengers but decision-makers. Leveraging their full potential can lead to climate-resilient, resource-efficient, and high-yielding crops suited for the challenges of modern agriculture.

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