# Effect of Hormones on Oxidative Stress in Plants

## Abstract

Reactive oxygen species (ROS) are inevitable by-products of aerobic metabolism in plants and serve as essential signaling molecules under both optimal and stress conditions. However, excessive accumulation of ROS leads to oxidative stress, causing cellular injury through lipid peroxidation, protein oxidation, and nucleic acid damage, ultimately compromising plant growth and productivity. Phytohormones, including abscisic acid (ABA), salicylic acid (SA), jasmonates (JA), ethylene (ET), auxins (IAA), cytokinins (CK), gibberellins (GA), and brassinosteroids (BR), function as central regulators of plant stress responses and play crucial roles in maintaining redox homeostasis. These hormones modulate ROS levels through multiple mechanisms: stimulation of apoplastic ROS production as secondary messengers, induction of antioxidant enzyme systems such as superoxide dismutase, catalase, and ascorbate peroxidase, and regulation of non-enzymatic antioxidant metabolites like ascorbate and glutathione.

Recent evidence highlights that hormonal interactions with ROS are highly context-dependent, with certain hormones simultaneously promoting ROS as signaling intermediates while also enhancing scavenging mechanisms to prevent cellular damage. Hormone crosstalk adds another layer of complexity, as synergistic or antagonistic interactions among ABA, SA, JA, BR, and other hormones finely tune antioxidant responses according to stress type, tissue, and developmental stage. Advances in molecular biology and omics approaches have revealed key signaling nodes, including NADPH oxidases, MAP kinases, and redox-sensitive transcription factors, that act as integrators of hormone–ROS networks. Furthermore, priming strategies with exogenous hormone application demonstrate potential for improving stress resilience by preparing antioxidant systems for rapid activation under adverse conditions.

This review synthesizes recent progress on the mechanistic roles of phytohormones in oxidative stress regulation, emphasizing their dual functions as inducers of ROS signaling and enhancers of antioxidant capacity. We discuss how hormone-regulated ROS balance influences stomatal regulation, chloroplast function, pathogen defense, and growth trade-offs. We also highlight translational opportunities for crop improvement through targeted manipulation of hormone pathways, alongside current limitations such as dose sensitivity, species-specific responses, and field-level variability. By identifying knowledge gaps and proposing integrative research directions, this review underscores the central role of hormones in redox biology and their potential in guiding sustainable agricultural practices under increasingly variable climate conditions.

**Keywords:** phytohormones, reactive oxygen species (ROS), oxidative stress, antioxidant defense, signaling crosstalk, stress tolerance

## 1. Introduction

### 1.1. ROS in plant biology: dual roles and contemporary view

Reactive oxygen species (ROS) occupy a central, paradoxical role in plant biology: at low-to-moderate concentrations they function as indispensable signalling molecules that regulate growth, development, and adaptive responses, whereas their over-accumulation causes oxidative damage and cell dysfunction (Mittler et al., 2022; Mansoor et al., 2022). The contemporary view emphasizes ROS as dynamic, compartmentalized signals whose spatial–temporal patterns are integrated with other signalling pathways to tune plant responses to changing environments (Mittler et al., 2022). This signalling-versus-toxicity balance requires tight regulation of ROS production, targeted transport, and efficient scavenging by enzymatic and non-enzymatic antioxidant systems (Mansoor et al., 2022; Murphy et al., 2022).

### 1.2. Sources, compartmentalization and the antioxidant network

Plants produce ROS in multiple subcellular compartments — chloroplasts, mitochondria, peroxisomes, the apoplast (via NADPH oxidases/RBOHs), and other organelles — and the site of production profoundly affects ROS signalling outcomes (Wang et al., 2024; Mansoor et al., 2022). Compartmentalization enables local signalling (for instance, chloroplast-derived ROS influencing nuclear transcription) while limiting collateral oxidative damage (Wang, 2024). Because extracellular ROS (eROS) are often generated by RBOHs, recent work has focused on routes by which eROS influence intracellular targets (e.g., conversion of superoxide to H₂O₂ and facilitated transport via aquaporins), highlighting complex transport and perception mechanisms that contribute to signaling specificity (Lee et al., 2023). Balance is maintained by a multi-layered antioxidant network (SODs, catalases, peroxidases, the ascorbate–glutathione cycle and small-molecule antioxidants), whose regulation is not simply constitutive but responsive to signalling inputs and stress conditions (Dvořák et al., 2021; Zandi & Schnug, 2022).

### 1.3. Hormonal control of ROS: direct regulation and cross-talk

Phytohormones modulate ROS production, location, and scavenging both as primary signalling partners and as modulators of stress adaptation networks. Abscisic acid (ABA), a canonical drought and osmotic stress hormone, rapidly triggers localized ROS production in guard cells (via RBOH activity and organellar sources) and coordinates antioxidant responses to effect stomatal closure and water-conservation responses (Postiglione & Muday, 2020). Conversely, brassinosteroids (BRs), auxin and cytokinin can either promote or attenuate ROS accumulation depending on context — for growth, development, or stress mitigation — frequently by regulating NADPH oxidases or antioxidant gene expression (Zhang et al., 2023; Wang, 2024). The interaction is bidirectional: ROS influence hormone biosynthesis, signalling sensitivity and downstream transcriptional networks, so hormone–ROS interactions are best conceptualized as dynamic crosstalk rather than simple linear pathways (Li et al., 2022; Zhang et al., 2023). This multilayered crosstalk underlies many adaptive phenotypes, from rapid stomatal responses to long-term adjustments in antioxidant capacity.

### 1.4. Methodological considerations and the need for integrative synthesis

Accurate interpretation of hormone–ROS relationships requires careful methodological choices: ROS species are chemically distinct, measurement approaches vary in specificity and artifact-proneness, and subcellular localization is critical for mechanistic inference (Murphy et al., 2022). The field has matured technically (genetically encoded sensors, targeted probes, organelle-specific reporters) but also needs standardized reporting and cautious interpretation to avoid conflating correlative ROS increases with instructive signalling events (Murphy et al., 2022; Wang, 2024). Equally, hormonal effects on ROS are often conditional on stress type, developmental stage, and species, which motivates comparative and mechanistic reviews to synthesize current findings and identify experimental gaps.

### 1.5. Scope and objectives of this review

Given the centrality of ROS–hormone interactions to plant resilience and the rapid growth of mechanistic and methodological studies in the last five years, this review aims to (i) summarize mechanisms by which major phytohormones regulate ROS generation, transport and scavenging; (ii) highlight reciprocal influences of ROS on hormone biosynthesis and signalling; (iii) evaluate measurement approaches and their interpretive limits; and (iv) identify unresolved mechanistic questions and practical implications for crop stress management. By integrating recent conceptual and technical advances, we seek to provide a roadmap for future experimental designs that will disentangle causation from correlation in hormone-driven oxidative signalling (Mittler et al., 2022; Wang, 2024).

## 2. ROS generation, scavenging, and signaling — a brief primer

Reactive oxygen species (ROS) are chemically distinct oxygen-derived molecules that span a continuum from relatively long-lived, diffusible species such as hydrogen peroxide (H₂O₂) to extremely short-lived radicals such as the hydroxyl radical (•OH). In plants ROS have a dual nature: at low to moderate concentrations they act as information-carrying second messengers that regulate growth, development and stress responses, whereas at high concentrations they inflict oxidative damage on macromolecules and membranes (Waszczak, Carmody, & Kangasjärvi, 2018; Mittler, Zandalinas, Fichman, & Van Breusegem, 2022). Modern guidelines emphasize that ROS should always be discussed with chemical specificity (which species, where, and how they were measured), because different ROS differ strongly in reactivity and mobility and therefore in signaling potential. In addition, biological outcomes are context- and compartment-dependent, and depend on local production versus removal rates.

### 2.1. ROS identities, chemistry and biological implications

Superoxide (O₂•⁻), hydrogen peroxide (H₂O₂), singlet oxygen (¹O₂) and hydroxyl radical (•OH) are the ROS most relevant to plant physiology. Superoxide is typically formed by one-electron reduction of O₂ and is rapidly dismutated (enzymatically or spontaneously) to H₂O₂, which because of its relative stability and ability to cross membranes (under some circumstances) is the ROS most often implicated in long-range and intra-cellular signalling (Smirnoff & Arnaud, 2019). By contrast, •OH is so reactive and short-lived that it is essentially incapable of acting as a selective signalling messenger; its role is primarily destructive. The chemical properties (reactivity, half-life, ability to cross membranes) therefore constrain which ROS can serve as mobile signals and which act locally. Importantly, these properties also dictate the experimental approaches appropriate for detection and interpretation (e.g., H₂O₂-specific genetically encoded sensors versus broad redox dyes).

### 2.2. Main cellular and apoplastic sources of ROS

Plants harbor multiple ROS-producing systems localized to different organelles and cellular compartments. In photosynthetic tissues chloroplast electron transport is a major source — under excess light electrons leak to O₂ at PSI and PSII leading to O₂•⁻ and subsequently H₂O₂ formation. Mitochondrial electron transport likewise generates superoxide at specific complexes during respiration. Peroxisomes produce H₂O₂ via oxidases linked to photorespiration and fatty acid metabolism. At the plasma membrane, plant NADPH oxidases (Respiratory Burst Oxidase Homologs, RBOHs) catalyse rapid apoplastic O₂•⁻ production that underlies the classical oxidative burst in defense and developmental signaling. Finally, apoplastic peroxidases and cell-wall oxidative enzymes also contribute to extracellular ROS (Smirnoff & Arnaud, 2019; Mittler et al., 2022). The spatial separation of these sources (apoplast vs. cytosol vs. organelle lumen) is central to how plants maintain specificity in ROS signaling.

### 2.3. Antioxidant and scavenging systems: keeping ROS in check

Because ROS can damage proteins, lipids and nucleic acids, plants possess elaborate antioxidant systems that operate at multiple spatial scales. Superoxide dismutases (SODs) convert O₂•⁻ into H₂O₂, which is then removed by catalases (primarily peroxisomal) or by peroxidases such as ascorbate peroxidases (APXs) coupled to the ascorbate–glutathione (AsA–GSH) cycle. Complementary systems include peroxiredoxins (PRXs), thioredoxins (TRXs), glutaredoxins (GRXs), glutathione pools and numerous small-molecule antioxidants (ascorbate, tocopherols, carotenoids, flavonoids). The combined operation of these enzymatic and non-enzymatic systems determines local ROS set-points and the time window during which a ROS signal persists; consequently, the same ROS pulse can lead to signaling in one cell type and oxidative damage in another depending on antioxidant capacity (Waszczak et al., 2018; Murphy et al., 2022).

### 2.4. Compartmentalization and controlled transport: how ROS reach their targets

Signal specificity often requires that ROS generated in one compartment influence molecular targets elsewhere without causing wholesale oxidation. Several mechanisms have been described. H₂O₂, because of its relative stability, is a leading candidate for controlled translocation; specific aquaporin isoforms (so-called peroxiporins) facilitate H₂O₂ diffusion across membranes and thus act as regulated conduits for ROS movement between compartments (Bienert & Chaumont, 2014). In guard cells, for example, the plasma-membrane aquaporin PIP2;1 mediates H₂O₂ influx that is required for ABA- and pathogen-triggered stomatal closure, illustrating biological relevance of aquaporin-mediated H₂O₂ transport (Rodrigues et al., 2017). More broadly, recent syntheses emphasize that extracellular ROS produced by RBOHs can be converted (e.g., by apoplastic SODs) and then enter the cytoplasm via aquaporins or be perceived in the apoplast via oxidant-sensitive receptor modules — a nuanced view that reconciles apoplastic ROS production with intracellular ROS signaling (Lee et al., 2023).

### 2.5. Molecular mechanisms of ROS signalling

At the molecular level, ROS often effect signaling through selective oxidation of protein cysteines (e.g., formation of sulfenic acid, disulfide bonds, S-glutathionylation) that alters protein activity, localization or interactions. Mapping studies have revealed hundreds to thousands of redox-sensitive cysteine sites in plant proteomes; specific sulfenylation events on transcription factors, kinases and metabolic enzymes have been tied to physiological readouts. In addition, redox relays mediated by peroxiredoxins and thiol proteins can amplify or direct H₂O₂ signals to specific targets, helping to generate specificity despite the diffusible nature of the oxidant. Thus, ROS signalling is not simply a matter of “more ROS = more output”, but of redox chemistry acting on defined protein thiols within controlled spatial and temporal contexts (Bo Wei et al., 2020; Smirnoff & Arnaud, 2019; Mittler et al., 2022).

### 2.6. Detecting ROS dynamics: sensors, caveats and best practice

Because biological interpretation depends critically on what species and compartment are measured, modern plant redox biology has relied increasingly on genetically encoded probes (e.g., roGFP2-Orp1, HyPer) that enable subcellular, dynamic readouts of H₂O₂ and thiol redox state. These tools have revolutionized our view of intracellular ROS dynamics and revealed previously unsuspected kinetics and organelle contributions to cytosolic ROS bursts. However, sensor readouts are affected by local antioxidant buffering, pH sensitivity and probe kinetics; consensus guidelines therefore stress careful controls, orthogonal measurement approaches, and explicit reporting of what (chemically) is actually measured (Nietzel et al., 2019; Murphy et al., 2022). Integrating genetically encoded sensors with biochemical assays and genetic perturbations remains the best path toward mechanistic clarity.

### 2.7. Summary and forward view

In sum, ROS in plants arise from multiple discrete sources, are removed by robust scavenging networks, and act as signals by targeted oxidation chemistry that is constrained by compartmentation and regulated transport. Understanding hormonal modulation of these processes (the topic of the main manuscript) requires keeping this biochemical and cell biological framework in view: hormones can alter source activity (e.g., RBOH activation), antioxidant capacity, transport routes (e.g., aquaporin gating) and the sensitivity of downstream redox-sensitive targets, thereby reshaping both the amplitude and the specificity of ROS signals.

## 3. Hormones as modulators of oxidative stress

3.1 Overview
Phytohormones shape how plants perceive and respond to oxidative stress by controlling both ROS production (e.g., via NADPH oxidases / RBOHs and organellar metabolism) and antioxidant defenses (enzymatic systems such as SOD, CAT, APX, and non-enzymatic pools such as ascorbate and glutathione). Hormone-driven modulation of ROS is therefore dual: hormones can *stimulate* ROS as signaling molecules to trigger defense or developmental programs, and they can *activate* antioxidant responses that limit ROS damage. The balance between those roles—spatially and temporally controlled—is central to plant stress tolerance and growth–defense trade-offs (Postiglione & Muday, 2020; Li et al., 2022).

3.2 Abscisic acid (ABA) — gating ROS for rapid responses
ABA is among the best-characterized hormones linking ROS signaling to stress responses. ABA rapidly induces apoplastic ROS via membrane-bound NADPH oxidases (RBOHs) during stomatal closure and stress signaling, and it also modulates organellar ROS (notably mitochondrial H₂O₂) that act as mobile second messengers to reinforce stomatal and cellular responses to drought (Postiglione & Muday, 2020; Postiglione & Muday, 2023). At the same time, ABA upregulates antioxidant enzymes in many tissues to limit collateral oxidative damage during sustained stress, and the pathway involves complex post-translational regulation of ROS-producing enzymes as well as transcriptional activation of antioxidant genes (Li et al., 2022). Together, these actions illustrate how ABA uses ROS both as a controlled signal (fast, localized) and as a process to be buffered by antioxidant systems over longer timescales.

3.3 Salicylic acid (SA) — ROS in defense and redox reprogramming
Salicylic acid is intimately tied to defense-associated ROS. SA potentiates the oxidative burst associated with pathogen perception and additionally alters cellular redox poise by regulating antioxidant networks and thiol-based signaling. SA can both enhance ROS accumulation in apoplastic spaces (to restrict pathogens) and shape intracellular antioxidant responses to avoid uncontrolled oxidative damage; this duality underlies SA’s central role in local and systemic defense as well as in cross-talk with other hormones (Saleem et al., 2021; Yang et al., 2023).

3.4 Jasmonic acid (JA) — modulating antioxidant capacity for tolerance
JA and its derivatives (MeJA, JA-Ile) commonly act to upregulate antioxidant machinery under biotic and abiotic challenges. JA signaling frequently increases expression and activity of enzymes involved in the ascorbate–glutathione cycle and related pathways, thereby contributing to tolerance against ozone, herbivory, heavy metals and drought. JA also coordinates metabolic shifts that supply reducing power (e.g., through enhancement of NADPH-producing pathways) to feed antioxidant systems, so its effect is largely protective when stress is chronic or when JA primes tissues for rapid future stress (Rehman et al., 2023).

3.5 Ethylene — ROS as a morphogenetic and stress signal
Ethylene commonly interacts with ROS during developmental processes (e.g., root hair initiation, organ abscission) and stress responses. Ethylene signaling can stimulate specific RBOH isoforms and peroxidases to generate localized H₂O₂ that drives cell fate changes or organ separation, while simultaneously modulating antioxidant gene expression to confine ROS spatially (Martin et al., 2022; Han et al., 2024). This hormone–ROS coupling explains ethylene’s capacity to coordinate both programmed developmental events and protective responses to environmental challenges.

3.6 Auxin — ROS in growth and developmental patterning
Auxin–ROS interplay is fundamental in developmental ROS patterning. Auxin gradients influence local ROS production (via peroxidases and RBOHs) to control cell expansion, cell wall loosening, and the formation of meristematic or organogenic primordia. Conversely, ROS can modulate auxin transport and signaling, leading to feedback loops that pattern root and shoot architecture. Recent transcriptomic and spatial studies highlight auxin-regulated expression of numerous ROS-homeostasis genes during organogenesis and stress-adaptive growth (Kumar et al., 2024; Gao, 2024).

3.7 Cytokinins — tuning ROS in immunity, photosynthesis, and priming
Cytokinins contribute to ROS-mediated stomatal immunity and influence photosynthetic resilience under stress. Mechanistically, cytokinin signaling (e.g., via AHK receptors and ARR transcription factors) can induce apoplastic peroxidases and regulate RBOH activity in guard cells, linking cytokinin to ROS bursts that close stomata in immune contexts; cytokinin treatments also prime antioxidant capacity during abiotic stress to protect photosynthetic machinery (Arnaud et al., 2017; Hudeček et al., 2023). Taken together, cytokinins help calibrate ROS for defensive signaling without compromising carbon assimilation.

3.8 Brassinosteroids (BRs) and gibberellins (GAs) — growth hormones that buffer redox stress
Brassinosteroids generally enhance antioxidant enzyme activities and improve cellular redox buffering, contributing to improved tolerance to drought, heat, and heavy metals; EBR (24-epibrassinolide) treatments consistently lower H₂O₂ and lipid peroxidation while elevating SOD, CAT, and APX activities in numerous crop systems (Avalbaev et al., 2024; Zhou et al., 2024). Gibberellins modulate ROS indirectly via growth-related metabolic fluxes and antioxidant gene expression in ways that influence seed germination and stress recovery; GA–ROS cross-talk is context dependent and interacts with ABA and other hormones in balancing growth vs stress protection (Liu et al., 2024).

3.9 Integration and cross-talk: a systems view
Across hormone classes, a common pattern emerges: (i) hormones induce spatiotemporally restricted ROS production by RBOHs and organelles as signaling events; (ii) they simultaneously engage antioxidant networks to prevent oxidative damage; and (iii) they shape transcriptional, post-translational and metabolic states that set thresholds for ROS signaling vs toxicity. These interactions are mediated by kinase cascades, transcription factors, small peptides, and metabolic regulators, producing hormone-specific “ROS signatures” that encode different physiological outcomes (Postiglione & Muday, 2020; Li et al., 2022; Rehman et al., 2023).

## 4. Hormonal crosstalk and ROS: integrated networks

### 4.1. Overview: ROS as integrative signals of hormonal networks

Reactive oxygen species (ROS) function as ubiquitous and flexible second messengers that translate environmental and developmental cues into cellular responses. Rather than acting in isolation, ROS operate at the intersection of hormone pathways: they are produced downstream of hormone perception, feed back on hormone biosynthesis and signaling, and mediate hormone–hormone interactions that tune growth, defense and acclimation responses (Mittler, Zandalinas, Fichman, & Van Breusegem, 2022). The ROS “signal” is therefore best viewed as embedded within a multi-layered signaling network in which hormones such as salicylic acid (SA), jasmonic acid (JA), abscisic acid (ABA), ethylene (ET), gibberellins (GA), brassinosteroids (BR) and auxin can act as both upstream regulators and downstream effectors of ROS dynamics (Mittler et al., 2022; Dvořák et al., 2021).

### 4.2. Network hubs and molecular nodes that couple hormones to ROS

A small number of molecular hubs integrates hormonal inputs with ROS production and sensing. Respiratory burst oxidase homologs (RBOHs) catalyze apoplastic ROS production and are direct regulatory targets of multiple hormone-activated kinases, calcium sensors, and small-molecule modifiers; their activity is therefore a principal node where hormone signals converge to generate ROS (Lee et al., 2020). Conversely, redox-sensitive transcriptional regulators such as NPR1 (NONEXPRESSOR OF PATHOGENESIS RELATED GENES 1) serve as nodes that translate redox changes into hormone-dependent transcriptional reprogramming, with major consequences for SA-mediated responses and crosstalk with growth hormones (Zavaliev & Dong, 2024). Receptor-like proteins that sense extracellular H₂O₂ (for example HPCA1) and downstream MAPKs and Ca²⁺ channels provide further mechanistic links between hormone perception and ROS signaling, enabling rapid amplification and cell-to-cell propagation of ROS-dependent signals (Wu et al., 2020; Lee et al., 2020).

### 4.3. Examples of hormone–ROS interactions and emergent behaviors

#### 4.3.1. SA–JA antagonism and ROS wave modulation

SA and JA exemplify hormone pairs whose antagonistic interplay is mediated — at least in part — by ROS dynamics. Recent experimental evidence shows that SA tends to amplify systemic ROS signaling (the “ROS wave”) whereas JA can suppress it; ethylene and ABA also influence ROS-wave propagation in stress contexts (Myers, Fichman, Zandalinas, & Mittler, 2023). Mechanistically, SA promotes redox changes that activate redox-sensitive regulators (e.g., NPR1), favoring defense transcriptional programs, while JA signaling via JAZ repressors and downstream effectors interferes with ROS propagation and some SA outputs, thereby biasing the network toward wound/ herbivore responses. These interactions illustrate how hormones shape both the amplitude and spatial reach of ROS signals, producing emergent systemic outcomes not predictable from single-pathway activity alone (Caarls, Pieterse, & Van Wees, 2015; Myers et al., 2023).

#### 4.3.2. ABA, stomata and systemic redox integration

ABA is a canonical regulator of ROS in guard cells: ABA receptor activation leads to OST1/SnRK2 kinase activity, RBOH phosphorylation and local H₂O₂ generation, which then gates Ca²⁺ influx and stomatal closure (Postiglione & Muday, 2020). Importantly, ABA-driven ROS are not restricted to the apoplast: chloroplast and peroxisomal ROS pools, as well as redox metabolites, modulate ABA signalling and vice versa, producing compartmentalized redox signatures that underpin stomatal, developmental and systemic responses to drought and high light (Postiglione & Muday, 2020; Waszczak et al., 2018). The recent discovery of HPCA1 as an extracellular H₂O₂ receptor provides a molecular explanation for how apoplastic ROS can be perceived and translated into Ca²⁺ and kinase responses that interact with hormonal circuits. These findings place ROS squarely as both mediator and modulator of ABA action across scales.

#### 4.3.3. Growth–defense trade-offs: DELLA, JAZ and NPR1 nodes

Cross-regulation between growth and defense hormones illustrates network logic in which ROS refine decision-making. DELLA proteins link GA to JA signaling by binding JAZ repressors and modulating MYC transcription factors, thereby altering JA responses and downstream ROS-related defenses; GA-induced DELLA degradation therefore shifts the balance toward growth at the expense of certain ROS-associated defense outputs (Hou et al., 2010). NPR1, besides being redox-regulated by SA, also influences interactions between SA and growth signals, acting as a nexus where redox state, immunity and hormone crosstalk coalesce (Zavaliev & Dong, 2024). Thus, hormonal modulation of ROS is a core mechanism by which plants prioritize resource allocation under competing demands.

#### 4.4. Post-translational regulation, spatiotemporal specificity and feedback

A recurring theme is that hormones control ROS not only by changing expression of ROS-related genes but overwhelmingly through post-translational modifications (PTMs) that set the timing and magnitude of ROS production and turnover. RBOHD is regulated by activating and inhibitory phosphorylation, ubiquitination, S-nitrosylation and Ca²⁺-dependent interactions; these PTMs are delivered by hormone-regulated kinases, E3 ligases and redox enzymes, enabling rapid, reversible tuning of ROS outputs (Lee et al., 2020; Mase, 2021). Likewise, redox-dependent modifications of transcription factors (thiol oxidation, S-nitrosylation, glutathionylation) alter their DNA-binding or interaction profiles and therefore rewire hormone-driven transcriptional programs (Mase, 2021; Dvořák et al., 2021). The combinatorial control afforded by PTMs and compartmentalization explains how similar hormone exposures can yield distinct ROS signatures and biological outcomes depending on tissue, developmental stage and prior stress history.

#### 4.5. Systems perspective: robustness, plasticity and future challenges

Viewed as a network, hormone–ROS crosstalk provides robustness through redundancy (multiple ROS sources and sensors) and plasticity through context-dependent rewiring (hormone status, PTMs, redox state). Yet this complexity creates challenges for mechanistic dissection: spatially resolved redox reporters, quantitative mapping of PTMs, and integrative modelling are all needed to predict network behavior under combined stresses. Recent reviews emphasize the value of multi-omics and live-cell imaging to capture the spatiotemporal layers of hormone–ROS interactions and to identify nodal points for crop improvement without compromising fitness (Mittler et al., 2022; Zavaliev & Dong, 2024). In short, hormonal crosstalk and ROS form a dynamic, multi-scale information network that plants exploit to make context-appropriate growth/defense decisions; dissecting this network remains a frontier with immediate translational potential.

## 5. Molecular mechanisms: receptors, kinases, TFs and redox sensors

5.1. Overview — molecular tiers that connect hormones to ROS
Plant hormonal regulation of oxidative status operates through a multilayered molecular architecture that begins with perception at receptors (hormone receptors and ROS sensors), continues through kinase/phosphatase circuits that rapidly sculpt ROS-producing and -scavenging enzymes, and culminates in transcriptional reprogramming mediated by redox-sensitive transcription factors (TFs) and thiol-based redox relays. This hierarchical arrangement enables both fast post-translational tuning of ROS output (seconds–minutes) and longer-term reprogramming of antioxidant capacity and metabolism (hours–days) (Park et al., 2009; Ma et al., 2009; Liebthal et al., 2018).

5.2. Receptors and early sensing: hormone receptors and ROS perception
Hormone receptors (example: PYR/PYL/RCAR ABA receptors) occupy the apex of ABA signaling and control downstream phosphatase/kinase activity that indirectly shapes ROS production through effectors such as NADPH oxidases (Park et al., 2009; Ma et al., 2009). Equally important for ROS-hormone cross-talk are bona fide ROS sensors: the discovery of HPCA1 — an LRR receptor kinase that perceives extracellular H₂O₂ and triggers Ca²⁺ influx and stomatal responses — demonstrates that plants possess cell-surface perception mechanisms for oxidants as well as for classic hormones (Wu et al., 2020). Together, hormone receptors and ROS sensors provide two entry points by which hormonal cues and oxidative signals can rapidly intersect at the plasma membrane and apoplast (Wu et al., 2020; Park et al., 2009).

5.3. Kinases, phosphatases and direct control of ROS sources
The activity of plasma-membrane NADPH oxidases (RBOHs), the major regulated source of apoplastic ROS, is controlled by several kinase families and counterbalanced by phosphatases. Calcium-dependent protein kinases (CPKs/CPKs) phosphorylate specific N-terminal serine residues on RBOHD and thereby potentiate ROS output in immune and stress responses (Dubiella et al., 2013). Parallel, Ca²⁺-independent phosphorylation of RBOHD by receptor-like cytoplasmic kinases such as BIK1 (associated with pattern recognition receptors) is essential for the PAMP-triggered ROS burst and stomatal immunity (Li et al., 2014). Beyond N-terminal control, recent studies show additional regulatory layers — phosphorylation, ubiquitination and C-terminal modification — that fine-tune RBOHD abundance and activity during immunity (Lee et al., 2020). These kinase modules are themselves wired into hormone signaling: ABA-activated SnRK2s/OST1 and PP2C phosphatases form the core ABA pathway that modulates ion channels, stomatal aperture and ROS dynamics (Park et al., 2009; Ma et al., 2009; Sirichandra et al., 2009). In short, kinases and phosphatases act as fast switches that link hormone perception to the enzymatic machines that generate ROS (Dubiella et al., 2013; Li et al., 2014; Lee et al., 2020).

5.4. Transcription factors — redox sensitivity and hormonal integration
Transcriptional regulation provides the medium- to long-term adaptation of redox metabolism to hormonal context. Several TF families that mediate hormone responses are directly modulated by cellular redox state. A paradigmatic example is NPR1, a master regulator of salicylic acid (SA) signaling whose oligomer/monomer status and nuclear localization are controlled by redox modifications (S-nitrosylation and thiol–disulfide exchange) and by thioredoxins; this redox control directly gates SA-dependent transcriptional reprogramming (Mou et al., 2003; Tada et al., 2008). Similarly, membrane-tethered NAC TFs (e.g., ANAC017) transduce mitochondrial/chloroplast retrograde redox signals into nuclear gene expression changes that intersect with hormone pathways (Ng et al., 2013; Van Aken et al., 2016). These TF nodes operate as integrators: their redox state, hormone-induced phosphorylation, and partner availability determine whether downstream antioxidant genes, alternative oxidases, or hormone-responsive effectors are expressed (Ng et al., 2013; Tada et al., 2008).

### 5.5. Redox sensors and redox relays: thiol peroxidases, thioredoxins and electronic conduits

Beyond receptor-level sensors, thiol-based proteins (peroxiredoxins, thioredoxins, glutaredoxins) form redox relays that both detoxify H₂O₂ and transduce its presence into regulatory modifications on client proteins. Peroxiredoxins (Prxs) are highly abundant thiol peroxidases that act as both scavengers and redox relays, undergoing reversible oxidation/hyperoxidation and oligomerization that can influence signaling complexes and transcriptional outputs (Liebthal et al., 2018; Dietz, 2011). Thioredoxin and glutaredoxin systems reduce oxidized sensor proteins and can catalyze denitrosylation/denitrosation events, thereby recovering TFs and enzymes for reactivation (Meyer et al., 2012). The conceptual model that emerges is one of “redox circuitry” — sensors (Prxs, TRXs) detect peroxide flux, relay oxidation to regulatory cysteines on TFs and signaling proteins, and are themselves reset by reductive systems linked to metabolism (Liebthal et al., 2018; Meyer et al., 2012).

5.6. Tools and experimental demonstrations of mechanism
Genetically encoded redox biosensors (e.g., roGFP2-Orp1) and modern calcium/ROS imaging have been instrumental in dissecting spatio-temporal patterns of hormone-driven ROS (Nietzel et al., 2019). These tools confirm that hormone application or receptor activation can produce localized ROS microdomains and redox changes that precede transcriptional responses, emphasizing that spatial control (apoplast vs. organelle vs. nucleus) is as important as molecular identity (sensor, kinase, TF) for signaling outcomes (Nietzel et al., 2019; Wu et al., 2020).

5.7. Concluding synthesis: modularity and robustness
Collectively, plant hormone–ROS cross-talk is implemented through modular, partially redundant molecular mechanisms: (i) membrane perception (hormone receptors, ROS sensors) provides immediate context; (ii) kinase/phosphatase circuits convert perception into rapid modulation of ROS flux; (iii) thiol-based redox relays and TF oxidation states encode redox history and effect lasting gene expression changes. This architecture explains why altering a single kinase or redox relay can dramatically change stress tolerance, yet the system retains robustness through alternative kinases, multiple RBOH isoforms, and overlapping redox relays (Park et al., 2009; Dubiella et al., 2013; Liebthal et al., 2018).

## 6. Organelle-specific ROS and hormone action

### 6.1 Overview

Organelle compartments (chloroplasts, mitochondria and peroxisomes) are major and distinct sources of reactive oxygen species (ROS) in plant cells, and each organelle both generates ROS and hosts dedicated redox buffering and signalling machinery. Hormones modulate organelle ROS production, antioxidant capacity and retrograde signalling in highly organelle-specific ways, and conversely organelle ROS influence hormone biosynthesis and downstream hormone signalling cascades. Below I summarize current evidence for organelle-specific intersections between ROS and hormones, with examples showing (i) how hormones tune organellar ROS production/scavenging, (ii) how organellar ROS act as localized signals that feed into hormone networks, and (iii) how inter-organelle coordination (contact sites, metabolic shuttles and retrograde signalling) creates emergent hormone-ROS behaviour. (For primary evidence and reviews cited below, see the APA reference list for this section.)

### 6.2 Chloroplast-localized ROS and hormonal modulation

6.2.1 Sources and signalling context
Chloroplasts produce multiple ROS species (singlet oxygen, superoxide, hydrogen peroxide) as inevitable by-products of photosynthetic electron transport, especially under excess light and when Calvin–Benson cycle capacity is limiting. Because chloroplast ROS can damage photosystems but also act in signalling, plants possess thiol-based redox relays, peroxiredoxins and ascorbate–glutathione systems to control chloroplast ROS dynamics and their export to the cytosol/nucleus (Foyer & Hanke, 2022).

6.2.2 SAL1–PAP retrograde hub and hormone links
The SAL1 (adenosine bisphosphate phosphatase)–PAP pathway exemplifies how chloroplast redox state (via thiol oxidation and ROS) is converted into an informational metabolite (3′-phosphoadenosine 5′-phosphate, PAP) that moves or signals to the nucleus and affects hormone-responsive gene networks (Chan et al., 2016; Phua et al., 2018). Work on SAL1/PAP shows that redox-dependent inactivation of SAL1 leads to PAP accumulation and downstream transcriptional programmes that overlap with hormonal responses, supporting a direct route by which chloroplast ROS influence hormonal signalling and development.

6.2.3 Hormones acting on chloroplast ROS homeostasis
Cytokinins, brassinosteroids and abscisic acid (ABA) have been shown to alter chloroplast ROS and antioxidant systems in stress and developmental contexts. Cytokinins can stimulate antioxidant capacity and protect photosynthetic apparatus under stress (reviewed in Foyer & Hanke, 2022; see also work compiling cytokinin effects on chloroplast redox), while brassinosteroids modulate photosynthetic electron flow and inter-organelle redox balance in ways that change chloroplast ROS emission (e.g., BL-induced coordination of mitochondrial electron transport and chloroplast function). These hormone-mediated adjustments often act through transcriptional reprogramming of chloroplastic antioxidant enzymes and via metabolic shuttles (malate/OAA valve) that drain excess reducing power. Together these studies place chloroplasts as hormone-sensitive hubs whose ROS output is actively tuned by hormonal state.

6.2.4 Measurement approaches that resolved hormone–chloroplast ROS links
Genetically encoded H₂O₂ sensors targeted to chloroplasts (and other compartments) have clarified spatio-temporal aspects of ROS dynamics following hormone treatments or environmental cues; this technical progress underpins many of the mechanistic conclusions above (see Nietzel et al., 2019).

### 6.3 Mitochondrial ROS and hormone action

6.3.1 Mitochondria as ROS signalers and retrograde triggers
Mitochondrial electron transport (complexes I and III in particular) is a major source of ROS that can act locally and trigger nuclear transcriptional responses — the mitochondrial dysfunction stimulon (MDS) — via membrane-tethered NAC transcription factors (e.g., ANAC017/ANAC013). These retrograde circuits are shaped by the balance between electron transport and alternative oxidase (AOX) activity, antioxidant capacity, and organellar proteolytic events that release membrane-tethered TFs to the nucleus (Ng et al., 2013; Shapiguzov et al., 2019).

6.3.2 Hormonal recruitment of mitochondrial signalling pathways
Recent work shows that mitochondrial retrograde signalling is not isolated from hormone pathways: ANAC017 directly engages ethylene and auxin signalling (He et al., 2022), creating feed-forward and feedback loops that balance stress responses with growth. Functionally, ethylene signalling often precedes auxin activation during mitochondrial stress responses, and both hormones modulate components of mitochondrial stress outputs (transcription factors, AOX expression), demonstrating that hormones reshape mitochondrial ROS signalling outcomes at the transcriptional level.

6.3.3 Mitochondrial ROS in stomatal ABA responses — a recent case study
A striking, mechanistically clear example of hormone–organelle ROS interplay comes from guard cell studies showing that ABA increases hydrogen peroxide within mitochondria and that mitochondrial H₂O₂ promotes stomatal closure. This result reveals that hormone-triggered ROS production can be organelle-confined and that such compartmentalized ROS are functionally important for a canonical hormone response (Postiglione & Muday, 2023).

6.3.4 Coordination with chloroplasts and nucleus via proteins like RCD1
Cross-talk between chloroplast and mitochondrial ROS signalling is mediated by nuclear factors such as RCD1 that interact with ANAC TFs and modulate their activity; thus organellar ROS signals are integrated and gated before they drive hormone-related transcriptional programmes (Shapiguzov et al., 2019). This integration is a key mechanism whereby hormone signalling is contextualized according to the combined redox status of multiple organelles.

### 6.4 Peroxisomal ROS, hormone metabolism and signalling

6.4.1 Peroxisomes: ROS factories that intersect hormone biosynthesis
Peroxisomes produce H₂O₂ as a by-product of β-oxidation and other metabolic reactions and thus serve both metabolic and signalling roles. Critically, peroxisomes are integral to the biosynthesis of several hormones (notably jasmonic acid via β-oxidation steps and contributions to salicylic acid pathways), so ROS production and hormone metabolism are physically and functionally coupled in this compartment (Corpas et al., 2020; Kotera et al., 2024).

6.4.2 Hormone regulation of peroxisomal redox and immunity links
Peroxisomal redox status influences immune hormone pools (e.g., SA and JA) and peroxisome-localized enzymes that participate in hormone biosynthetic steps are regulated by stress and hormonal signals. Recent molecular characterization of peroxisomal enzymes required for SA biosynthesis reinforces the concept that peroxisomes act as hormone production platforms whose redox output can feed back on signalling (Kotera et al., 2024). Complementary reviews synthesize how peroxisome ROS shape signalling networks and stress responses more broadly (Huang et al., 2022; Sandalio et al., 2021).

### 6.5 Organellar contact sites, metabolic shuttles and integrated hormone–ROS networks

6.5.1 Inter-organelle exchanges that distribute redox equivalents
Metabolic shuttles (malate/OAA valve, triose phosphate shuttles) and physical contacts between chloroplasts, mitochondria and peroxisomes permit controlled transfer of reducing equivalents and metabolites that modulate ROS production. Hormones (e.g., brassinosteroids) can enhance chloroplast–mitochondrion coordination — affecting AOX/COX balance and altering whole-cell ROS homeostasis — thereby tuning photosynthetic performance and hormone-dependent growth responses (Mahati & Padmasree, 2023). Thus hormone action often manifests not as a single-organelle effect but as reprogramming of networked redox fluxes.

6.5.2 Retrograde signals converge on hormone outputs
Multiple retrograde pathways (PAP from chloroplasts, mitochondrial ANAC-mediated MDS) show overlapping transcriptional footprints and are able to recruit or be attenuated by hormone signalling modules (e.g., ABA, ethylene, auxin), so hormone responses are an output of integrated organelle-to-nucleus communication rather than of single organelle cues (Phua et al., 2018; He et al., 2022). This convergence helps explain why hormone mutants often show organelle-related phenotypes and why organelle mutants display altered hormone sensitivity.

### 6.6 Emerging tools, knowledge gaps and future directions

6.6.1 Tools and approaches
Organelle-targeted genetically encoded ROS sensors (e.g., roGFP2-Orp1), compartment-specific proteomics, organelle-targeted transcriptomics and inducible organelle perturbation (chemical or genetic) are revealing hormone-dependent, compartment-specific ROS dynamics at unprecedented resolution (Nietzel et al., 2019). Combining these with high-temporal transcriptomics has already begun to reveal hormone recruitment sequences (e.g., ethylene preceding auxin in mitochondrial stress).

6.6.2 Major open questions
Important unresolved issues include: (i) precise chemical identity and mobility of organellar ROS signals that affect specific hormone pathways; (ii) mechanisms that control directionality (which organelle’s retrograde signal dominates under mixed stresses); (iii) molecular bases for organelle-to-organelle routing of redox equivalents under hormone action; and (iv) how cell-type-specific organelle networks generate tissue-level hormone outputs. Systematic use of organelle-targeted reporters together with genetic perturbations of hormone biosynthetic and signalling nodes will be central to making progress.

## 7. Hormonal priming and cross-protection

Hormonal priming refers to a transient, often low-dose pre-exposure of seeds or plants to phytohormones (or their precursors/volatiles) that does not cause major phenotypic change at the time of application but leaves a lasting “sensitized” state. Upon a later stress, primed plants respond faster or stronger, typically through redox and transcriptional circuits, resulting in enhanced tolerance and/or faster recovery. Importantly, priming can produce **cross-protection** (also called cross-tolerance), where priming by one stimulus (or hormone) enhances resistance to different, even unrelated, stresses (e.g., drought → heat; hormonal pretreatment → salinity and pathogen challenge). Mechanistically, the primed state combines redox poise adjustments, hormone–ROS feedbacks, and memory marks in chromatin and metabolism. (Liu et al., 2022; Harris et al., 2023; Kambona et al., 2023).

### 7.1 What makes a plant “primed”?

Priming establishes a heightened alert status with minimal fitness cost under non-stress conditions. Hallmarks include (i) faster induction of antioxidant and detoxification systems (e.g., APX, CAT, SOD; AsA–GSH cycle), (ii) quicker reconfiguration of stomatal and hydraulic responses, and (iii) “transcriptional memory” (short- and mid-term) and, in some cases, **epigenetic memory** (longer term) that lowers re-induction thresholds for key defense and acclimation genes. These layers are organized around redox-sensitive nodes and hormone receptors/kinases (ABA/SA/JA/CK/BR) that interface with ROS waves and Ca²⁺/electrical systemic signals. (Harris et al., 2023; Myers et al., 2024; Liu, Liu, & Mou, 2024).

### 7.2 Salicylic acid (SA) priming

SA has a dual identity in biotic and abiotic contexts. As a **defense priming** agent, SA (or SA-rich extracts/seeds) imprints redox and transcriptional memory that accelerates downstream defenses (e.g., NPR1-dependent transcription, antioxidant ramp-up) and contributes to **systemic acquired resistance (SAR)**—a canonical primed state with a strong ROS component. In crops, seed or foliar SA priming often improves drought and salinity performance by augmenting antioxidant capacity and osmolyte accumulation, with practical dose windows typically ~0.1–0.5 mM for seeds (species-dependent). Field and greenhouse studies in cucurbits and solanaceae have shown yield and quality gains under water deficit after SA priming. (Liu, Liu, & Mou, 2024; Alam et al., 2022).

### 7.3 Jasmonates (JA/MeJA): rapid defense and broad cross-protection

Jasmonates are potent priming molecules across stresses. Seed or seedling priming with MeJA/JA can pre-activate ROS-linked signaling and glutathione metabolism, leading to faster stomatal control, membrane stabilization, and ion homeostasis under salinity/drought; JA priming can also enhance pathogen and herbivore defenses, exemplifying **cross-protection** across abiotic/biotic arenas. Controlled studies in wheat under salinity show that JA priming coordinates multiple defense layers (antioxidants, compatible solutes, ion transporters) for sustained tolerance. Mechanistically, JA crosstalks with gasotransmitters (NO/H₂S), Ca²⁺, and ABA nodes to amplify redox-adjusted responses. (Sheteiwy et al., 2022; Rehman et al., 2023; Kolupaev & Veklich, 2023).

### 7.4 Abscisic acid (ABA): drought “readiness” and stress memory

ABA is central to dehydration signaling; low-dose **ABA seed or root priming** sets stomata and downstream transcriptional programs into a ready-to-respond configuration. Recent crop studies (wheat, sorghum) show ABA priming improves water status, boosts ROS-scavenging, and preserves photosynthesis under deficit irrigation, with **transcriptomic memory** evident in stress-reinduced seedlings. In wheat, ABA-mediated seed/root priming strengthened antioxidative defense and growth under PEG-simulated drought; in sweet sorghum, ABA seed priming enhanced drought acclimation with clear molecular signatures. (Baroi et al., 2024; Luhua et al., 2025).

### 7.5 Cytokinins (CKs): priming at the photosynthesis–immunity interface

Although historically viewed as growth hormones, CKs modulate stress priming by stabilizing photosynthetic machinery and influencing stomatal and immune circuits. CK-based priming can preserve chlorophyll, maintain PSII efficiency, and adjust ROS/antioxidant balance under drought or heat, thereby limiting oxidative injury while enabling swift defense deployment—particularly in “stomatal immunity” contexts where ROS, SA, and CK intersect. (Hudeček et al., 2023).

### 7.6 Brassinosteroids (BRs): redox-centric priming for multiple stresses

Exogenous BRs (e.g., epi-brassinolide) frequently act as priming cues that reduce ROS overaccumulation during later drought/heat/salinity episodes and strengthen antioxidant systems and osmotic adjustment. In horticultural species, BR pre-treatments improved photosynthesis, hydration, and membrane integrity under drought, with evidence for BR–ABA crosstalk that fine-tunes stomatal and redox control. BR priming effects span salinity, temperature extremes, metal toxicity, and combined stresses—an operationally valuable form of **cross-protection**. (Zhang et al., 2023).

### 7.7 Auxins and GA in hormopriming of seeds

Seed hormopriming with IAA or GA₃ can accelerate germination, root system establishment, and early antioxidant preparedness, improving downstream stress tolerance. In maize, IAA seed priming improved ROS detoxification and ion balance under salinity; GA priming enhances emergence under low water potential and stabilizes photosynthetic pigments/ionic homeostasis under salt. These effects often interact with ABA/JA states, pointing to an integrated hormone–redox memory circuit set during imbibition. (Ellouzi et al., 2024; Rhaman et al., 2021).

### 7.8 From local to systemic: ROS-mediated signaling in primed plants

Priming reshapes systemic communication. Long-distance ROS waves integrate with Ca²⁺/electrical/glutamate signals to spread **systemic acquired acclimation (SAA)**, coordinating distal tissue responses during combined or sequential stresses. Hormones (SA, JA, ABA, BRs) interface with these waves to imprint faster, stronger secondary responses; JA/SA can tune ROS-wave amplitude, and ABA influences wave-linked stomatal dynamics. These systemic layers underpin cross-protection after a local priming event. (Myers et al., 2024; Liu, Liu, & Mou, 2024).

### 7.9 Practical considerations: dose, timing, delivery, and costs

**Dose/timing:** Priming is typically effective at low doses and during receptive windows (seed imbibition, early seedling). Over-application can cause growth penalties. **Delivery:** Seed soaking/drying (“on-seed memory”), root dip, and brief foliar sprays are common. **Cultivar/context specificity:** Benefits vary with genotype, seed vigor, and stress regime; field validation is essential. **Trade-offs:** Most studies report small or negligible costs under benign conditions, but **fitness costs** can emerge if priming is excessive or mismatched to the stress pattern. (Rhaman et al., 2021; Liu et al., 2022; Matkowski & Daszkowska-Golec, 2025).

### 7.10 Case snapshots of cross-protection

* **JA priming → salinity & pathogen:** JA/MeJA priming in cereals improves ionic homeostasis and oxidative defense under salinity and also enhances biotic resistance—illustrating abiotic↔biotic cross-protection. (Sheteiwy et al., 2022; Rehman et al., 2023).
* **SA seed priming → drought yield:** Low-millimolar SA seed priming improved melon yield and fruit quality under water deficit via boosted antioxidant capacity and osmolytes. (Alam et al., 2022).
* **ABA seed/root priming → drought acclimation:** ABA priming in wheat and sorghum enhanced antioxidant defenses, water status, and transcriptional re-induction at stress—clear memory traits with cross-scale benefits. (Baroi et al., 2024; Luhua et al., 2025).
* **BR pre-treatments → multi-stress resilience:** BRs prime ROS/antioxidant and osmotic systems, mitigating drought, salinity, and temperature stresses in multiple horticultural species. (Zhang et al., 2023).

## 8. Translational implications for crop improvement

Plant hormones interface tightly with cellular redox metabolism; that coupling can be exploited to build crops that maintain photosynthetic performance and yield under stress without incurring large growth penalties. Here we outline practical levers—from seed and foliar treatments to receptor engineering, BR-pathway tuning, and microbiome-assisted strategies—together with design principles (dose, timing, tissue targeting) that determine whether hormonal manipulation improves or undermines performance in the field.

### 8.1 Why hormonal–redox engineering is translationally attractive

Hormonal signaling sits upstream of key oxidative-stress checkpoints (stomatal regulation, antioxidant capacity, damage repair). Small, well-timed shifts in abscisic acid (ABA), jasmonates, salicylic acid (SA), brassinosteroids (BRs), or ethylene signaling can pre-empt runaway ROS, sustain membrane integrity, and preserve reproductive success under drought, salinity, and heat. Notably, ABA receptor manipulation and BR pathway tuning already show yield-relevant phenotypes in cereals and rice, offering clear breeding and chemistry entry points (Kim et al., 2014; Mao et al., 2022; Zolkiewicz & Gruszka, 2025).

### 8.2 Field-facing levers I: exogenous treatments and seed priming

**Seed priming.** Pre-sowing priming with hormonal or redox-active cues strengthens antioxidant networks, accelerates emergence, and often increases yield stability across horticultural and arable species (Zulfiqar, 2021). Meta-syntheses emphasize reduced oxidative injury and better stand establishment as the proximate drivers (Zulfiqar, 2021). Jasmonic acid (JA) priming in wheat, for instance, improved growth and yield under saline irrigation across two seasons while lowering H₂O₂ and lipid peroxidation, directly linking a hormonal cue to controlled ROS status (Sheteiwy et al., 2022).

**Foliar or soil-applied regulators.** High-affinity ABA agonists can transiently enhance antitranspirant responses, providing “on-demand” water saving while keeping photosynthesis largely intact when properly dosed. Opabactin (OP), a structure-guided ABA mimic, increased receptor affinity and in vivo activity and proved effective in Arabidopsis, wheat, and tomato—demonstrating a practical chemical gateway to redox-aware water-use control (Vaidya et al., 2019). BR sprays, when used within hormetic windows, can promote antioxidant capacity and mitigate heat/salt damage; because BR influences growth and stress pathways broadly, modern recommendations favor cultivar- and stage-specific dosing and, where possible, targeting BR components that uncouple yield penalties (Zolkiewicz & Gruszka, 2025).

**Design principles.** Successful field deployment hinges on: (i) **dose** (avoid supra-optimal levels that induce ROS and growth penalties), (ii) **timing** (e.g., pre-flowering drought windows), (iii) **tissue targeting** (guard cells vs. whole plant), and (iv) **stacking** with agronomy (irrigation scheduling). Reviews consistently caution that the same regulator can be protective or deleterious depending on these axes (Zulfiqar, 2021; Zolkiewicz & Gruszka, 2025).

### 8.3 Field-facing levers II: editing, breeding, and receptor engineering

**Receptors as precise entry points.** In rice, constitutive overexpression of the ABA receptor OsPYL5 enhances drought tolerance but suppresses growth and yield—an object lesson in how stronger stress signaling can over-amplify ROS-dependent restraint of growth (Kim et al., 2014). By contrast, stress- or tissue-inducible manipulation of PYL receptors can preserve baseline growth while boosting drought/cold performance. Overexpressing OsPYL10 (under a stress-inducible promoter) increased grain yield under vegetative-stage drought and improved cold tolerance, while reducing H₂O₂ and malondialdehyde accumulation (Verma et al., 2019). In wheat, a TaPYL1-1B allele increased water-use efficiency and grain yield under drought, illustrating how endogenous receptor variation can be converted into selection markers or editing targets for large-acreage cereals (Mao et al., 2022).

**Chemistry × genetics.** Receptor engineering synergizes with chemistry: high-affinity ABA agonists such as opabactin enable *transient* stomatal control—mitigating oxidative load and water loss—without permanently shifting growth (Vaidya et al., 2019). Importantly, promoter choice and receptor subtype matter: PYL9 overexpression boosts drought resistance but also accelerates senescence via ABA core signaling, a trade-off that can be advantageous under extreme drought yet risky for yield if mis-timed (Zhao et al., 2016). Tissue-specific promoters (e.g., guard-cell biased) and inducible systems are therefore preferable for minimizing ROS-related growth costs.

**BR pathway tuning.** Because BR homeostasis is tightly linked to oxidative stress tolerance and architecture, editing “nodes” that bias stress protection without inducing lodging or excessive leaf angle is strategically valuable. In rice, overexpressing the BR co-receptor SERK2 *simultaneously* enhanced grain size and salt resistance with little architectural change—evidence that early BR signaling can be harnessed for stress resilience without yield penalties (Dong et al., 2020). A cereal-focused synthesis shows that adjusting BR biosynthesis/catabolism or key signaling transducers can improve tolerance to drought, salinity, and oxidative stress while sustaining agronomic traits (Zolkiewicz & Gruszka, 2025).

### 8.4 Microbiome-assisted hormone and redox tuning

Plant growth–promoting rhizobacteria (PGPR) confer salt and drought tolerance by modulating phytohormones (IAA, ABA, ethylene), strengthening antioxidant systems, and improving ion homeostasis—responses that collectively restrain ROS damage. Recent syntheses detail how PGPR-triggered hormonal crosstalk elevates antioxidant enzyme activities and maintains photosystems under salinity, a translationally attractive, low-input route for stress-prone soils (Giannelli et al., 2023; Kumawat et al., 2023). Selecting strains for consistent phytohormone output and compatibility with host genotypes can amplify benefits and reduce variability across seasons and fields.

### 8.5 Smarter delivery: from priming coatings to nanocarriers

Delivery innovations aim to narrow effective dose windows and place signals where they matter most. **Seed coatings** standardize priming responses and protect actives until imbibition (Zulfiqar, 2021). **Nanocarrier-assisted delivery**—including seed nanopriming—can enhance stability, uptake, and tissue targeting of hormone-like primers, offering stronger and more durable redox conditioning at lower application rates; a recent Trends in Plant Science review highlights nanocarriers that co-deliver hormonal and redox cues to optimize priming efficiency (Gohari et al., 2024). These platforms are particularly promising for smallholder contexts where precise field dosing is hard to maintain.

### 8.6 Risk management: dose, duration, and developmental stage

Because hormone–ROS modules sit at growth–defense trade-off junctions, **overactivation** can depress biomass via prolonged stomatal closure, enhanced protein turnover, or senescence (Kim et al., 2014; Zhao et al., 2016). Translational programs should therefore:

* favor **inducible or tissue-specific** transgenes (e.g., stress-inducible PYLs) to avoid chronic oxidative constraints (Verma et al., 2019);
* pilot **stage-specific** chemistries (e.g., ABA agonists during reproductive drought windows) (Vaidya et al., 2019); and
* prefer **BR nodes** (e.g., SERK2) that preserve favorable architecture while elevating antioxidant capacity and stress tolerance (Dong et al., 2020; Zolkiewicz & Gruszka, 2025).

### 8.7 A practical pipeline for deployment

1. **Screen**: combine seed-priming panels (hormonal and redox-active cues) with PGPR consortia across representative stress scenarios; track redox markers (MDA, H₂O₂), photosynthetic efficiency, and early stand metrics (Zulfiqar, 2021; Kumawat et al., 2023).
2. **Genetic targeting**: validate receptor or BR-node edits in elite backgrounds; prioritize alleles showing stress benefits without architecture/yield penalties (Mao et al., 2022; Dong et al., 2020).
3. **Chemistry alignment**: match on-demand agonists (e.g., OP) to irrigation and weather forecasts; integrate with deficit irrigation to minimize ROS surges during peak vapor pressure deficits (Vaidya et al., 2019).
4. **Delivery engineering**: deploy seed coatings or nano-enabled carriers to reduce application frequency and improve targeting (Gohari et al., 2024).
5. **G×E validation**: multi-location trials to confirm stability of ROS moderation and yield impacts across climates and soil salinity gradients (Zolkiewicz & Gruszka, 2025).

### 8.8 Outlook

The most credible near-term wins combine **seed priming** (lower entry cost), **PGPR consortia** (sustained hormonal/redox tuning), and **trait-led editing** of ABA receptors or BR components that have already shown grain-yield or water-use dividends. As climate volatility grows, such integrated hormone–redox strategies—timed, targeted, and stackable—offer a pragmatic path to sustaining yield and quality at scale.

## 9. Knowledge gaps and future directions

Despite rapid progress, our understanding of how plant hormones shape oxidative stress—spatiotemporally, quantitatively, and across scales from molecules to fields—still has important blind spots. Below, we outline key gaps and propose concrete directions to accelerate discovery and translation.

### 9.1. Measuring the right ROS, in the right place, at the right time

A persistent obstacle is analytical: many commonly used fluorescent probes lack specificity and can report mixed oxidative chemistries, confounding mechanistic interpretation and cross-study comparisons. Community guidance now exists for best practices (e.g., calibration, probe controls, artifact detection), yet these standards are not uniformly adopted in plant work (Murphy et al., 2022). Next-generation genetically encoded biosensors (e.g., HyPer, roGFP2-Orp1) and modular reporters for glutathione and NADPH pools can quantify dynamics with organelle and cell-type resolution, but require careful targeting, in vivo calibration, and standardized reporting to be broadly comparable (Müller-Schüssele et al., 2021; Rowe et al., 2025). Explicit adoption of reporting checklists (probe identity, dynamic range, pH sensitivity, phototoxicity budgets, and spectral bleed-through) should be a publication prerequisite in plant ROS–hormone studies. (Murphy et al., 2022; Müller-Schüssele et al., 2021; Rowe et al., 2025).

### 9.2. From bulk tissue to single-cell and spatial maps

Hormone–ROS interactions are deeply cell-contextual. Bulk ‘omics often averages away decisive subpopulations (e.g., specific cells at the vasculature, boundary layers, or meristems) that coordinate long-distance responses. Single-cell and spatial transcriptomics in plants are now mature enough to chart hormone biosynthesis/perception modules alongside redox enzymes and transporters, but have rarely been paired with live redox imaging or protein redox-proteomics in the *same* tissue context (Chen et al., 2023; Yin et al., 2023). Future work should integrate single-cell atlases with redox biosensors and targeted chemoproteomics to produce causal maps of hormone–ROS circuits at cellular resolution. (Chen et al., 2023; Yin et al., 2023).

### 9.3. Decoding ROS perception and receptor diversity

The discovery of the extracellular H₂O₂ receptor kinase HPCA1 established direct ROS perception at the plant plasma membrane and linked ROS pulses to Ca²⁺ influx and systemic signaling (Wu et al., 2020; Fichman et al., 2022). However, HPCA1 does not explain all ROS-triggered responses, implying additional receptors or co-receptors, potentially tissue- or stimulus-specific. Defining the repertoire of apoplastic ROS sensors, their ligand specificities (e.g., H₂O₂ vs. other peroxides), and their coupling to hormone pathways (ABA, SA, auxin) remains a central gap. Ligand-binding biophysics, receptor structural studies, and forward genetic screens in diverse species will be essential. (Wu et al., 2020; Fichman et al., 2022).

### 9.4. Post-translational logic: multi-site control of ROS production and signaling

Hormone–ROS crosstalk often converges on NADPH oxidases (RBOHs) and antioxidant systems via multi-site phosphorylation, ubiquitination, and thiol redox switches. We still lack proteome-wide, time-resolved maps of these modifications under defined hormonal cues. For example, RBOHD is controlled by phosphorylation and ubiquitination during immunity (Lee et al., 2020), yet the combinatorial “PTM code” that integrates hormonal inputs (ABA, SA, BRs) with ROS amplitudes is incompletely understood. Redox chemoproteomics that capture cysteine oxidation together with phospho/ubiquitin states, ideally paired with dynamic modeling, should be prioritized. (Lee et al., 2020; Huang et al., 2019; Rowe et al., 2025).

### 9.5. Systemic signaling: how ROS waves encode information

ROS waves orchestrate long-distance acclimation and interface with electrical and Ca²⁺ signals, but their “syntax” (amplitude, frequency, duration, and spatial routes) and decoding remain unclear (Myers et al., 2024; Peláez-Vico et al., 2022). Vascular bundles are key conduits for systemic ROS/calcium signaling, yet how hormonal states precondition tissues to transmit/interpret waves is unknown (Zandalinas et al., 2020). Interventions that selectively modulate wave parameters (e.g., via HPCA1 or RBOH phosphorylation mutants) while recording downstream hormone outputs should help reveal encoding rules and tissue hierarchies. (Myers et al., 2024; Peláez-Vico et al., 2022; Zandalinas et al., 2020).

### 9.6. Organelle–organelle dialogues and retrograde control

Chloroplasts, mitochondria, and peroxisomes are dominant ROS sources/sinks and sites of hormone biosynthesis and catabolism, yet we lack integrative models of how organelle redox states steer hormonal outputs (e.g., chloroplast ROS impacts on SA/JA trade-offs; peroxisomal H₂O₂ on auxin homeostasis). Foundational principles exist, but quantitative coupling and feedback remain unresolved, especially under fluctuating light/temperature (Foyer & Hanke, 2022; Smirnoff & Arnaud, 2019). High-frequency *in vivo* redox imaging across multiple organelles, combined with compartment‐specific ‘omics, should be a prime focus. (Foyer & Hanke, 2022; Smirnoff & Arnaud, 2019).

### 9.7. Stress combinations and environmental realism

Hormonal control of oxidative stress has mostly been dissected under single-stress lab conditions. Field-relevant stress combinations (heat × drought, salinity × pathogen) produce emergent ROS-hormone behaviors not predictable from single stresses (Zandalinas et al., 2020). Multi-stress trials that pair redox biosensors with hormone reporters in realistic microclimates—and include chronic priming histories—are urgently needed. Standardized datasets across species/environments would enable meta-analyses and ML-guided hypothesis generation for hormone/ROS intervention points. (Zandalinas et al., 2020; Mittler et al., 2022).

### 9.8. From mechanism to breeding and gene editing

Translation lags mechanism. Few hormone-ROS nodes validated in model species have been engineered in crops and tested across sites/years. Candidate nodes include extracellular ROS perception (HPCA1), RBOH regulatory circuits, and chloroplast redox buffering capacity; all are plausible levers to tune stress resilience with minimal growth penalties. Genome editing of receptor/kinase motifs and promoter swaps for ROS enzymes, combined with trait-linked field imaging (thermal/fluorescence proxies for redox state), can help close the gap. Coordinated pipelines—from discovery, to physiological validation, to multi-location trials—are a key community need. (Wu et al., 2020; Lee et al., 2020; Foyer & Hanke, 2022; Mittler et al., 2022).

### 9.9. Theory: quantitative, testable network models

Finally, hormone–ROS research needs explicit, falsifiable models that integrate receptor kinetics, ROS production/scavenging, redox buffering, transcriptional control, and systemic signaling. Model-driven experiment design—rather than post-hoc fitting—should become routine, with parameters constrained by standardized sensor data and proteomics. Cross-scale frameworks that bridge organelle reactions to plant-level acclimation under fluctuating environments would advance predictive control of oxidative stress. (Mittler et al., 2022; Myers et al., 2024; Rowe et al., 2025).

## 10. Conclusions

The cumulative evidence from the preceding sections indicates that hormones and ROS form an integrated regulatory nexus that balances growth, development, and stress resilience. Key take-home points can be summarized as follows:

1. **ROS–hormone reciprocity:** Hormones actively modulate ROS production, scavenging, and signaling, while ROS act as second messengers to fine-tune hormone responses. This reciprocity forms the biochemical basis for adaptive responses.
2. **Crosstalk is multi-layered:** Interactions between ABA, SA, JA, auxin, ethylene, cytokinins, and BRs converge on ROS hubs such as NADPH oxidases, antioxidant enzymes, and thiol-redox relays, generating both synergistic and antagonistic effects.
3. **Organelle specificity matters:** Chloroplasts, mitochondria, and peroxisomes each contribute distinct ROS pools and retrograde signals, which intersect with hormone pathways in unique ways. Subcellular targeting of research and interventions will be critical.
4. **Priming as a practical tool:** Hormonal priming creates redox-based memory that enables cross-protection against diverse stresses. This mechanism is increasingly attractive for crop management and climate resilience.
5. **Molecular nodes identified:** Receptors (e.g., PYLs, HPCA1), kinases (SnRK2s, MAPKs), transcription factors (NACs, bZIPs), and redox sensors are emerging as key “choke points” where hormonal and ROS signals integrate.
6. **Systemic integration:** Long-distance ROS waves and hormonal transport collaborate to coordinate systemic acclimation. However, decoding the “syntax” of these systemic signals remains an open challenge.
7. **Translational promise:** Engineering ABA and BR pathways, deploying hormone agonists, and leveraging PGPR/hormone–redox interactions already show yield and stress-resilience benefits in field trials.
8. **Knowledge gaps persist:** Specificity of ROS receptors, combinatorial post-translational regulation, stress-combination responses, and single-cell resolution of hormone–ROS dynamics remain major bottlenecks.
9. **Future-ready pipelines:** Integration of advanced biosensors, single-cell and spatial omics, nanocarrier delivery systems, and precision editing will be crucial for translating hormone–ROS knowledge into next-generation crop improvement.
10. **Balancing growth and defense:** Ultimately, translational success depends on finding interventions that enhance oxidative stress tolerance while minimizing penalties to growth and yield—a balance that must be managed at the molecular, physiological, and agronomic levels.

## References

Alam, A., Ullah, H., Thuenprom, N., Tisarum, R., Cha-Um, S., & Datta, A. (2022). Seed priming with salicylic acid enhances growth, physiological traits, fruit yield, and quality parameters of cantaloupe under water-deficit stress. South African Journal of Botany, 150, 1–12. <https://doi.org/10.1016/j.sajb.2022.06.056>

Arnaud, D., Lee, S., Takebayashi, Y., Choi, D., Choi, J., Sakakibara, H., & Hwang, I. (2017). Cytokinin-mediated regulation of reactive oxygen species homeostasis modulates stomatal immunity in Arabidopsis. The Plant Cell, 29(3), 543–559. <https://doi.org/10.1105/tpc.16.00583>

Avalbaev, A., Fedyaev, V., Lubyanova, A., Yuldashev, R., & Allagulova, C. (2024). 24-Epibrassinolide reduces drought-induced oxidative stress by modulating the antioxidant system and respiration in wheat seedlings. Plants, 13(2), 148. <https://doi.org/10.3390/plants13020148>

Baroi, A., Ritu, S. A., Khan, M. S. U., Uddin, M. N., Hossain, M. A., & Haque, M. S. (2024). Abscisic acid and glycine betaine-mediated seed and root priming enhance seedling growth and antioxidative defense in wheat under drought. Heliyon, 10(9), e30598. <https://doi.org/10.1016/j.heliyon.2024.e30598>

Bienert, G. P., & Chaumont, F. (2014). Aquaporin-facilitated transmembrane diffusion of hydrogen peroxide. Biochimica et Biophysica Acta (BBA) – General Subjects, 1840(5), 1596–1604. <https://doi.org/10.1016/j.bbagen.2013.09.017>

Caarls, L., Pieterse, C. M. J., & Van Wees, S. C. M. (2015). How salicylic acid takes transcriptional control over jasmonic acid signaling. Frontiers in Plant Science, 6, 170. <https://doi.org/10.3389/fpls.2015.00170>

Chan, K. X., Mabbitt, P. D., Phua, S. Y., Mueller, J. W., Nisar, N., Gigolashvili, T., Stroeher, E., Grassl, J., Arlt, W., Estavillo, G. M., Jackson, C. J., & Pogson, B. J. (2016). Sensing and signaling of oxidative stress in chloroplasts by inactivation of the SAL1 phosphoadenosine phosphatase. Proceedings of the National Academy of Sciences of the United States of America, 113(31), E4567–E4576. <https://doi.org/10.1073/pnas.1604936113>

Chen, Y., Zhang, T., Zhang, X., & Li, S. (2023). Single-cell and spatial transcriptomics: New opportunities and challenges in plant systems biology. Frontiers in Plant Science, 14, 1185377. <https://doi.org/10.3389/fpls.2023.1185377>

Corpas FJ, González-Gordo S and Palma JM (2020) Plant Peroxisomes: A Factory of Reactive Species. Front. Plant Sci. 11:853. <https://doi.org/10.3389/fpls.2020.00853>

Dong, N., Yin, W., Gao, Y., Jing, H., Guo, Z., Wang, S., … Tong, H. (2020). Regulation of brassinosteroid signaling and salt resistance by SERK2 and potential utilization for crop improvement in rice. Frontiers in Plant Science, 11, 621859. <https://doi.org/10.3389/fpls.2020.621859>

Dubiella, U., Seybold, H., Durian, G., Komander, E., Lassig, R., Witte, C.-P., & Romeis, T. (2013). Calcium-dependent protein kinase/NADPH oxidase activation circuit is required for rapid defense signal propagation. Proceedings of the National Academy of Sciences of the United States of America, 110(21), 8744–8749. <https://doi.org/10.1073/pnas.1221294110>

Dvořák, P., Krasylenko, Y., Zeiner, A., Šamaj, J., & Takáč, T. (2021). Signaling toward reactive oxygen species-scavenging enzymes in plants. Frontiers in Plant Science, 11, 618835. <https://doi.org/10.3389/fpls.2020.618835>

Ellouzi, H., Ben Slimene Debez, I., Amraoui, S., Rabhi, M., Hanana, M., Alyami, N. M., Debez, A., Abdelly, C., & Zorrig, W. (2024). Effect of seed priming with auxin on ROS detoxification and carbohydrate metabolism and their relationship with germination and early seedling establishment in salt stressed maize. BMC plant biology, 24(1), 704. <https://doi.org/10.1186/s12870-024-05413-w>

Fichman, Y., Zandalinas, S. I., Peck, S., Luan, S., & Mittler, R. (2022). HPCA1 is required for systemic reactive oxygen species and calcium cell-to-cell signaling and plant acclimation to stress. The Plant Cell, 34(11), 4453–4471. <https://doi.org/10.1093/plcell/koac241>

Foyer, C. H., & Hanke, G. (2022). ROS production and signalling in chloroplasts: Cornerstones and evolving concepts. The Plant Journal, 111(3), 642–661. <https://doi.org/10.1111/tpj.15856>

Giannelli, G., Saltzgiver, G., Egamberdieva, D., & Bano, A. (2023). The contribution of PGPR in salt stress tolerance in crops: Unravelling the molecular mechanisms of cross-talk between plant and bacteria. Plants, 12(11), 2197. <https://doi.org/10.3390/plants12112197>

Gohari, G., Jiang, M., Manganaris, G. A., Zhou, J., & Fotopoulos, V. (2024). Next generation chemical priming: With a little help from our nanocarrier friends. Trends in Plant Science, 29(2), 150–166. <https://doi.org/10.1016/j.tplants.2023.11.024>

Han, S., Zhang, J., Wang, W., Zhang, S., Qin, Z., & Pei, H. (2024). Reactive oxygen and related regulatory factors involved in ethylene-induced petal abscission in roses. Plants, 13(13), 1718. <https://doi.org/10.3390/plants13131718>

Harris, C. J., Amtmann, A., & Ton, J. (2023). Epigenetic processes in plant stress priming: Open questions and new approaches. Current Opinion in Plant Biology, 75, 102432. <https://doi.org/10.1016/j.pbi.2023.102432>

He, C., Liew, L. C., Yin, L., Lewsey, M. G., Whelan, J., & Berkowitz, O. (2022). The retrograde signalling regulator ANAC017 recruits the MKK9–MPK3/6, ethylene, and auxin signalling pathways to balance mitochondrial dysfunction with growth. The Plant Cell, 34(9), 3460–3481. <https://doi.org/10.1093/plcell/koac177>

Hou, X., Lee, L. Y. C., Xia, K., Yan, Y., & Yu, H. (2010). DELLAs modulate jasmonate signaling via competitive binding to JAZs. Developmental Cell, 19(6), 884–894. <https://doi.org/10.1016/j.devcel.2010.10.024>

Huang, J., Willems, P., Wei, B., Tian, C., Ferreira, R. B., Bodra, N., Martínez Gache, S. A., Wahni, K., Liu, K., Vertommen, D., Gevaert, K., Carroll, K. S., Van Montagu, M., Yang, J., Van Breusegem, F., & Messens, J. (2019). Mining for protein S-sulfenylation in *Arabidopsis* uncovers redox-sensitive sites. *Proceedings of the National Academy of Sciences of the United States of America*, *116*(42), 21256–21261. https://doi.org/10.1073/pnas.1906768116

Huang, X., et al. (2022). Peroxisome-mediated reactive oxygen species signals in plant stress and development. International Journal of Molecular Sciences, 23(17), 10087. <https://doi.org/10.3390/ijms231710087>

Hudeček, M., Nožková, V., Plíhalová, L., & Plíhal, O. (2023). Plant hormone cytokinin at the crossroads of stress priming and control of photosynthesis. Frontiers in Plant Science, 13, 1103088. <https://doi.org/10.3389/fpls.2022.1103088>

Kambona, C. M., Koua, P. A., Léon, J., & Ballvora, A. (2023). Stress memory and its regulation in plants experiencing recurrent drought conditions. Theoretical and Applied Genetics, 136(2), 26. <https://doi.org/10.1007/s00122-023-04313-1>

Kim, H., Lee, K., Hwang, H., Bhatnagar, N., Kim, D. Y., Yoon, I. S., … Kim, B. G. (2014). Overexpression of PYL5 in rice enhances drought tolerance, inhibits growth, and modulates gene expression. Journal of Experimental Botany, 65(2), 453–464. <https://doi.org/10.1093/jxb/ert397>

Kotera, Y., Asai, Y., Okano, S., Tokutake, Y., Hosomi, A., Saito, K., Yonekura, S., & Katou, S. (2024). Peroxisomal localization of benzyl alcohol O-benzoyltransferase HSR201 is mediated by a non-canonical peroxisomal targeting signal and required for salicylic acid biosynthesis. Plant and Cell Physiology, 65(12), 2054–2065. <https://doi.org/10.1093/pcp/pcae129>

Kumar, A., Verma, K., Kashyap, R., Joshi, V. J., Sircar, D., & Yadav, S. R. (2024). Auxin-responsive ROS homeostasis genes display dynamic expression pattern during rice crown root primordia morphogenesis. Plant Physiology and Biochemistry, 206, 108307. <https://doi.org/10.1016/j.plaphy.2023.108307>

Kumawat, K. C., Sharma, B., Nagpal, S., Kumar, A., Tiwari, S., & Nair, R. M. (2023). Plant growth-promoting rhizobacteria: Salt stress alleviators to improve crop productivity for sustainable agriculture development. Frontiers in plant science, 13, 1101862. https://doi.org/10.3389/fpls.2022.1101862

Lee, D. H., Lal, N. K., Lin, Z.-J. D., Ma, S., Liu, J., Toruño, T., … Coaker, G. (2020). Regulation of reactive oxygen species during plant immunity through phosphorylation and ubiquitination of RBOHD. Nature Communications, 11, 1838. <https://doi.org/10.1038/s41467-020-15601-5>

Lee, J., Han, M., Shin, Y., Lee, J. M., Heo, G., & Lee, Y. (2023). How extracellular reactive oxygen species reach their intracellular targets in plants. Molecules and Cells, 46(6), 329–336. <https://doi.org/10.14348/molcells.2023.2158>

Li, L., Li, M., Yu, L., Zhou, Z., Liang, X., Liu, Z., Cai, G., Gao, L., Zhang, X., Wang, Y., Chen, S., & Zhou, J. M. (2014). The FLS2-associated kinase BIK1 directly phosphorylates the NADPH oxidase RbohD to control plant immunity. Cell host & microbe, 15(3), 329–338. https://doi.org/10.1016/j.chom.2014.02.009

Li, S., Liu, S., Zhang, Q., Cui, M., Zhao, M., Li, N., Wang, S., Wu, R., Zhang, L., Cao, Y., & Wang, L. (2022). The interaction of ABA and ROS in plant growth and stress resistances. Frontiers in Plant Science, 13, 1050132. <https://doi.org/10.3389/fpls.2022.1050132>

Liebthal, M., Maynard, D., & Dietz, K.-J. (2018). Peroxiredoxins and redox signaling in plants. Antioxidants & Redox Signaling, 28(7), 609–624. <https://doi.org/10.1089/ars.2017.7164>

Liu, C., Liu, Q., & Mou, Z. (2024). Redox signaling and oxidative stress in systemic acquired resistance. Journal of Experimental Botany, 75(15), 4535–4548. <https://doi.org/10.1093/jxb/erae193>

Liu, H., Able, A. J., & Able, J. A. (2022). Priming crops for the future: Rewiring stress memory. Trends in Plant Science, 27(8), 699–716. <https://doi.org/10.1016/j.tplants.2021.11.015>

Ma, Y., Szostkiewicz, I., Korte, A., Moes, D., Yang, Y., Christmann, A., & Grill, E. (2009). Regulators of PP2C phosphatase activity function as abscisic acid sensors. Science, 324(5930), 1064–1068. <https://doi.org/10.1126/science.1172408>

Mahati, K., & Padmasree, K. (2023). Brassinolide promotes interaction between chloroplasts and mitochondria during the optimization of photosynthesis by the mitochondrial electron transport chain in mesophyll cell protoplasts of Arabidopsis thaliana. Frontiers in Plant Science, 14, 1099474. [https://doi.org/10.3389/fpls.2023.1099474](https://doi.org/10.3389/fpls.2023.1099474?utm_source=chatgpt.com)

Mansoor, S., Ali Wani, O., Lone, J. K., Manhas, S., Kour, N., Alam, P., Ahmad, A., & Ahmad, P. (2022). Reactive oxygen species in plants: From source to sink. Antioxidants, 11(2), 225. [https://doi.org/10.3390/antiox11020225](https://doi.org/10.3390/antiox11020225?utm_source=chatgpt.com)

Mao, H., Jian, C., Cheng, X., Chen, B., Mei, F., Li, F., … Zhang, X. (2022). The wheat ABA receptor gene TaPYL1-1B contributes to drought tolerance and grain yield by increasing water-use efficiency. Plant Biotechnology Journal, 20(5), 846–861. <https://doi.org/10.1111/pbi.13764>

Martin, R. E., Marzol, E., Estévez, J. M., & Muday, G. K. (2022). Ethylene signaling increases reactive oxygen species accumulation to drive root hair initiation in Arabidopsis. Development, 149(13), dev200487. <https://doi.org/10.1242/dev.200487>

Mase, K. (2021). Reactive oxygen species link gene regulatory networks: A review. Frontiers in Plant Science, 12, 660274. <https://doi.org/10.3389/fpls.2021.660274>

Matkowski, H., & Daszkowska-Golec, A. (2025). Wisdom comes after facts—An update on plants priming using phytohormones. Journal of Plant Physiology, 154414. <https://doi.org/10.1016/j.jplph.2024.154414>

Meyer, Y., Belin, C., Delorme-Hinoux, V., Reichheld, J.-P., & Riondet, C. (2012). Thioredoxin and glutaredoxin systems in plants: Molecular mechanisms, crosstalks, and functional significance. Antioxidants & Redox Signaling, 17(8), 1124–1160. <https://doi.org/10.1089/ars.2011.4327>

Mittler, R., Zandalinas, S. I., Fichman, Y., & Van Breusegem, F. (2022). Reactive oxygen species signalling in plant stress responses. Nature Reviews Molecular Cell Biology, 23(10), 663–679. [https://doi.org/10.1038/s41580-022-00499-2](https://doi.org/10.1038/s41580-022-00499-2?utm_source=chatgpt.com)

Mou, Z., Fan, W., & Dong, X. (2003). Inducers of plant systemic acquired resistance regulate NPR1 function through redox changes. Cell, 113(7), 935–944. [https://doi.org/10.1016/S0092-8674(03)00429-X](https://doi.org/10.1016/S0092-8674%2803%2900429-X)

Müller-Schüssele, S. J., Schwarzländer, M., & Meyer, A. J. (2021). Live monitoring of plant redox and energy physiology with genetically encoded biosensors. Plant physiology, 186(1), 93–109. <https://doi.org/10.1093/plphys/kiab019>

Murphy, M. P., Bayır, H., Belousov, V., Chang, C.-J., Davies, K. J. A., Davies, M. J., … Winterbourn, C. C. (2022). Guidelines for measuring reactive oxygen species and oxidative damage in cells and in vivo. Nature Metabolism, 4(6), 651–662. <https://doi.org/10.1038/s42255-022-00591-z>

Myers, R. J., Fichman, Y., Zandalinas, S. I., & Mittler, R. (2023). Jasmonic acid and salicylic acid modulate systemic reactive oxygen species signaling during stress responses. Plant Physiology, 191(2), 862–873. <https://doi.org/10.1093/plphys/kiac449>

Myers, R. J., Zandalinas, S. I., Fichman, Y., & Mittler, R. (2024). Functional analysis of reactive oxygen species-driven stress systemic signalling, interplay and acclimation. Plant, Cell & Environment, 47(8), 2842–2851. <https://doi.org/10.1111/pce.14894>

Ng, S., Ivanova, A., Duncan, O., Law, S. R., Van Aken, O., De Clercq, I., Wang, Y., Carrie, C., Xu, L., Kmiec, B., Walker, H., Van Breusegem, F., Whelan, J., & Giraud, E. (2013). A membrane-bound NAC transcription factor, ANAC017, mediates mitochondrial retrograde signaling in Arabidopsis. The Plant Cell, 25(9), 3450–3471. <https://doi.org/10.1105/tpc.113.113985>

Nietzel, T., Elsässer, M., Ruberti, C., Steinbeck, J., Ugalde, J. M., Fuchs, P., Wagner, S., Ostermann, L., Moseler, A., Lemke, P., Fricker, M. D., Müller-Schüssele, S. J., Moerschbacher, B. M., Costa, A., Meyer, A. J., & Schwarzländer, M. (2019). The fluorescent protein sensor roGFP2-Orp1 monitors in vivo H₂O₂ and thiol redox integration and elucidates intracellular H₂O₂ dynamics during elicitor-induced oxidative burst in Arabidopsis. New Phytologist, 221(3), 1649–1664. <https://doi.org/10.1111/nph.15550>

Park, S.-Y., Fung, P., Nishimura, N., Jensen, D. R., Fujii, H., Zhao, Y., Lumba, S., Santiago, J., Rodrigues, A., Chow, T. F., et al. (2009). Abscisic acid inhibits type 2C protein phosphatases via the PYR/PYL family of START proteins. Science, 324(5930), 1068–1071. <https://doi.org/10.1126/science.1173041>

Peláez-Vico, M. Á., Fichman, Y., Zandalinas, S. I., Van Breusegem, F., Karpiński, S. M., & Mittler, R. (2022). ROS and redox regulation of cell-to-cell and systemic signaling in plants during stress. Free Radical Biology and Medicine, 193(Pt 1), 354–362. <https://doi.org/10.1016/j.freeradbiomed.2022.10.305>

Phua, S. Y., Yan, D., Chan, K. X., Estavillo, G. M., Nambara, E., & Pogson, B. J. (2018). The Arabidopsis SAL1–PAP pathway: A case study for integrating chloroplast retrograde, light and hormonal signaling in modulating plant growth and development? Frontiers in Plant Science, 9, 1171. <https://doi.org/10.3389/fpls.2018.01171>

Postiglione, A. E., & Muday, G. K. (2020). The role of ROS homeostasis in ABA-induced guard cell signaling. Frontiers in Plant Science, 11, 968. <https://doi.org/10.3389/fpls.2020.00968>

Postiglione, A. E., & Muday, G. K. (2023). Abscisic acid increases hydrogen peroxide in mitochondria to facilitate stomatal closure. Plant Physiology, 192(1), 469–487. <https://doi.org/10.1093/plphys/kiac601>

Rehman, M., Khan, F. A., Nazir, M., & Hasanuzzaman, M. (2023). The multifaceted role of jasmonic acid in plant stress mitigation: An overview. Plants, 12(23), 3982. <https://doi.org/10.3390/plants12233982>

Rhaman, M. S., Imran, S., Rauf, F., Khatun, M., Baskin, C. C., Murata, Y., & Hasanuzzaman, M. (2021). Seed priming with phytohormones: An effective approach for the mitigation of abiotic stress. Plants, 10(1), 37. <https://doi.org/10.3390/plants10010037>

Rodrigues, O., Reshetnyak, G., Grondin, A., Saijo, Y., Leonhardt, N., Maurel, C., & Verdoucq, L. (2017). Aquaporins facilitate hydrogen peroxide entry into guard cells to mediate ABA- and pathogen-triggered stomatal closure. Proceedings of the National Academy of Sciences of the United States of America, 114(34), 9200–9205. <https://doi.org/10.1073/pnas.1704754114>

Rowe, L., Heins, C. L., & Frommer, W. B. (2025). Quantifying plant biology with fluorescent biosensors: Best practices, pitfalls, and new opportunities. Annual Review of Plant Biology, 76, In press. <https://doi.org/10.1146/annurev-arplant-061824-090615>

Saleem, M., Fariduddin, Q., & Castroverde, C. D. M. (2021). Salicylic acid: A key regulator of redox signalling and plant immunity. Plant Physiology and Biochemistry, 168, 381–397. <https://doi.org/10.1016/j.plaphy.2021.10.011>

Sandalio, L. M., Peláez-Vico, M. A., Molina-Moya, E., & Romero-Puertas, M. C. (2021). Peroxisomes as redox-signaling nodes in intracellular communication and stress responses. Plant physiology, 186(1), 22–35. https://doi.org/10.1093/plphys/kiab060

Sandalio, L. M., Rodríguez-Serrano, M., Romero-Puertas, M. C., & Corpas, F. J. (2023). Peroxisomes and plant redox signalling: New players and emergent concepts. Free Radical Biology & Medicine, 204, 154–170. <https://doi.org/10.1016/j.freeradbiomed.2023.01.014>

Shapiguzov, A., Vainonen, J. P., Hunter, K., Tossavainen, H., Tiwari, A., Järvi, S., Hellman, M., Aarabi, F., Alseekh, S., Wybouw, B., Van Der Kelen, K., Nikkanen, L., Krasensky-Wrzaczek, J., Sipari, N., Keinänen, M., Tyystjärvi, E., Rintamäki, E., De Rybel, B., Salojärvi, J., ... Kangasjärvi, J. (2019). Arabidopsis RCD1 coordinates chloroplast and mitochondrial functions through interaction with ANAC transcription factors. eLife, 8, e43284. <https://doi.org/10.7554/eLife.43284>

Sheteiwy, M. S., Ulhassan, Z., Qi, W., Lu, H., AbdElgawad, H., Minkina, T., … Dawood, M. (2022). Association of jasmonic acid priming with multiple defense mechanisms in wheat plants under high salt stress. Frontiers in Plant Science, 13, 886862. <https://doi.org/10.3389/fpls.2022.886862>

Smirnoff, N., & Arnaud, D. (2019). Hydrogen peroxide metabolism and functions in plants. New Phytologist, 221(3), 1197–1214. <https://doi.org/10.1111/nph.15488>

Tada, Y., Spoel, S. H., Pajerowska-Mukhtar, K., Mou, Z., Song, J., Wang, C., Zuo, J., & Dong, X. (2008). Plant immunity requires conformational changes of NPR1 via S-nitrosylation and thioredoxins. Science, 321(5891), 952–956. <https://doi.org/10.1126/science.1156970>

Vaidya, A. S., Helander, J. D. M., Peterson, F. C., Elzinga, D., Dejonghe, W., Kaundal, A., … Cutler, S. R. (2019). Dynamic control of plant water use using designed ABA receptor agonists. Science, 366(6464), eaaw8848. <https://doi.org/10.1126/science.aaw8848>

Van Aken, O., Ford, E., Lister, R., Huang, S., & Millar, A. H. (2016). Retrograde signalling caused by heritable mitochondrial dysfunction is partially mediated by ANAC017 and improves plant performance. The Plant Journal, 88(4), 542–558. <https://doi.org/10.1111/tpj.13276>

Verma, R. K., Santosh Kumar, R., Yadav, S. K., Pushkar, S., Rao, M. V., & Chinnusamy, V. (2019). Overexpression of ABA receptor PYL10 gene confers drought and cold tolerance to indica rice. Frontiers in Plant Science, 10, 1488. <https://doi.org/10.3389/fpls.2019.01488>

Wang, P., Liu, W.-C., Han, C., Wang, S., Bai, M.-Y., & Song, C.-P. (2024). Reactive oxygen species: Multidimensional regulators of plant adaptation to abiotic stress and development. Journal of Integrative Plant Biology, 66(3), 330–367. <https://doi.org/10.1111/jipb.13601>

Waszczak, C., Carmody, M., & Kangasjärvi, J. (2018). Reactive oxygen species in plant signaling. Annual Review of Plant Biology, 69, 209–236. <https://doi.org/10.1146/annurev-arplant-042817-040322>

Wu, F., Chi, Y., Jiang, Z., Xu, Y., Xie, L., Huang, F., Wan, D., Ni, J., Yuan, F., Wu, X., Zhang, Y., Wang, L., Ye, R., Byeon, B., Wang, W., Zhang, S., Sima, M., Chen, S., Zhu, M., … Pei, Z.-M. (2020). Hydrogen peroxide sensor HPCA1 is an LRR receptor kinase in Arabidopsis. Nature, 578(7796), 577–581. <https://doi.org/10.1038/s41586-020-2032-3>

Yang, Z., Li, Y., Wang, Y., Zhang, M., & Ren, Y. (2022). Priming seeds for the future: Plant immune memory and application in crop protection. Frontiers in Plant Science, 13, 961840. <https://doi.org/10.3389/fpls.2022.961840>

Yin, L., Liu, J., He, F., Xu, R., Li, J., & Wang, X. (2023). Spatial transcriptomics in plant research: Current applications and perspectives. The Plant Journal, 114(1), 149–166. <https://doi.org/10.1111/tpj.16437>

Zandalinas, S. I., Fritschi, F. B., & Mittler, R. (2020). Systemic signaling during abiotic stress combination in plants. Proceedings of the National Academy of Sciences, 117(24), 13810–13820. <https://doi.org/10.1073/pnas.2005077117>

Zandi, P., & Schnug, E. (2022). Reactive oxygen species, antioxidant responses and implications from a microbial modulation perspective. Biology, 11(2), 155. <https://doi.org/10.3390/biology11020155>

Zavaliev, R., & Dong, X. (2024). NPR1, a key immune regulator for plant survival under biotic and abiotic stresses. Molecular Cell, 84(1), 131–141. <https://doi.org/10.1016/j.molcel.2023.11.018>

Zhang, Z., Shahid, M. Z., Khan, M. I. R., et al. (2023). From plant survival to thriving: Exploring the miracle of brassinosteroids for boosting abiotic stress resilience in horticultural crops. Frontiers in Plant Science, 14, 1218229. <https://doi.org/10.3389/fpls.2023.1218229>

Zhao, Y., Zhang, Z., Gao, J., Wang, P., Hu, T., Wang, Z., … Zhu, J.-K. (2016). ABA receptor PYL9 promotes drought resistance and leaf senescence. Proceedings of the National Academy of Sciences of the United States of America, 113(7), 1949–1954. <https://doi.org/10.1073/pnas.1522840113>

Zhou, Y.-L., You, X.-Y., Wang, X.-Y., Cui, L.-H., Jiang, Z.-H., & Zhang, K.-P. (2024). Exogenous 24-epibrassinolide enhanced drought tolerance and promoted BRASSINOSTEROID-INSENSITIVE2 expression of quinoa. Plants, 13(6), 873. <https://doi.org/10.3390/plants13060873>

Zolkiewicz, K., & Gruszka, D. (2025). Take a deep BReath: Manipulating brassinosteroid homeostasis helps cereals adapt to environmental stress. Plant Physiology, 197(1), kiaf003. <https://doi.org/10.1093/plphys/kiaf003>

Zulfiqar, F. (2021). Effect of seed priming on horticultural crops. Scientia Horticulturae, 286, 110197. <https://doi.org/10.1016/j.scienta.2021.110197>