biomolecules

MDPI

Review

Molecular Mechanisms of Hormone–Transcription Factor Crosstalk in Horticultural Crop Development and Stress Adaptation

Liqin Chen [1,†], Liangcheng Deng [1,†], Jing Dong [1], Jiajing Wu [1], Aneesa Gul [2], Zongqing Qiu [1], Yuan Liu [1], Jiaying Yuan [1], Liangliang Zhang [1], Huilin Wang [1] and Liangliang Hu [1,3,*]

[1] College of Horticulture, Xinjiang Agricultural University, Urumqi 830052, China
[2] College of Coastal Agricultural Sciences, Guangdong Ocean University, Zhanjiang 524088, China
[3] Postdoctoral Station of Horticulture, Xinjiang Agricultural University, Urumqi 830052, China
* Correspondence: hull0501@xjau.edu.cn
† The authors have equally contributed to this work.

Abstract

Hormone signaling in horticultural crops is increasingly understood as a networked transcriptional system rather than a set of linear pathways. Phytohormones, as key regulators of plant growth, development, and stress adaptation, converge on shared repressors, transcription factors, co-regulators, protein-interaction hubs, and chromatin states that determine context-specific outputs. Here, we synthesize the major regulatory architectures linking hormone perception to transcription, including proteolysis, phosphorylation, transcription-factor stabilization, co-regulator recruitment, and chromatin control. We highlight convergence hubs such as DELLA, BZR1/BES1, JAZ–MYC, EIN3/EIL–ERF, WRKY/NAC, and NPR–TGA that coordinate development, stress adaptation, immunity, and postharvest physiology. We propose that future crop improvement will depend on engineering specific regulatory interactions, rather than entire hormone pathways, to achieve precise trait control with reduced pleiotropic costs.

Keywords: phytohormone crosstalk; transcriptional convergence; precision crop engineering

1. Introduction

Plant development, reproduction, and stress adaptation emerge from interconnected hormone–transcription factor (TF) networks rather than independent linear pathways [1,2]. Canonical modules such as TIR1/AFB–Aux/IAA–ARF, GID1–DELLA, COI1–JAZ–MYC, PYR/PYL–PP2C–SnRK2–ABF, BRI1–BIN2–BZR1/BES1, and EIN2–EIN3/ERF provide the core signaling framework [3,4], but they do not operate as closed circuits. Their outputs depend on receptor identity, repressor abundance, protein turnover, phosphorylation, TF partnerships, promoter accessibility, and the transcriptional competence of the responding cell. Recent work illustrates this contextual control: AFB1 partitions rapid auxin responses by timescale and compartment, whereas gibberellin can reshape auxin distribution through PIN1-dependent transport [5,6]. Thus, hormonal specificity is generated by network architecture as much as by the ligand itself.

These network events are especially important in horticultural crops, whose harvestable organs must coordinate growth, texture, pigmentation, flavor, reproduction, stress responses, and postharvest behavior. Perenniality, grafting, clonal propagation, high heterozygosity, and polyploidy further impose stage- and tissue-specific constraints that are

Check for updates

Academic Editor: Kyoungwhan Back

Received: 19 October 2026
Revised: 13 November 2026
Accepted: 16 November 2026
Published: 17 November 2026

Copyright: © 2026 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license.

poorly captured by annual model plants. Crop studies already reveal such integration: peach PpIAA1–PpERF4 couples auxin and ethylene during ripening [7]; strawberry FvRIF and FaRIPK1–FaTCP7 connect ripening with cell-wall remodeling, pigmentation, hormone responses, and sugar transport [8,9]. Apple MdSVPa–DAM complexes regulate dormancy [10]; and cambial auxin signaling promotes graft adhesion and vascular reconnection [11]. These examples show why mechanisms inferred from *Arabidopsis* require direct validation in crop organs and developmental contexts.

Accordingly, this review adopts a conditional framework in which hormone perception reshapes repressors and signaling intermediates, which remodel TF complexes and chromatin occupancy to determine gene expression and phenotype. We first compare the principal molecular architectures of hormone-to-TF signaling, then examine how they govern fruit set, ripening, flowering, dormancy, rooting, senescence, abiotic stress, and immunity. We subsequently focus on shared convergence hubs and emerging strategies for precise manipulation. Because causal evidence remains uneven across horticultural species, emphasis is placed on studies linking hormone status to TF activity, direct DNA or protein interactions, genetic perturbation, and trait-level outcomes. The central argument is that crop improvement will require control of regulatory interactions in the correct tissue, developmental stage, dose, and time window rather than broad activation of entire hormone pathways.

2. Molecular Architecture of Hormone to Transcription Factor Signaling

Hormone signaling reaches transcription through a limited set of recurring regulatory events, including repressor degradation, kinase/phosphatase control, changes in TF stability or nuclear competence, co-regulator exchange, and chromatin-dependent promoter access [12] (Figure 1).

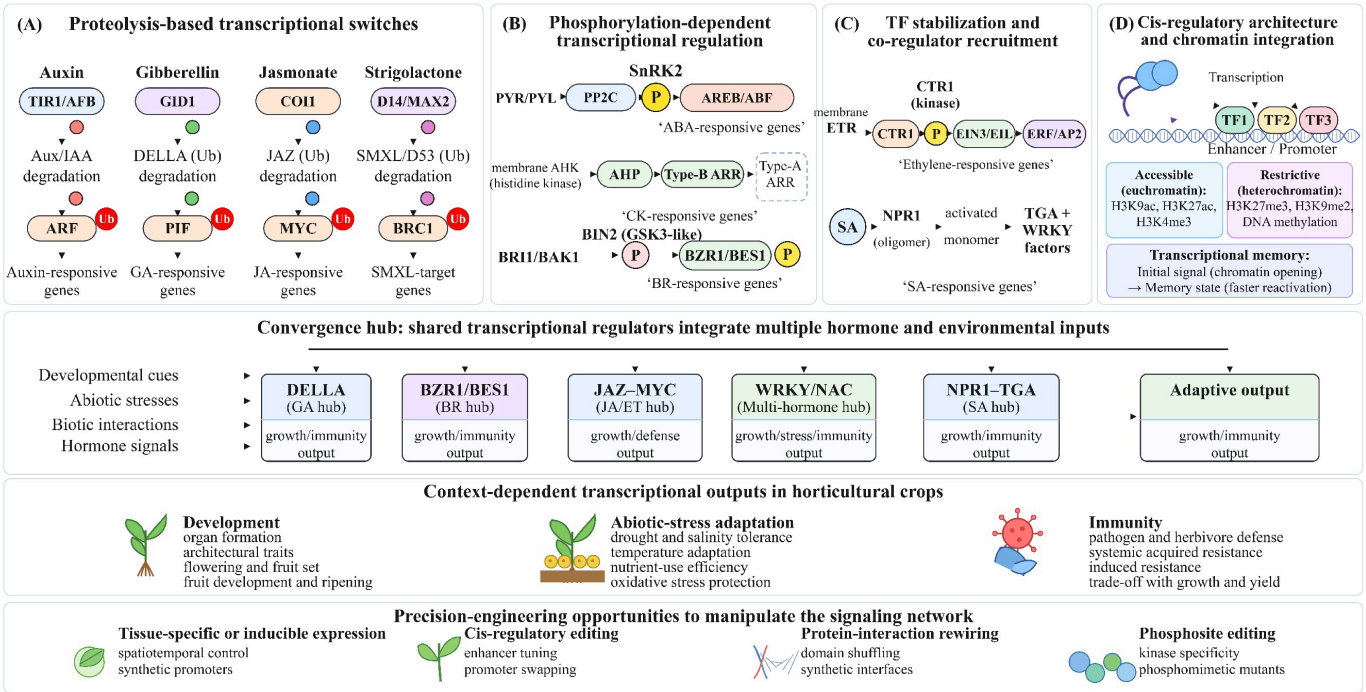


Figure 1. Molecular architecture and convergence of hormone–TF signaling in horticultural crops. Phytohormones regulate transcription through recurring architectures, including repressor proteolysis, phosphorylation-dependent TF control, TF stabilization/co-regulator recruitment, and cis-regulatory or chromatin-level regulation. These pathways converge on shared hubs such as DELLA, BZR1/BES1, JAZ–MYC, EIN3/EIL–ERF, WRKY/NAC, and NPR–TGA, whose context-dependent outputs govern development, stress adaptation, and immunity while highlighting opportunities for precision engineering.

2.1. Proteolysis-Based Transcriptional Switches

Proteolysis-based signaling converts hormone perception into transcriptional responses by promoting the ubiquitin-dependent degradation of regulatory repressors [13]. Because repressor removal can outlast a transient hormone pulse, these pathways provide a mechanism for sustained yet resettable transcriptional regulation. In auxin signaling, TIR1/AFB receptors facilitate Aux/IAA degradation, releasing ARFs to regulate target genes according to their interacting partners and promoter context [14,15]. In tomato, the SlARF7–SlIAA9 module restricts fruit initiation before pollination, illustrating how auxin-dependent transcription is developmentally specified [16]. Gibberellin (GA) similarly promotes GID1-dependent DELLA degradation through the SCF^SLY1/GID2 ubiquitin ligase, releasing growth-associated regulators, including PIFs, BZR1/BES1, and ARFs [17,18]. In grapevine, GA-induced *VvDELLA2* degradation releases *VvCEB1* to activate cell-expansion genes during berry enlargement [19]. Jasmonate (JA) and strigolactone (SL) extend this regulatory logic: JA-Ile promotes COI1-dependent JAZ degradation to release MYC-associated transcriptional programs [20], whereas D14–MAX2 signaling targets SMXL/D53 repressors to regulate branching and developmental plasticity [21]. In pea, D53/SMXL proteins participate in the RMS branching network and feedback on RMS1 expression, linking repressor turnover to hormone biosynthesis [22]. Thus, proteolysis provides a shared signaling mechanism, whereas specificity emerges from the identity of the degraded repressor, the regulatory proteins released, and the developmental context of their target genes.

2.2. Phosphorylation-Dependent Transcriptional Regulation

Phosphorylation-dependent signaling regulates TF activity, localization, and protein interactions through opposing kinase and phosphatase activities, enabling rapid and often reversible responses to hormonal and environmental cues [23,24]. In ABA signaling, PYR/PYL receptors inhibit clade-A PP2Cs, releasing SnRK2 kinases to phosphorylate AREB/ABF factors and activate stress-responsive transcription [25]. This pathway also connects transcriptional control with physiological and metabolic protection: OST1/SnRK2.6 phosphorylates the SLAC1 anion channel to promote stomatal closure [26], whereas SnRK2.4-mediated ABF2 phosphorylation enhances arginine decarboxylase expression and putrescine accumulation during drought [27]. Cytokinin signaling employs a distinct multistep phosphorelay in which histidine-kinase receptors transfer phosphate through HPt proteins to nuclear type-B ARRs, activating cytokinin-responsive genes, including type-A ARRs that provide negative feedback [28]. BR signaling, by contrast, regulates TF activity through a kinase–phosphatase switch: BRI1/BAK1-dependent signaling suppresses BIN2 activity, while PP2A-mediated dephosphorylation promotes nuclear accumulation and transcriptional activity of BZR1/BES1 [29]. Through interactions with DELLA, PIF, ARF, and ethylene-associated regulators, BZR1/BES1 integrate BR signaling with other hormonal and environmental inputs [30,31]. Together, these pathways demonstrate how phosphorylation controls the activity of shared regulatory proteins, complementing proteolysis-dependent regulation of repressor abundance. Their developmental and stress-specific outputs depend on kinase–substrate specificity, feedback regulation, TF partnerships, and target-promoter context.

2.3. Hormone-Controlled Transcription Factor Stabilization and Nuclear Competence

Ethylene provides a canonical example in which hormone perception regulates transcription primarily by controlling TF stability, co-regulator recruitment, and chromatin competence. In the absence of ethylene, endoplasmic-reticulum-localized ethylene receptors maintain *CTR1* kinase activity, which phosphorylates EIN2 and suppresses downstream

signaling. Ethylene perception attenuates receptor–CTR1 activity, enabling EIN2 C-terminal signaling and nuclear responses [32]. EIN2 also represses translation of EBF1 and EBF2 mRNAs and directs them toward processing bodies; because EBF1/EBF2 promote proteasomal turnover of EIN3/EIL proteins, their reduction permits EIN3/EIL accumulation and activation of downstream ERF-centered transcriptional programs [33]. In tomato, this response extends beyond TF stabilization. MED25 physically interacts with EIL1–EIL4 and is recruited to EIL-bound promoters, coupling ethylene-responsive TFs to the RNA polymerase II pre-initiation machinery. The EIL–MED25 module directly regulates both structural and regulatory ripening genes and establishes positive and negative feedback circuits that fine-tune ethylene homeostasis [34]. Ethylene signaling can additionally modify transcriptional competence at the chromatin level; HAF2 interacts with EIN2-C and promotes H3K14/H3K23 acetylation, linking hormone perception to chromatin activation [35]. SA employs a distinct but conceptually related nuclear mechanism. SA promotes the transcriptional activity of NPR1, which assembles with promoter-bound TGA factors to activate defense genes; structural analyses show that NPR1 can bridge TGA3 dimers within an SA-responsive enhanceosome. SA signaling is further modulated by BIN2-dependent phosphorylation of *TGA3* at Ser33, which enhances *TGA3* DNA binding and NPR1–TGA3 complex formation [36]. Recent structural evidence further demonstrates that SA-bound NPR1 recruits MED15A, directly connecting the NPR1–TGA complex to Mediator and the general transcription machinery [37]. Together, these pathways demonstrate that hormonal transcriptional output depends not simply on TF abundance, but on the coordinated regulation of protein stability, post-translational modification, chromatin state, and co-regulator assembly.

2.4. Transcriptional Co-Regulators and Protein-Interaction Networks

Hormone-responsive transcription factors rarely function as autonomous regulators; instead, their output is determined by dynamic assemblies of co-repressors, co-activators, Mediator subunits, and signaling proteins that connect sequence-specific TFs to chromatin and the RNA polymerase II machinery. Aux/IAA proteins, for example, recruit TOPLESS/TOPLESS-RELATED (TPL/TPR) co-repressors to suppress ARF-dependent transcription, whereas JAZ proteins establish repressive complexes around MYC factors, often through NINJA–TPL/TPR recruitment [38]. DELLA proteins operate differently, largely by directly binding and sequestering transcriptional regulators such as PIFs, BZR1/BES1, ARFs, and JAZ-associated factors [3]. Thus, hormone-dependent degradation or modification of these repressors remodels entire transcriptional complexes rather than merely releasing individual TFs.

Importantly, co-repressors can act directly on the general transcription machinery. In *Arabidopsis*, TPL contains a repression domain that interacts with the Mediator middle-module subunits MED10 and MED21, and disruption of this interaction compromises transcriptional repression, demonstrating that pathway-specific repressors can modulate core Mediator activity [39]. Conversely, Mediator can function as a signal-selective co-activator. In tomato, *MED25* is recruited to ethylene-responsive promoters through EIN3/EIL proteins and promotes pre-initiation-complex formation during fruit ripening [34]. Recent structural work further shows that SA-bound *NPR1* recruits *MED15A*, directly linking hormone perception to Mediator-dependent immune transcription [37]. Mediator-dependent transcriptional specificity may additionally arise from differences in subunit abundance, tissue-specific regulatory partners, and selective recruitment to hormone-responsive promoters. In tomato, *MED25* is essential for normal ethylene-regulated fruit ripening, illustrating functional specialization among Mediator subunits [34]. However, the extent to which tissue-specific Mediator expression determines distinct hormonal outputs across

horticultural organs remains to be established. Collectively, competitive partner binding, co-regulator exchange, post-translational modification, subcellular sequestration, and selective protein turnover provide a molecular basis for signal prioritization, allowing the same TF to produce distinct transcriptional outputs according to hormonal, developmental, and environmental context.

2.5. Cis-Regulatory Architecture and Chromatin Remodeling

The final specificity of hormone-responsive transcription is encoded at the level of *cis*-regulatory architecture and chromatin state [40]. Hormone-responsive elements, combinatorial TF-binding motifs, enhancers, promoter organization, and motif spacing determine whether an activated TF can engage a particular target gene [41]. Consequently, the presence of a cognate TF alone is insufficient to predict transcriptional output; regulatory competence also depends on the accessibility and local configuration of its binding sites [42].

Chromatin provides an additional regulatory filter. Histone acetylation and methylation, nucleosome positioning, DNA methylation, and ATP-dependent chromatin-remodeling complexes can expose or occlude hormone-responsive regulatory elements, thereby establishing permissive or refractory transcriptional states. Hormonal and environmental signals can in turn alter chromatin accessibility and co-regulator recruitment, allowing identical signaling inputs to generate different outputs among cell types, developmental stages, and organs [43]. Chromatin regulation can also provide transcriptional memory. Persistent histone modifications, altered nucleosome occupancy, or retained TF/co-regulator configurations may prime selected loci for faster or stronger responses to subsequent developmental or environmental signals. Hormone signaling should therefore be viewed as a series of conditional regulatory filters from receptor activation and protein turnover to TF-complex assembly, chromatin accessibility, and promoter selection that collectively determine when, where, and to what extent a hormone signal is converted into gene expression and phenotype.

3. Hormone–Transcription Factor Networks Governing Horticultural Crop Development

Hormonal regulation of horticultural development is best understood as a dynamic network of interacting transcriptional modules rather than a series of independent pathways. For horticultural crop improvement, the principal opportunity therefore lies not in broad manipulation of hormone levels, but in precise modification of regulatory nodes that shift developmental thresholds while minimizing pleiotropic effects on growth, fertility, and fruit quality. These interacting networks coordinate developmental transitions from fruit initiation and ripening to flowering, dormancy, rooting, and postharvest senescence (Figure 2), while representative crop-validated hormone–TF modules and their mechanistic evidence are summarized in Table 1.

3.1. Fruit Set and Early Fruit Development

Fruit set represents a developmental switch in which fertilization releases the ovary from a quiescent state and initiates rapid sink establishment and growth. This transition is governed principally by auxin–gibberellin crosstalk, whereby auxin promotes TIR1/AFB-dependent degradation of Aux/IAA repressors and activation of ARF transcription factors, while increased GA signaling promotes GID1-mediated DELLA degradation to relieve growth restraint [17,44]. Direct evidence from diploid strawberry places these pathways within a shared regulatory module: the DELLA protein *FveRGA1* physically interacts with *FveARF8*, loss of *FveRGA1* induces parthenocarpy, and disruption of either *FveRGA1* or *FveARF8* increases fruit size [17]. These findings indicate that fruit initiation and sub-

sequent expansion are controlled not simply by hormone abundance, but by the state of transcriptional repressors and their interacting TFs. Cytokinin- and brassinosteroid-responsive regulators likely further shape proliferative and expansion capacity [17], but the ARF–DELLA interface currently provides one of the clearest mechanistic frameworks for fruit-set control [44].

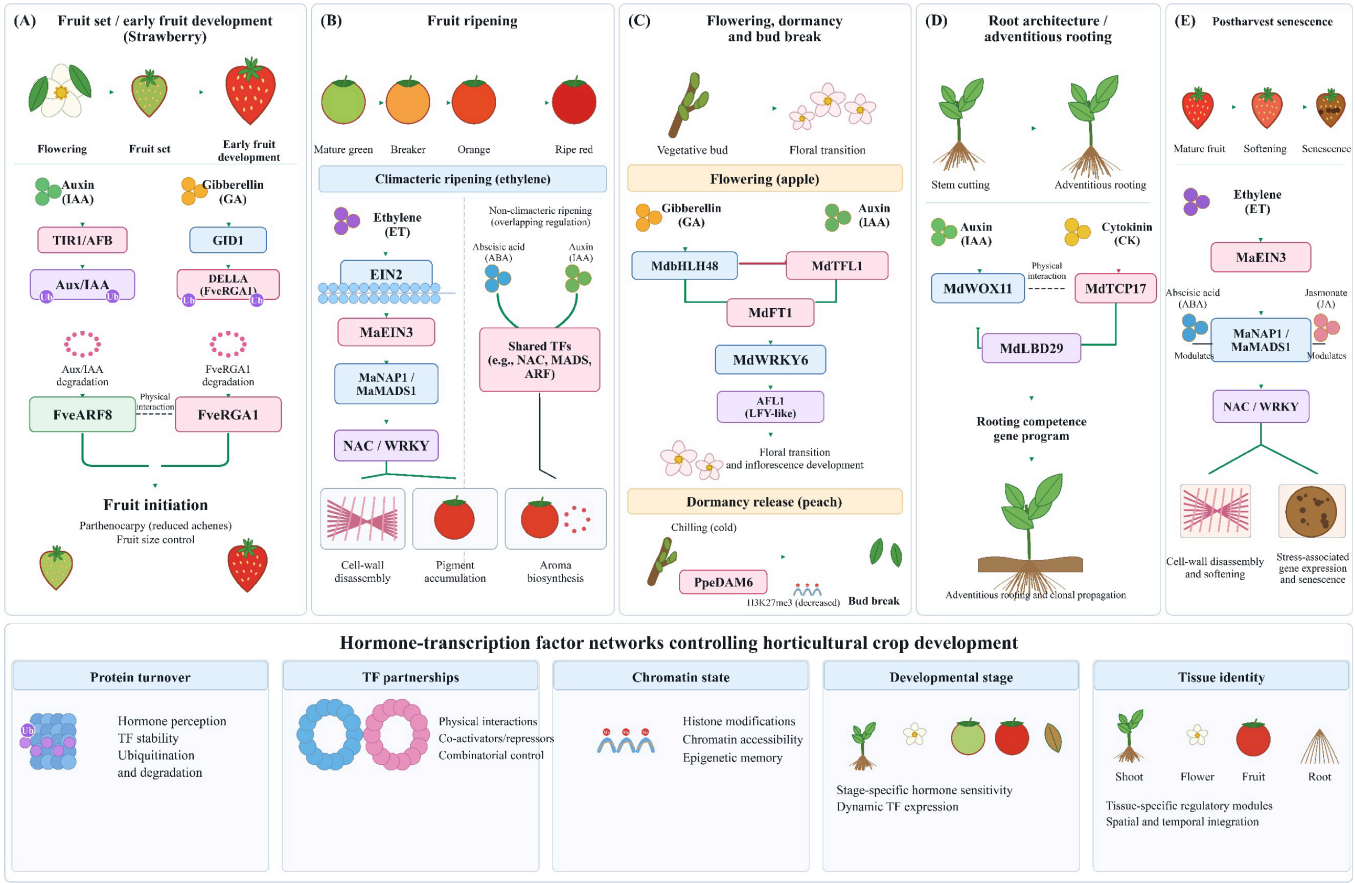


Figure 2. Hormone–TF networks controlling horticultural crop development. Representative crop-validated hormone–TF modules coordinate fruit set, ripening, flowering, dormancy release, adventitious rooting, and postharvest senescence. Examples include the strawberry FveARF8–FveRGA1 module, the banana MaEIN3–MaNAP1/MaMADS1 cascade, the apple MdbHLH48–MdFT1/MdTFL1–MdWRKY6–AFL1 flowering module, peach *PpeDAM6*-associated dormancy release, and the MdTCP17–MdWOX11–MdLBD29 rooting pathway, illustrating how shared hormonal logic governs distinct developmental transitions.

3.2. Hormone–Transcription Factor Networks in Fruit Ripening

Fruit ripening is governed by interacting hormone–transcription factor networks rather than a single dominant hormonal switch. In climacteric fruits, autocatalytic ethylene provides a major regulatory input through EIN3/EIL–ERF cascades that intersect with NAC, MADS-box, and MYB factors controlling ethylene biosynthesis, cell-wall remodeling, pigmentation, aroma, and softening [45]. Recent mechanistic work in banana illustrates this hierarchical architecture. *MaEIN3* directly activates *MaNAP1* and *MaMADS1*, which in turn recruit broader NAC/WRKY regulatory programs controlling ripening-associated genes; perturbation of *MaEIN3* correspondingly alters ethylene production, yellowing, and softening [46]. Further evidence highlights the contributions of NAC and WRKY TFs to fruit-quality regulation. In banana, MaWRKY49/111 interact with MabHLH28 to enhance the transcription of fruit-softening-related genes, linking WRKY-associated regulatory com-

plexes with fruit texture and ripening [47]. In grapevine, *NAC61* directly regulates *MYB14*, *DHN1b*, and *WRKY52* and cooperates with *NAC60*, connecting late ripening and postharvest dehydration with stilbenoid metabolism and stress responses [48]. These findings underscore the importance of NAC- and WRKY-associated transcriptional networks in coordinating fruit development, quality attributes, and postharvest physiology. Tomato further demonstrates that ripening is distributed across multiple regulators rather than governed by a solitary master switch. RIN, NOR, and extensive TF networks are progressively reconfigured during the mature-green-to-breaker transition, together with coordinated changes in auxin, ABA, JA, BR, cytokinin, and GA signaling [49].

In contrast to climacteric fruits, such as tomato and banana, which exhibit a pronounced respiratory climacteric and autocatalytic ethylene production, non-climacteric fruits, including strawberry, grape, and citrus, generally ripen without a comparable ethylene burst. Their ripening is more strongly associated with ABA-centered signaling, together with auxin, sugar, and other hormonal inputs that regulate pigmentation, softening, and metabolic maturation. Nevertheless, ethylene contributes to selected developmental and metabolic processes in non-climacteric fruits, indicating that these ripening categories differ primarily in the relative contribution and organization of their hormonal regulatory networks [50]. In strawberry, ABA interacts with auxin and other developmental signals rather than functioning as an isolated ripening trigger, reinforcing the view that hormone effects are strongly stage- and tissue-dependent [51,52]. Rather than belonging to distinct categories, climacteric and non-climacteric fruits represent points along a shared regulatory continuum: climacteric species focus their regulatory control on ethylene autocatalysis, whereas non-climacteric fruits rely more heavily on ABA, auxin, sugars, and tissue-specific developmental signals. The key distinction therefore lies not in the exclusive presence of particular hormones, but in how strongly, when, and through which TF networks those signals are integrated during ripening.

3.3. Flowering Juvenility Dormancy and Bud Break

Perennial reproductive development is governed by coordinated transitions between floral competence, dormancy maintenance, and meristem reactivation. In apple, the FT1–TFL1 module is embedded within a competitive transcriptional network. *MdbHLH48* promotes *MdFT1* while repressing *MdTFL1*, and the resulting *FT1* and *TFL1* proteins compete for interaction with *MdWRKY6* to differentially regulate the *LFY-like* gene *AFL1*, thereby linking developmental status with floral induction [53]. Dormancy, by contrast, is maintained through a growth-repressive state involving ABA, DAM/SVP-like MADS factors, and chromatin control [54]. In peach, fulfillment of chilling requirements is accompanied by declining *PpeDAM6* expression and increased H3K27me3, while perturbation of *PpeDAM6* alters ABA, GA, cytokinin, and JA pathways, identifying DAM factors as integrators of seasonal cues and hormone homeostasis [54]. Release from dormancy can additionally involve activating chromatin states; in tree peony, chilling and GA_3 promote an ATX1–COMPASS-like complex and H3K4me3 accumulation at growth-associated loci, facilitating bud break [55]. Together, these studies support a model in which flowering and bud break are controlled by hormone–TF–chromatin networks that establish developmental thresholds rather than by single hormonal switches. This architecture offers potential targets for adjusting flowering time and chilling requirement, although excessive weakening of dormancy may compromise cold hardiness and increase frost susceptibility.

Beyond reproductive transitions, GA–cytokinin crosstalk also regulates shoot architecture in perennial fruit crops. In apple, *MdKNOX19* directly represses the GA-biosynthetic gene *MdGA20ox3* while activating the cytokinin-biosynthetic gene *MdIPT1*, promoting dwarfing and increased branching. MdKNOX19 additionally interacts with MdOFP to coor-

dinately regulate these targets, establishing a transcriptional mechanism linking hormone homeostasis with tree architectural development [56].

3.4. Root Architecture Adventitious Rooting and Vegetative Propagation

Root regeneration illustrates how hormonal information is translated into cell identity through interacting transcriptional modules. Auxin promotes founder-cell specification and regenerative competence through ARF-centered networks involving WOX, LBD, and PLT factors, whereas cytokinin can oppose early root specification through ARR-associated signaling [57]. A particularly clear horticultural example comes from apple rootstock, where the auxin–cytokinin balance is resolved through a MdTCP17–MdWOX11 transcriptional checkpoint [58]. Cytokinin induces *MdTCP17*, whose protein product interacts with *MdWOX11* and suppresses WOX11-dependent activation of *MdLBD29* [58]. Consistent with this mechanism, *MdWOX11* overexpression promotes adventitious-root primordia, whereas *MdTCP17* overexpression inhibits root formation [58]. These findings show that regenerative capacity depends not simply on hormone concentration, but on the state of downstream transcriptional complexes that determine whether cells acquire root identity. This distinction is directly relevant to clonal propagation, because manipulating the TCP17–WOX11 module may provide greater precision than increasing exogenous auxin alone. Hormonal crosstalk also regulates stress-induced root plasticity. In cucumber, the JA-signaling repressor CsJAZ8 physically interacts with CsMYB6 and inhibits its transcriptional activity. *CsMYB6* directly activates the ethylene-biosynthetic gene *CsACO2* and the GA-biosynthetic gene *CsGA20ox2*, promoting ethylene and GA accumulation and adventitious root formation under waterlogging stress. This CsJAZ8–CsMYB6 regulatory module provides a mechanistic link between JA signaling, hormone biosynthesis, and adaptive root development [59]. Strigolactone–SMXL signaling and long-distance hormonal communication during graft healing likely provide additional layers of control [60], but direct crop-level causal evidence remains comparatively limited. Thus, targeting cell-type-specific regenerative regulators may offer a more effective strategy for improving rooting and vegetative propagation while minimizing whole-plant hormonal perturbation.

3.5. Senescence and Postharvest Physiology

Postharvest senescence can be viewed as a progressive shift in the relative activities of ripening-associated hormone–TF networks toward tissue deterioration. This regulatory reprogramming may involve changes in transcription factor abundance, phosphorylation-dependent activity, protein stability, co-regulator recruitment, and chromatin accessibility, which collectively influence the balance between ripening, stress protection, and senescence-associated transcription. However, the relative contributions of these mechanisms remain species- and developmental-stage-dependent. In climacteric fruit, ethylene-driven EIN3/EIL–ERF signaling extends from ripening into advanced softening, cell-wall disassembly, pigment loss, and senescence [61]. Banana provides a strong mechanistic example. *MaEIN3* activates a MaNAP1–MaMADS1 regulatory module that recruits downstream NAC and WRKY factors controlling cell-wall-associated genes, directly linking ethylene perception with softening and postharvest deterioration [46]. Additional evidence comes from grapevine, where *NAC61* expression increases during postharvest berry dehydration and responds to environmental and pathogen-associated signals. Functional analyses demonstrate that *NAC61* directly regulates *MYB14*, *DHN1b*, and *WRKY52*, connecting late-ripening transcriptional responses with stilbenoid metabolism, osmotic adjustment, and biotic stress signaling [48]. Although these findings support NAC-associated regulatory reprogramming during postharvest development, their direct contribution to terminal fruit senescence requires further validation. As harvested tissues undergo dehydration,

oxidative stress, and wounding, ABA-, JA-, NAC-, and WRKY-associated responses are likely to become increasingly important, although direct causal evidence for these pathways in terminal fruit senescence remains less developed than for ethylene. Postharvest outcome therefore depends on the timing and relative activity of multiple hormonal inputs rather than on ethylene alone. From an applied perspective, interventions such as 1-MCP, temperature management, and stage-specific hormonal regulation should aim not simply to delay softening, but to preserve flavor, pigmentation, membrane integrity, and nutritional quality. The key challenge is thus to identify regulatory nodes that extend marketable life without uncoupling desirable ripening traits from normal developmental progression.

Table 1. Representative hormone–transcription factor modules controlling horticultural crop development.

Hormone–TF Module	*Representative Crop*	*Developmental Process*	*Mechanistic Evidence*	*Ref*
Auxin–ARF/GA–GID1–DELLA	*Strawberry*	*Fruit set and growth*	*FveRGA1 loss induces parthenocarpy; FveRGA1 physically interacts with FveARF8, integrating auxin and GA control of fruit initiation and size.*	[17]
Ethylene–EIN3–NAC/MADS	*Banana*	*Ripening and softening*	*MaEIN3 activates MaNAP1 and MaMADS1, initiating downstream NAC/WRKY and cell-wall regulatory cascades.*	[46]
Ethylene–EIL–MYB	*Apple*	*Fruit coloration*	*MdEIL1 coordinates antagonistic MdMYB1–MdMYB17 activity to fine-tune anthocyanin accumulation.*	[62]
Auxin/ethylene–MADS/NAC/AP2	*Tomato*	*Ripening transition*	*Hormonal reprogramming precedes breaker-stage ripening, while RIN, NOR and associated TFs form a distributed regulatory network.*	[63]
ABA–auxin	*Strawberry*	*Non-climacteric ripening*	*ABA feedback and auxin–ABA interactions coordinate ripening-associated developmental and metabolic transitions.*	[64]
FT1/TFL1–WRKY6–LFY	*Apple*	*Floral induction*	*FT1 and TFL1 compete for MdWRKY6 and differentially regulate the LFY-like gene AFL1, determining floral-transition output.*	[53]
DAM/SVP–hormone–chromatin	*Peach*	*Dormancy and bud break*	*Chilling-associated repression of PpeDAM6, accompanied by chromatin remodeling, links winter progression with hormone homeostasis.*	[54]
Cytokinin–TCP17–WOX11	*Apple rootstock*	*Adventitious rooting*	*Cytokinin-induced MdTCP17 interacts with MdWOX11 and suppresses WOX11-dependent MdLBD29 activation, restricting root-primordium formation.*	[58]

4. Hormone–Transcription Factor Networks in Abiotic Stress Adaptation

Abiotic-stress adaptation in horticultural crops emerges from interconnected hormone–TF networks that balance protection, growth restraint, and recovery. Core ABA–SnRK2–ABF, DREB/CBF, and HSF pathways integrate with auxin, GA, JA, BR, ethylene, SA, and light signaling to coordinate water use, ion homeostasis, redox balance, and development under single or combined stresses.

4.1. Drought and Osmotic Stress

Drought adaptation is coordinated through an ABA-centered but multihormonal transcriptional network that integrates water conservation, redox control, root plasticity, and reversible growth restraint (Figure 3). Declining water potential activates PYR/PYL–PP2C–SnRK2 signaling, leading to phosphorylation of AREB/ABF TFs, which bind ABREs in the promoters of drought-responsive genes, including late embryogenesis abundant (LEA) proteins and dehydrins, to promote cellular dehydration protection and osmotic adjustment (Figure 3A). In tomato, *SlAREB1* directly activates *AOX1a*, linking ABA signaling to mitochondrial alternative respiration and ROS homeostasis [65]. *DREB2* factors provide a partially overlapping route. *SlDREB2* activates dehydration-responsive genes in tomato,

whereas apple *MdDREB2A* directly regulates *MdARF6*, connecting drought signaling with auxin-dependent root development [66].

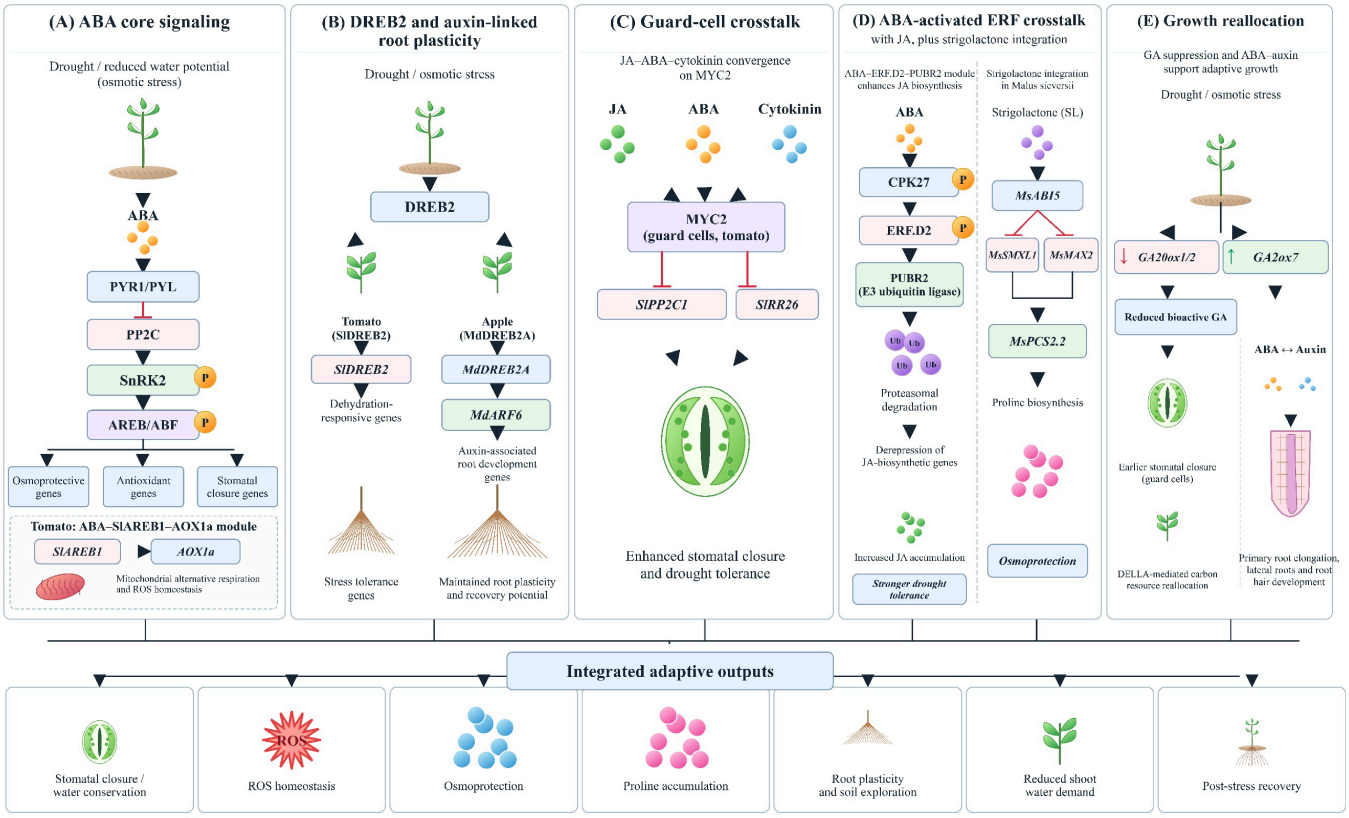


Figure 3. Multi-hormonal transcriptional control of drought and osmotic-stress adaptation in horticultural crops. Drought activates interconnected ABA-, JA-, auxin-, GA-, and strigolactone-responsive modules that coordinate stomatal regulation, osmoprotection, redox homeostasis, root plasticity, and reversible growth restraint. Representative mechanisms include ABA–AREB/ABF signaling, MdDREB2A–MdARF6-mediated root responses, JA–ABA–cytokinin crosstalk in guard cells, MsABI5–MsSMXL1–MsNAC022-dependent proline accumulation, and GA-mediated growth reallocation.

Importantly, ABA signaling is reinforced by other hormone–TF modules. In tomato guard cells, JA-responsive MYC2 directly represses both the ABA-signaling repressor *SlPP2C1* and the cytokinin response regulator *SlRR26*. The loss of *SlRR26* enhances stomatal closure and drought tolerance, revealing a mechanistic JA–ABA–cytokinin convergence point [67]. A second tomato mechanism shows that ABA-activated *CPK27* phosphorylates the negative regulator *ERF.D2*, promoting its *PUB22*-dependent degradation. The removal of *ERF.D2* derepresses JA-biosynthetic genes and increases JA accumulation, thereby strengthening drought tolerance [68]. These studies place AP2/ERF proteins not simply downstream of ethylene, but at points of crosstalk among ABA, JA, and drought-responsive transcription.

Strigolactone signaling provides an additional integration layer. In *Malus sieversii*, strigolactones enhance drought resistance through an MsABI5–MsSMXL1–MsNAC022 cascade [69]. *MsABI5* activates proline biosynthesis, represses the SL-associated repressor *MsSMXL1*, and promotes *MsNAC022*-dependent activation of *MsP5CS2.2*, thereby increasing osmoprotective proline accumulation [69]. Thus, ABA, JA, cytokinin, ethylene-associated ERFs, auxin, GA/DELLA, and strigolactone pathways converge on a limited set of TFs and repressors that determine whether plants prioritize stomatal closure, root growth, osmotic protection, or growth restraint.

This adaptive response also involves potentially reversible growth restraint, whereby drought-associated changes in GA metabolism restrict shoot expansion and water demand while maintaining root functions that support water acquisition and subsequent recovery. In tomato, water deficit represses the GA-biosynthetic genes *GA20ox1/2* while inducing the GA-deactivating gene *GA2ox7*, thereby lowering bioactive GA [70]. This operates at two scales: GA deactivation in guard cells promotes earlier stomatal closure, whereas reduced GA biosynthesis in leaves restricts canopy expansion and transpiring surface area. Accordingly, *ga2ox7* mutants close stomata later and wilt more rapidly under soil drying [70]. At the same time, ABA–auxin crosstalk helps preserve below-ground growth. Wild-type tomato maintains greater primary-root elongation, lateral-root abundance, and post-rewatering root production than ABA-deficient plants, while exogenous IAA partially restores these defects, indicating that ABA supports root-system plasticity partly through auxin responses [71]. A comparable developmental connection occurs in apple, where *MdDREB2A* directly activates *MdARF6* and downstream *MdGH3* genes, linking dehydration-responsive transcription with auxin-mediated root development [66]. Together, these examples show that drought tolerance is not generalized growth arrest, but a spatially coordinated redistribution of growth, reducing shoot water demand while maintaining root exploration and recovery capacity.

4.2. Salinity and Ion Homeostasis

Salinity adaptation involves a temporally coordinated transition from osmotic protection to ionic homeostasis, requiring integration of ABA, calcium, JA, ethylene, and growth-regulatory signals (Figure 4). Salinity adaptation shifts from rapid ABA-mediated osmotic signaling to sustained ion homeostasis, with Ca^{2+}–CBL4/SOS3–CIPK24/SOS2–SOS1, HKT, and NHX systems coordinating Na^+ exclusion, sequestration, and K^+ retention [72]. These transport processes are integrated with hormone-dependent transcriptional networks that preserve redox balance and root function. Recent horticultural studies demonstrate that NAC and WRKY TFs integrate ionic regulation with hormone and redox signaling. For instance, in tomato, *SlNAP1* overexpression decreases Na^+ accumulation, increases K^+ retention, and enhances antioxidant enzyme activity while upregulating *SlSOS1*, *SlHKT1;2*, and *SlNHX1*. The resulting reduction in ROS accumulation and membrane damage indicates that ion homeostasis and oxidative protection are coordinated components of the same transcriptional response [73]. More direct mechanistic evidence comes from pumpkin rootstocks grafted onto cucumber. *CmoNAC1* binds the promoters of *CmoNCED6* and *CmoRBOHD1*, promoting ABA biosynthesis and H_2O_2 signaling, while also directly regulating *CmoAKT1;2* and *CmoHKT1;1* to improve K^+/Na^+ homeostasis. Root-specific perturbation of *CmoNAC1* alters ABA and H_2O_2 accumulation, ion balance, and salt tolerance in the grafted plant, establishing a causal connection between root-localized transcriptional regulation and systemic adaptation [74]. JA provides an additional layer of transcriptional control. Under saline–alkaline stress, tomato *SlWRKY80* interacts with the JA repressor *SlJAZ1* and directly activates *SlSPDS2* and *SlNHX4*, coupling spermidine biosynthesis with Na^+/K^+ homeostasis. Exogenous methyl jasmonate reinforces this module and improves tolerance, illustrating how JA signaling can coordinate osmoprotective metabolism with ion compartmentalization [75]. ABA-associated transcription also includes negative regulators. The nuclear repressor *SlAITR3* is induced by both ABA and salt, yet its silencing or knockout reduces the Na^+/K^+ ratio and ROS accumulation while improving tomato salt tolerance [76]. Together, these findings indicate that salt-responsive transcription is governed by competing positive and negative regulatory nodes, rather than uniform activation of stress-associated genes. Beyond ionic and redox regulation, stress-responsive NAC, WRKY, and MYB TFs can also influence cell-cycle-associated gene expression and adaptive growth reprogramming. Such regulation may contribute to temporary growth restriction

under adverse conditions, although direct transcriptional connections to cell-cycle regulators remain insufficiently characterized in horticultural crops [77]. A further adaptation layer involves hormone-dependent root plasticity, through which plants adjust water acquisition and nutrient uptake under saline conditions. Auxin–ARF, cytokinin–ARR, ethylene–EIN3/ERF, and GA–DELLA networks influence root elongation, lateral-root initiation, and developmental allocation, but their effects on ion homeostasis are strongly genotype-dependent. Notably, the ethylene-insensitive *Never-ripe* (*nr*) mutant exhibited increased root growth but impaired K^+ retention and a higher shoot Na^+/K^+ ratio [78]. These findings indicate that enhanced root proliferation does not necessarily translate into improved salinity tolerance, as its adaptive significance depends on hormonal interactions, ion homeostasis, and the genetic background of wild and cultivated tomato accessions [78]. Collectively, these findings highlight that salinity resilience depends on the coordinated regulation of osmotic adjustment, ion homeostasis, redox balance, and developmental plasticity, with outcomes shaped by hormonal crosstalk and genetic background. The central challenge is therefore to identify context-specific hormone–TF modules that optimize ion balance and root adaptation while sustaining photosynthesis, reproductive performance, and overall crop productivity.

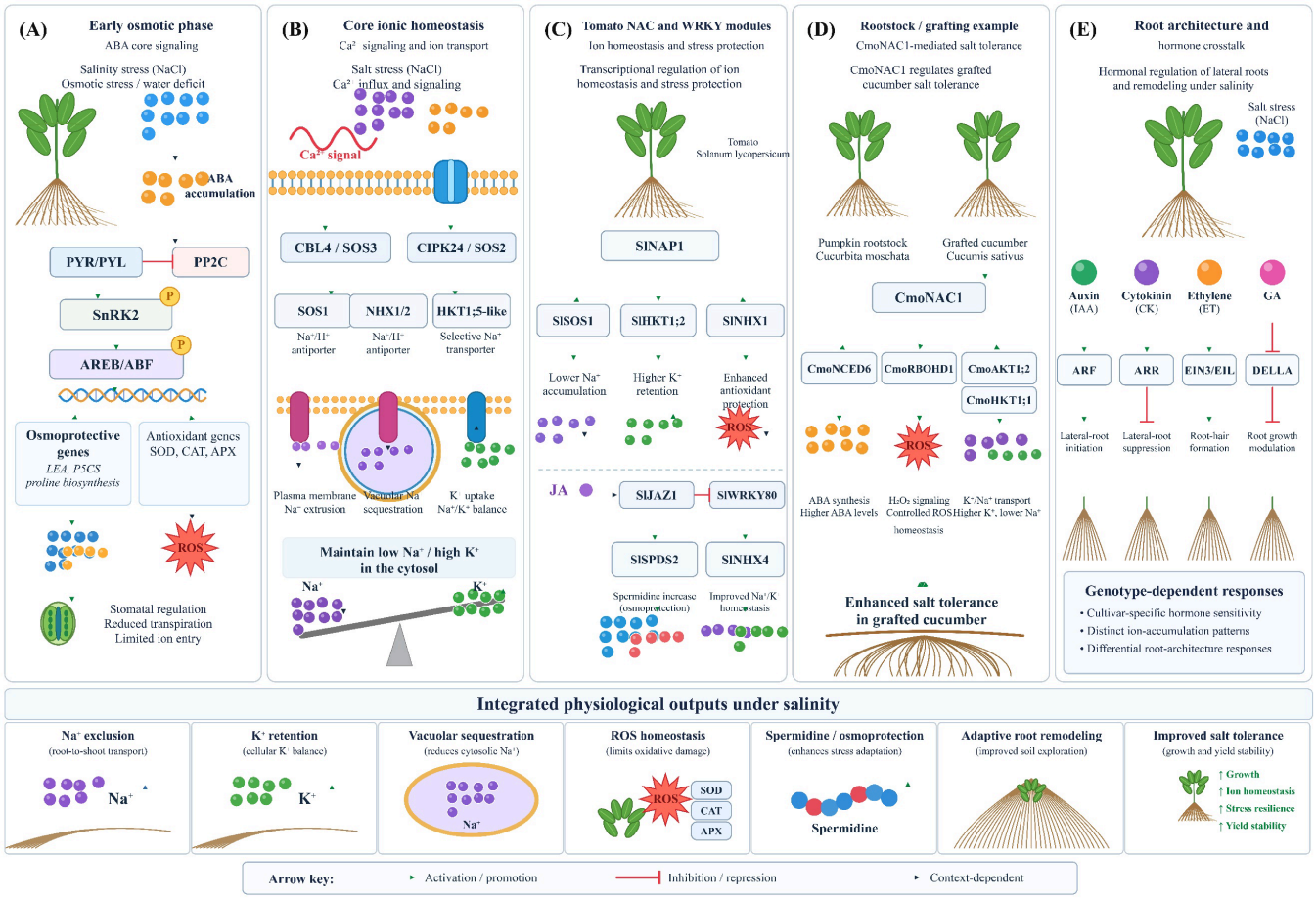


Figure 4. Hormone–transcription factor regulation of salinity adaptation, ion homeostasis, and root plasticity in horticultural crops. (**A**) ABA–PYR/PYL–PP2C–SnRK2–AREB/ABF signaling coordinates early osmotic protection. (**B**) Ca^{2+}-activated CBL4/SOS3–CIPK24/SOS2 signaling regulates Na^+ exclusion, vacuolar sequestration, and K^+ retention. (**C**) Tomato NAC/WRKY modules integrate ion homeostasis, redox control, and spermidine metabolism. (**D**) Rootstock-derived *CmoNAC1* coordinates ABA, H_2O_2, and K^+/Na^+ transport in grafted cucumber. (**E**) Auxin, cytokinin, ethylene, and GA reshape root architecture, collectively supporting ion balance, ROS homeostasis, and adaptive root remodeling.

4.3. Temperature Stress

4.3.1. Cold Stress

Cold adaptation activates the protective ICE-CBF-COR pathway, regulated by upstream transcription factors rather than cold perception alone. In apple, *MdbHLH4* directly represses *MdCBF1/3*, interacts with *MdICE1L* to impair its promoter-binding activity, and activates *MdCAX3L-2*, which promotes cold-induced *MdICE1L* degradation. This regulatory module therefore constrains CBF activation through both transcriptional repression and altered TF stability [79]. ABA and JA provide an additional layer of transcriptional and chromatin-level control. The ABA-responsive bZIP factor *MdABI5* directly activates *MdCBF3* while repressing the JA-signaling repressors *MdJAZ1/3*. *MdABI5* also recruits the histone deacetylase *MdHDA6* to *MdJAZ1/3* promoters, reinforcing their repression, whereas *MdTCP15* antagonizes *MdABI5*-dependent transcription. This MdABI5–MdTCP15–MdHDA6 module demonstrates how ABA and JA signaling converge through promoter regulation, protein interactions, and chromatin modification to enhance cold tolerance [80]. A complementary mechanism occurs in tomato, where the ethylene-response factor *SlERF.B8* directly activates the JA-biosynthetic genes *SlLOXD*, *SlAOC*, and *SlOPR3*. JA-responsive *SlMYC2*, in turn, activates *SlERF.B8*, establishing a positive-feedback loop that reinforces JA accumulation and cold tolerance [81].

BRs and ethylene further reshape cold-responsive transcription. In tomato, *BZR1* directly activates *SlCBF1*, *SlCBF3*, and *SlRBOH1*, linking BR signaling with RBOH1-dependent H_2O_2 production and redox regulation. This interaction promotes CBF expression and cold acclimation, illustrating how controlled ROS production can function as a signaling input rather than merely an indicator of oxidative damage [82].

Ethylene-dependent regulation also operates in harvested tissues. Tomato fruit with impaired ethylene perception exhibits reduced cold-induced *SlCBF1* expression, altered ABA and JA accumulation, increased oxidative injury, and greater chilling susceptibility [83]. Subsequent genetic analysis further demonstrated that *SlCBF1* and ethylene signaling interact to maintain fruit quality during prolonged cold storage [83]. Thus, the consequences of ethylene manipulation during postharvest storage extend beyond ripening control to chilling tolerance.

For perennial horticultural crops, cold acclimation must additionally be interpreted within the context of bud dormancy and seasonal temperature history. Dormant grapevine buds integrate temperature cues with carbohydrate metabolism, protective pathways, and hormone-associated transcription. In endodormant grapevine buds, temperature cycling (7 ± 5 °C), unlike constant cold, enhances freezing tolerance and alters key metabolic and signaling transcripts [84]. This distinguishes physiological cold acclimation from mere cold-responsive gene induction under constant temperatures. Together, these studies frame cold tolerance as a context-dependent balance of CBF activation, hormone feedback, redox homeostasis, and developmental competence. Identifying regulatory nodes that enhance cold protection without compromising fruit quality, bud-break timing, or recovery will be key to horticultural resilience under fluctuating temperatures.

4.3.2. Heat Stress

Heat adaptation is governed by a multi-hormonal transcriptional network that integrates proteostasis, redox homeostasis, water balance, and reproductive development. Recent tomato studies extend this canonical framework by demonstrating that HSF activity is integrated with co-regulator recruitment and autophagy. *SlHsfA1a* directly activates *SlBAG5b* and interacts with its protein product to enhance transcription of *SlATG10*, promoting autophagosome formation and clearance of ubiquitinated proteins. A complementary SlHsfA1a–BAG2–BAG5b complex reinforces *HSP70* and *HSP90* expression, establishing coordinated protein protection and degradation as interconnected components of thermo-

tolerance [85]. ABA signaling connects cellular protection with stomatal regulation and additional hormonal inputs. In this regard, *SlSnRK2.3* physically interacts with *SlSUI1* and contributes to ABA-dependent stomatal movement and ROS regulation during heat stress [86]. Nevertheless, stomatal closure may conserve water at the expense of evaporative cooling, making the adaptive value of ABA-dependent responses contingent on water availability and atmospheric demand. SL signaling provides another layer of regulation: disruption of SL biosynthesis or signaling compromises tomato heat tolerance, whereas application of the SL analogue GR24 enhances ABA accumulation, HSP70 production, and antioxidant responses. These protective effects are substantially reduced in ABA-deficient plants, supporting an SL–ABA interaction, although the precise downstream TF connections remain incompletely resolved [87]. JA and BRs further connect heat perception with transcriptional and chromatin-level regulation. In tomato, *SlMYC2* directly activates HSP genes and interacts with the histone demethylase *SlJMJC3*, which removes repressive H3K9me1/3 and H3K27me3 marks at HSP loci. *SlMYC2* also activates *SlJMJC3*, establishing a positive-feedback module that reinforces heat-responsive transcription and thermotolerance [88]. Ethylene-associated transcriptional regulation additionally links heat adaptation with fruit development. The tomato NAC factor *SlJA2L* directly activates *SlHsfA3* and *SlCAT3* during heat stress, while promoting ethylene biosynthesis through *SlACO1* and carotenoid accumulation through *SlCRTISO* during ripening. Genetic perturbation establishes *SlJA2L* as a convergence node between thermotolerance and fruit maturation. However, hormonal effects on reproductive tissues are not uniformly protective. Under prolonged mild heat, reduced SA accumulation improved tomato pollen viability, with transcriptomic and genetic evidence implicating enhanced JA signaling. This demonstrates that SA-mediated protection depends on tissue identity, exposure duration, and the competing demands of defense and reproduction [89]. Collectively, these studies identify HSF–HSP, JA–MYC2–JMJC3, BR–BZR1, ABA–SnRK2, and ethylene-associated NAC networks as interacting components of heat adaptation. The principal horticultural challenge is therefore not simply enhancing acute heat survival, but identifying regulatory nodes that sustain pollen viability, fruit set, yield, and quality during prolonged or recurrent warming.

4.4. Light and Ultraviolet Stress

Light and ultraviolet-B (UV-B) signaling constitute environmental inputs into hormone–transcription factor networks that balance growth, photoprotection, and metabolic adaptation. Red: far-red light ratios are perceived primarily through phytochromes, which regulate *PHYTOCHROME-INTERACTING FACTOR* (PIF) stability and activity. In model plants, PIFs interact with DELLA, BZR1/BES1, and auxin-associated ARF networks, integrating light availability with GA, BR, and auxin-dependent growth. However, these interactions do not necessarily generate conserved outputs across horticultural species. A tomato mutant lacking all eight identified PIF genes exhibited strongly impaired shade-avoidance responses under low red: far-red light, whereas warm-temperature-induced elongation remained largely PIF-independent. *SlPIF1a*, *SlPIF4*, and *SlPIF8a* also displayed partially redundant functions in stem elongation and chlorophyll regulation [90]. These findings demonstrate that conserved signaling components can be embedded within species-specific regulatory architectures.

Light-dependent hormone regulation is also spatially constrained. In *Arabidopsis*, direct root illumination activates phyA/phyB signaling, suppresses PIF1/PIF4-dependent auxin biosynthesis, and restricts root elongation; related light-responsive components are conserved in tomato [91]. This has experimental implications because illuminated roots in conventional plate-based assays may not reproduce the hormonal environment of roots developing in darkness. In tomato, combined red, white, and blue illumination promoted photosynthesis and pigment accumulation through a SlPHYB1/SlCRY1–SlHY5

module. *SlHY5* directly activated *SlLHCA*, *SlLHCB*, and *SlCYCB*, linking light perception with photosynthetic capacity and carotenoid metabolism [92].

UV-B introduces an additional transcriptional control layer through the UVR8–COP1–HY5 pathway. For instance, *UVR8* also modulates PIF activity, providing a mechanism through which UV-B can redirect transcription away from elongation toward acclimation [93]. In horticultural crops, *HY5*-dependent transcription connects photoprotection with fruit pigmentation. Apple *MdHY5* cooperates with *MdMYB22* to promote flavonol-biosynthetic gene expression under UV-B [94]. In grapevine, *VvHY5* directly activates *VvMYB24*, whose protein product interacts with *VvMYBA1* to enhance VvDFR and VvUFGT expression and anthocyanin accumulation [95]. Although this grapevine study establishes a light-responsive TF cascade, it does not by itself demonstrate that *UVR8* or a particular hormone initiates the interaction.

ABA, SA, JA, and BR can further modify UV-associated responses, particularly stomatal regulation, antioxidant protection, specialized metabolism, and defense. However, their direct regulatory connections with UVR8–HY5 remain less comprehensively established in horticultural crops than the core photoreceptor pathway. Moderate supplemental UV-B can enhance protective metabolites and reduce disease or pest incidence, whereas excessive exposure can inhibit photosynthesis and growth. Collectively, these findings identify light–hormone crosstalk as a dose-, tissue-, and species-dependent regulatory system. The translational challenge is to optimize light quality and UV-B exposure to enhance crop resilience and fruit quality without incurring penalties in biomass, reproductive performance, or yield.

Beyond UV-B signaling, controlled UV-C irradiation has emerged as a promising postharvest treatment for modulating fruit quality and senescence. In tomato, UV-C-induced *SlERF7* directly activates *SlPAL5* transcription, promoting phenolic biosynthesis and antioxidant-associated metabolism [96]. More recent multi-omics evidence indicates that postharvest UV-C treatment delays tomato ripening through coordinated changes in hormone signaling, cell-wall metabolism, volatile composition, chromatin accessibility, and epigenetic regulation [97]. These findings suggest that UV-C-responsive transcriptional networks contribute to postharvest quality maintenance, although their protective effects depend on treatment intensity, fruit developmental stage, and cultivar-specific responses.

4.5. Combined and Sequential Stress Responses

Horticultural crops rarely experience environmental stresses in isolation; instead, they encounter overlapping or sequential episodes of drought, heat, salinity, nutrient limitation, and pathogen pressure that place competing demands on growth and survival. For example, combined salinity and heat elicit transcriptional and metabolic responses distinct from either treatment alone, with oxidative phosphorylation emerging as an important integrated pathway under the conditions examined [98]. Stress history adds another layer of regulation: an initial challenge can alter hormone abundance, redox balance, metabolite pools, and TF availability, thereby shaping responses to subsequent exposure. Thus, the nature, sequence, intensity, and duration of stress, together with the affected tissue type and developmental stage, determine whether protective responses can be sustained without compromising growth and reproduction. A central question is how hormone–TF networks prioritize conflicting signals and subsequently reset during recovery. Conceptually, this process can be divided into three overlapping phases: stress perception and activation of protective signaling, prioritization of competing hormonal and transcriptional programs, and regulatory resetting during recovery. The transition between these phases may depend on hormone turnover, TF stability, feedback regulation, and chromatin state, allowing protective responses to diminish as favorable conditions return while supporting restoration of growth and reproductive functions. Addressing this question will require experiments that

reproduce fluctuating production environments and assess not only molecular acclimation or seedling survival, but also flowering, fruit set, marketable yield, and postharvest quality.

5. Hormone–TF Networks in Biotic Stress and Immunity

Plant immunity is governed by interconnected hormonal and transcriptional networks that translate the recognition of diverse pathogens, including bacteria, fungi, oomycetes, and plant-parasitic nematodes, into locally and systemically coordinated defense responses. Among these networks, the interplay of SA, JA, and ethylene is the best-characterized framework for orchestrating immune outputs.

5.1. Salicylic Acid, Jasmonate, and Ethylene Crosstalk

SA, JA, and ethylene form interconnected, context-dependent immune signaling networks. Recent work in *Arabidopsis* has further challenged the strict antagonism of SA–JA signaling, showing that rapid local and systemic JA signaling contributes to systemic acquired resistance (SAR) and thus to immune processes traditionally associated with SA [99]. Hormone crosstalk is therefore best understood as the selective assembly and regulation of transcriptional complexes, rather than competition between entire signaling pathways. SA-dependent defense is coordinated principally through *NONEXPRESSOR OF PATHOGENESIS-RELATED GENES1* (*NPR1*), which functions as an SA-responsive transcriptional co-regulator.

Crop studies reveal additional regulatory layers. In tomato, SA treatment and *Clavibacter michiganensis* infection induce overlapping defense programs enriched in WRKY-binding W-box elements, accompanied by repression of cell-cycle-associated genes [100]. More direct evidence for immune feedback comes from apple, where *MdWRKY1* promotes SA accumulation through *MdEPS1* while regulating a MdWRKY40–MdNPR3g module that limits sustained *PR1* expression. *MdNPR3g* interferes with a specific NPR1–TGA2c complex, suggesting that WRKY-mediated feedback can constrain excessive defense activation during resistance to powdery mildew [101]. Together, these findings place NPR–TGA complexes within a wider transcriptional network that coordinates defense induction with immune restraint.

JA signaling operates through a complementary mechanism involving regulated repressor degradation. Perception of bioactive JA-Ile by the COI1–JAZ co-receptor promotes JAZ turnover, releasing MYC2 and other associated TFs [102]. In tomato, this canonical pathway is reinforced by a distinct ubiquitination mechanism: MYC2 directly activates PUB22, whose E3 ligase product promotes JAZ4 degradation, establishing a positive-feedback circuit that contributes to defense against herbivory and Botrytis cinerea [103]. The downstream consequences of JA signaling are also developmentally specified. During *B. cinerea* infection of tomato fruit, *MYC2* physically interacts with the ripening-associated TFs *NOR* and *RIN*, which enhance *MYC2*-mediated activation of *ERF.F4*. This activation subsequently induces expression of the defense-related gene *PR-STH2*, contributing to pathogen resistance [104]. This MYC2–NOR/RIN–ERF.F4 cascade demonstrates that ripening regulators can be recruited into JA-mediated immunity rather than acting exclusively in fruit development.

ET introduces further specificity through EIN3/EIL-dependent transcription and downstream ERF activity. Importantly, ET–JA interactions can either reinforce or attenuate defense, depending on the regulatory branch engaged. In tomato fruit, EIL factors directly activate *CYP94C1*, which inactivates JA-Ile and consequently weakens JA-mediated resistance during ripening; *CYP94C1* knockout improves resistance to necrotrophic pathogens without compromising the fruit-quality traits assessed [105]. Although the core components of ethylene and jasmonate signaling are broadly conserved, the EIL–CYP94C1 and SlMPK8–SlERF.C1 regulatory modules have been functionally characterized primarily in tomato. Whether these specific transcriptional interactions are conserved across other horticultural crops or repre-

sent lineage-dependent regulatory adaptations remains to be determined. By contrast, *B. cinerea*-induced ET production activates a protective SlMPK8–SlERF.C1 module. SlMPK8 phosphorylates SlERF.C1 at Ser174, increasing its ability to directly activate PR1 and PR1a. Genetic enhancement of this module improves gray-mold resistance without detectable disruption of normal ripening under the experimental conditions [106].

These contrasting mechanisms illustrate why the effect of ET cannot be inferred from hormone abundance alone. Its contribution to immunity depends on which TFs and downstream targets are engaged. Shared TF hubs extend this integration beyond the three principal defense hormones. In sweet orange, *CsWRKY53* directly activates *CsWAKL01*, encoding a wall-associated receptor-like kinase that modifies SA-, JA-, and ABA-associated signaling and enhances resistance to citrus bacterial canker [107]. Collectively, these studies identify co-regulator assembly, selective repressor turnover, TF phosphorylation, and promoter-specific interactions as mechanisms through which SA, JA, and ET acquire distinct immune outputs. They also provide a mechanistic basis for the context-dependent antagonism and synergy examined in Section 5.2. The relevant distinction is not simply whether a hormone promotes or suppresses immunity, but which transcriptional branch it activates, in which tissue, and at what developmental stage. The principal SA-, JA-, and ET-responsive transcriptional branches and their crop-specific points of convergence are summarized in Figure 5.

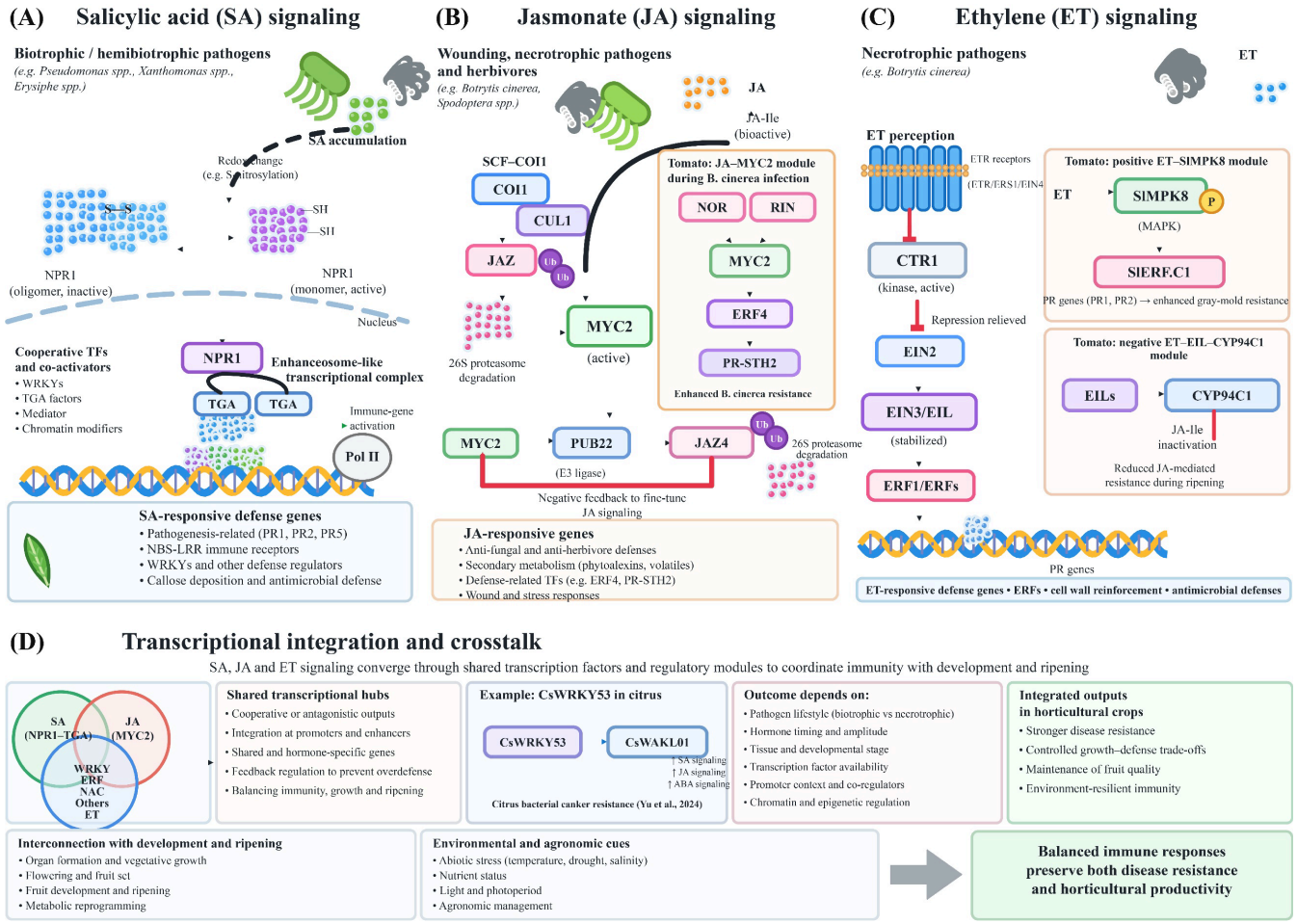


Figure 5. Transcriptional integration of SA, JA, and ethylene signaling during horticultural crop immunity. SA–NPR1–TGA, JA–COI1–JAZ–MYC2, and ET–EIN2–EIN3/EIL–ERF modules generate interconnected, context-dependent defense outputs. Crop-specific examples include MYC2–NOR/RIN–ERF.F4–PR-STH2 and SlMPK8–SlERF.C1 in tomato and CsWRKY53–CsWAKL01 in citrus, illustrating how hormone crosstalk coordinates immunity with development and ripening [107].

5.2. Synergistic and Antagonistic Hormone Crosstalk Networks

The frequently invoked model in which SA controls biotrophic resistance whereas JA/ET controls necrotrophic resistance remains useful as a first approximation, but it is insufficient to describe immune behavior in crops. SA and JA can antagonize one another, yet they can also act sequentially, additively, or cooperatively depending on pathogen lifestyle, infection stage, hormone concentration, and cellular context. At the molecular level, SA-dependent *NPR1* can suppress subsets of JA-responsive genes by interfering with MYC transcriptional activation [108]; whereas WRKY proteins and other transcriptional regulators can further adjust the balance between the pathways [109]. The resulting network is therefore conditional rather than a binary SA–JA switch.

Recent evidence from mulberry further illustrates the context-dependent transcriptional responses associated with JA and SA signaling during herbivore challenge. Ahmad et al. (2026) [110] demonstrated that exogenous JA and SA differentially modulate antioxidant enzyme activities and defense-associated gene expression in *Glyphodes pyloalis*-challenged mulberry. JA treatment was associated with enhanced phenylpropanoid and flavonoid biosynthesis-related transcription, MAPK signaling, and antioxidant responses, whereas SA preferentially influenced photosynthesis-related transcription, chloroplast organization, and redox metabolism. Several hormone-responsive transcription factor families, including HD-ZIP, LBD, GATA, TALE, ARF, and SBP, also displayed altered expression. These findings highlight overlapping yet distinct JA- and SA-associated regulatory programs, although direct interactions among the identified transcription factors require further functional validation.

JA signaling itself contains functionally distinct transcriptional outputs. A MYC-centered branch is strongly associated with wounding, herbivory, specialized metabolism, and several antimicrobial responses, whereas an ERF-centered branch becomes particularly important when JA cooperates with ET during defense against necrotrophic pathogens [111]. These branches nevertheless communicate extensively. Tomato *MYC2*, for example, directly activates pathogen-responsive ERFs, and the MYC2–ERF.C3 and MYC2–ERF.F4 modules contribute *to B. cinerea* resistance [103]. Therefore, MYC and ERF activities are better regarded as alternative but interacting transcriptional states than as mutually exclusive pathways.

The ecological relevance of this flexibility becomes apparent during multiple infections. In tomato simultaneously challenged with tomato yellow leaf curl virus and the root-knot nematode *Meloidogyne incognita*, viral infection enhanced SA- and ET-associated defenses that suppressed nematode parasitism; this protective interaction disappeared in plants deficient in SA or ET accumulation [112]. Such interactions show that the immune response elicited by one organism can alter susceptibility to another, making hormone prioritization dependent on infection sequence and community context rather than pathogen lifestyle alone.

5.3. Pathogen Manipulation of Host Hormone Networks

The centrality of hormone signaling to immunity makes it an attractive target for pathogen effectors. The bacterial phytotoxin coronatine, for example, functions as a structural and functional mimic of JA-Ile and activates JA-associated transcription to suppress SA-dependent defenses [113]. More generally, viral, bacterial, fungal, oomycete, and nematode effectors increasingly appear to target regulatory hubs shared between hormone perception and immunity.

A recent horticultural example is provided by tomato spotted wilt orthotospovirus. The pepper NLR protein Tsw surveils interference by the viral NSs effector with host hormone-receptor signaling, illustrating how immune receptors themselves can evolve to detect pathogen manipulation of hormone-associated proteins [114]. In citrus, the

phloem-colonizing Huanglongbing-associated bacterium '*Candidatus Liberibacter asiaticus*' deploys the effector SECP8 to target the TF *CsTCP15*. *CsTCP15* normally activates SA-responsive *CsPR5* and *CsWRKY22*, whereas SECP8 inhibits *CsTCP15* dimerization and promotes its destabilization, thereby suppressing SA-mediated immunity and facilitating bacterial colonization [115]. These findings illustrate how vascular pathogens can directly reprogram hormone–TF modules during chronic disease.

Auxin and cytokinin pathways are particularly vulnerable to manipulation because they control cell proliferation and differentiation. *Agrobacterium tumefaciens* provides the canonical example: integration of T-DNA carrying iaaM/iaaH and *ipt* genes drives excessive auxin and cytokinin production, transforming host developmental programs and generating crown-gall tumors that function as strong metabolic sinks [116]. Root-knot nematodes achieve comparable developmental reprogramming without genomic transformation. In tomato, *M. incognita* induces giant feeding cells from vascular tissues, and the miR167–ARF8 regulatory module contributes to this auxin-dependent process; perturbation of ARF8 activity modifies feeding-site development and nematode success [117].

The bacterial effector AvrRpt2 requires particular mechanistic precision. AvrRpt2 is a cysteine protease whose established immune target is *RPM1-INTERACTING PROTEIN4* (*RIN4*); cleavage of *RIN4* alters immune signaling and is monitored by the RPS2 resistance protein. AvrRpt2 also enhances auxin responses by promoting proteasome-dependent turnover of Aux/IAA transcriptional repressors such as AXR2/IAA7 and AXR3/IAA17 [118]. However, available evidence does not support describing AvrRpt2 as a protease that directly cleaves ARFs. Direct cleavage of AXR2 could not be demonstrated experimentally, suggesting that its effects on auxin signaling are indirect or mediated through additional host substrates. This distinction is important because effector-driven hormone reprogramming commonly arises through modification of signaling regulators rather than direct destruction of the final transcription factor.

5.4. Systemic Acquired Resistance and Induced Systemic Resistance

Local infection and beneficial root-associated microorganisms establish systemic immunity through overlapping but distinct hormone–transcription factor networks. Pathogen-induced SAR involves SA, NPR1–TGA-dependent defense transcription, and mobile signals such as N-hydroxypipecolic acid, which enhance immune readiness in distal tissues. By contrast, microbe-ISR frequently relies on JA and ethylene signaling. In tomato, root application of *Bacillus cereus* EC9 enhances resistance to *Fusarium oxysporum* f. sp. *lycopersici* by priming JA/ET-associated defenses [119]. The *Arabidopsis*–Pseudomonas model further identifies *MYB72* as a link between root exudation, iron acquisition, microbiome assembly, and ISR through BGLU42-associated coumarin release, although conservation of this mechanism in horticultural crops remains to be established [120]. Together, SAR and ISR illustrate how hormone–TF networks translate local pathogen or microbial signals into systemic, context-dependent protection, offering opportunities to enhance crop resistance without sustained developmental costs.

5.5. Growth–Immunity Trade-Offs in Horticultural Crops

Immune activation competes with growth for metabolic resources and can suppress photosynthesis, cell division, organ expansion, and reproduction, making the growth–immunity balance particularly consequential for horticultural productivity. In tomato, SA-associated resistance to *Clavibacter michiganensis* coincides with repression of cell-cycle genes [100], whereas repeated wounding triggers JA-dependent reductions in shoot growth, leaf expansion, and flowering [102]. Inhibition of *TARGET OF RAPAMYCIN* (*TOR*), a central regulator of cellular growth and metabolism, likewise enhances resistance to multiple

pathogens largely through SA-dependent immunity, highlighting the connection between nutrient-sensitive growth signaling and defense activation [121]. However, these costs are not inevitable. Immune priming can maintain heightened defense readiness without sustained activation of downstream responses. In tomato, ISR-associated cytokinin signaling can support both growth and immunity under the conditions examined [122]. Similarly, the ET–SlMPK8–SlERF.C1 module enhances resistance to *B. cinerea* without detectably impairing fruit ripening [106], illustrating how a specific transcriptional branch can be strengthened without broadly disrupting development. The translational objective is therefore not maximal or constitutive defense activation, but precise control of its timing, amplitude, and tissue specificity. Pathogen-inducible TFs, post-translational regulatory switches, microbiome-mediated priming, and *cis*-regulatory editing offer potential routes to this goal. Such approaches are especially relevant to perennial fruit crops, where persistent defense costs may accumulate across seasons through reduced reserve formation, flowering, and yield.

6. Transcriptional Convergence Hubs and Hormone Crosstalk

Building upon the molecular signaling architectures and their developmental, abiotic stress, and immune functions discussed in Sections 2–5, this section synthesizes the shared transcriptional convergence hubs through which horticultural crops coordinate hormonal and environmental signals. Rather than operating as independent pathways, DELLA, BZR1/BES1, JAZ–MYC, EIN3/EIL–ERF, WRKY/NAC, and NPR–TGA networks integrate multiple signaling inputs to regulate growth, stress adaptation, and immunity in a context-dependent manner. Consequently, the same regulatory node can promote growth in one developmental context while favoring stress protection or immunity in another. This connectivity explains both the versatility and pleiotropic risk of hormone manipulation in horticultural crops. The major convergence hubs and the contextual determinants that shape their outputs are summarized in Figure 6. Importantly, direct regulatory interactions supported by molecular binding assays and genetic perturbation should be distinguished from putative associations inferred through co-expression, motif enrichment, or integrated omics analyses. While the latter provide valuable insights into candidate regulatory networks, functional validation remains essential for establishing causality and physiological relevance.

6.1. DELLA and BZR1/BES1 as Growth-Integration Hubs

DELLA proteins provide a direct molecular interface between GA-dependent growth and other hormonal programs. GA–GID1 signaling promotes DELLA degradation, whereas DELLA accumulation under growth-restricting conditions alters transcription by interacting with PIFs, BZR1/BES1, ARFs, and JAZ-associated regulators [123]. This enables DELLA proteins to redistribute transcriptional output between elongation, reproductive development, defense, and stress adaptation. Crop evidence illustrates this context dependence. In diploid strawberry, *FveRGA1* interacts with FveARF8 to regulate fruit initiation and size, with loss of *FveRGA1* inducing parthenocarpy [17]. In tomato, *PROCERA* contributes to ABA-dependent stomatal regulation, while drought-induced GA depletion promotes earlier stomatal closure and reduced canopy expansion [70]. Recent evidence from apple further reveals a direct connection between DELLA-mediated signaling and GA homeostasis. The DELLA protein *MdSLR1b* physically interacts with MdKNOX15, which directly represses the GA-biosynthetic gene *MdGA20ox2* while activating the GA-catabolic gene *MdGA2ox7*. *MdKNOX15* also enhances *MdSLR1b* stability, reinforcing GA signaling inhibition and suppressing stem elongation. This regulatory module illustrates how DELLA–TF interactions coordinate hormone metabolism and signaling to control tree architecture

and dwarfing [124]. Thus, DELLA activity links reversible growth restraint with stress protection but also influences fruit development and architecture.

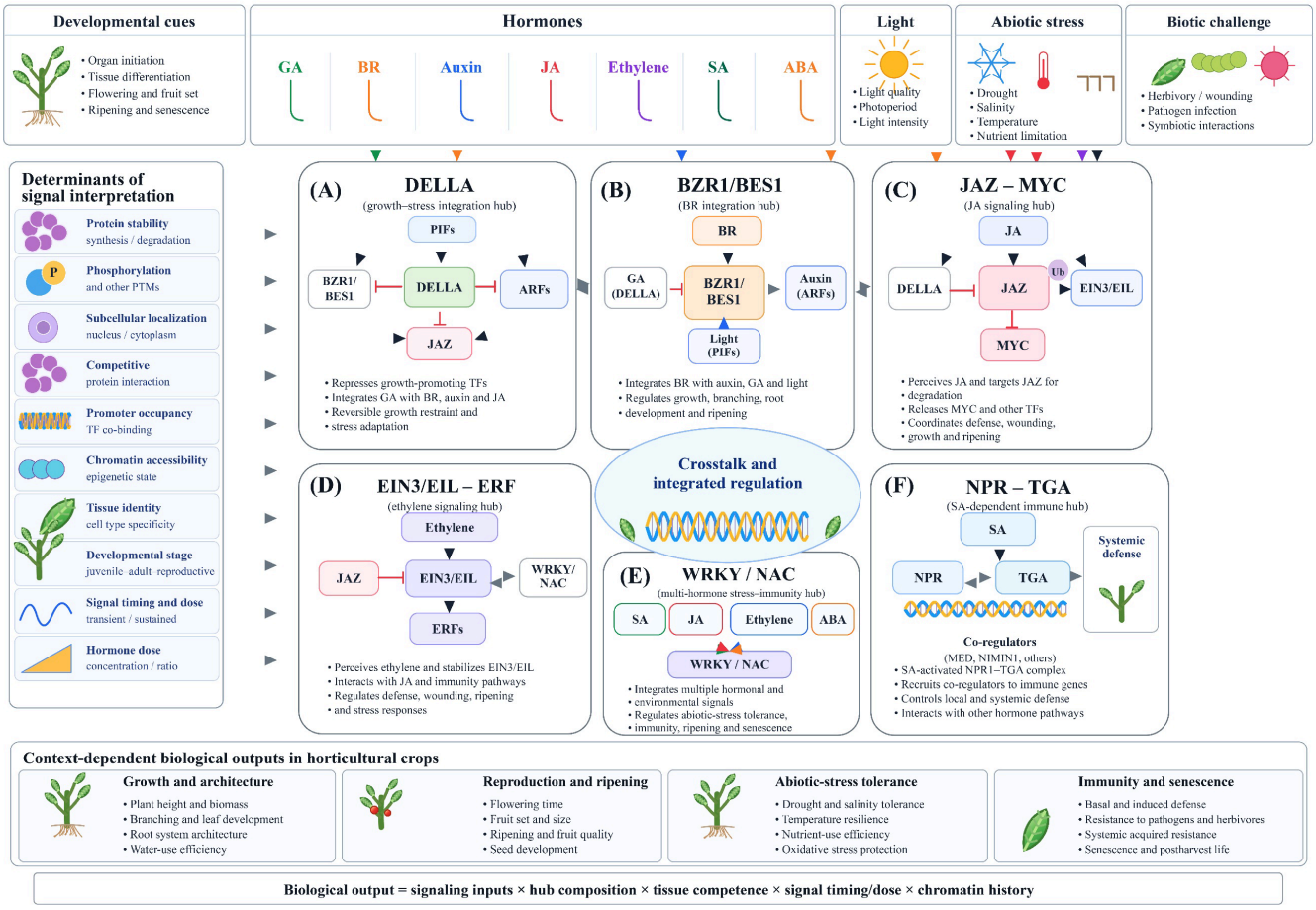


Figure 6. Transcriptional convergence hubs integrating hormonal, developmental, and environmental signals in horticultural crops. Hormonal and environmental inputs converge on a restricted set of highly connected regulatory hubs that redistribute transcriptional output according to cellular context. (**A**) DELLA proteins integrate GA signaling with PIF, BZR1/BES1, ARF, and JAZ–MYC networks, coupling growth restraint with development and stress responses. (**B**) BZR1/BES1 integrate BR signaling with auxin, GA, light, root development, and ripening. (**C**,**D**) JAZ–MYC and EIN3/EIL–ERF networks mediate context-dependent cooperation or antagonism between JA, ethylene, defense, and ripening. (**E**) WRKY and NAC factors integrate SA, JA, ABA, ethylene, ROS, senescence, and immunity. (**F**) NPR1–TGA complexes link SA perception with MED15A/Mediator-dependent immune transcription and competitive regulation by *NIMIN1*. Biological output is further conditioned by protein stability, phosphorylation, competitive interactions, subcellular localization, promoter occupancy, chromatin accessibility, tissue identity, developmental state, and signal timing. Together, these mechanisms explain how shared signaling hubs can control architecture, fruit development, stress tolerance, immunity, and postharvest physiology while creating substantial pleiotropic potential.

BZR1/BES1 provide a complementary growth-integration node. These BR-responsive TFs interact functionally with auxin-, GA-, and light-associated regulators, allowing cell expansion and developmental allocation to respond to broader signaling context. In tomato, *BZR1* promotes axillary-bud outgrowth through repression of *BRANCHED1* [125] and activates *PIN5* and *PIN10*, linking BR signaling with intracellular auxin homeostasis during lateral-root development [126]. Together, DELLA and BZR1/BES1 illustrate why constitutive manipulation of major growth hubs is likely to generate pleiotropic effects and why tissue- or stage-specific intervention should provide greater precision.

Recent evidence from flowering Chinese cabbage further demonstrates the involvement of BR-responsive transcriptional regulation in leaf development. *BraBZR1* directly binds the *BraGRP5* promoter and activates its expression, while *BraGRP5* promotes leaf expansion. Functional characterization and transcriptomic analyses associate this regulatory module with cell-wall remodeling and hormone-responsive pathways, highlighting a molecular connection between BR signaling, cell expansion, and horticultural organ development [127].

6.2. JAZ–MYC and EIN3/EIL–ERF Networks Couple Defense with Development

JA and ethylene converge through JAZ–MYC and EIN3/EIL–ERF networks that couple defense signaling with developmental state. JA-Ile promotes COI1-dependent JAZ degradation and releases MYC activity, whereas ethylene stabilizes EIN3/EIL proteins and activates downstream ERF transcription. Their interaction is not intrinsically synergistic or antagonistic; the biological outcome depends on the regulatory branch engaged.

Tomato fruit provides a clear example. During *B. cinerea* infection, MYC2 interacts with the ripening regulators NOR and RIN to enhance the ERF.F4–PR-STH2 defense module [104], directly recruiting developmental TFs into JA-mediated immunity. Conversely, ripening-associated *SlEIL1* can suppress JA-responsive defense, and *sleil1* mutants show increased JA-associated transcription and gray-mold resistance [45]. These contrasting outcomes demonstrate that hormone crosstalk is determined by TF partnerships and developmental competence rather than by hormone abundance alone. JAZ–MYC and EIN3/EIL–ERF modules therefore function as adjustable interfaces through which ripening and immunity can be coordinated rather than obligatorily traded against one another.

6.3. WRKY/NAC and NPR–TGA Networks Integrate Stress and Immunity

WRKY and NAC factors operate as broad transcriptional relays connecting hormone, redox, developmental, and immune signals. WRKY proteins participate in SA-, JA-, ABA-, and ethylene-associated regulation, whereas NAC factors frequently connect stress responses with ripening, senescence, and postharvest physiology. In sweet orange, *CsWRKY53* directly activates *CsWAKL01*, linking receptor-like kinase signaling with SA-, JA-, and ABA-associated responses during citrus canker resistance [107]. In grapevine, *NAC61* regulates *DHN1b*, *MYB14*, and *WRKY52* and cooperates with *NAC60*, connecting late ripening and postharvest dehydration with osmotic protection, stilbene metabolism, and *B. cinerea* susceptibility [48]. These examples position WRKY and NAC proteins as network relays rather than outputs of single hormone pathways.

NPR1 illustrates a different form of convergence based on co-regulator assembly. SA promotes formation of NPR1–TGA transcriptional complexes, while structural evidence shows that SA-bound *NPR1* can recruit MED15A and connect promoter-bound TGAs with the general transcription machinery [36,37]. Competition by *NIMIN1* and changes in NPR1 abundance further modify this output. *NPR1* therefore functions as a conditional molecular scaffold rather than a simple SA on/off switch, allowing immune transcription to vary according to co-regulator availability and cellular state.

6.4. Context Determines Signal Priority and Engineering Outcome

The activity of convergence hubs is ultimately determined by where, when, and for how long they operate. Shared regulators compete for partners and promoter occupancy, so DELLA sequestration of growth regulators, *MYC2* association with ripening TFs, or *NPR1* recruitment of TGA–Mediator complexes can redirect transcription without requiring major changes in hormone abundance. Growth–defense trade-offs are therefore regulatory as well as metabolic, arising from changes in the availability and interaction of shared signaling components. Spatial and temporal context further modifies these decisions. Hormone

gradients interact with receptor abundance, TF and repressor availability, chromatin accessibility [40–43], and developmental identity, allowing neighboring tissues to interpret similar signals differently. Signal duration is equally important: transient activation may promote acclimation, whereas prolonged signaling can reinforce growth restraint or senescence. These principles are particularly relevant in horticultural crops because ripening stage, organ identity, and reproductive status strongly influence hormone-responsive transcription. Single-cell and spatial approaches should therefore help resolve where convergence hubs become functionally active rather than merely expressed.

Chromatin adds a history-dependent layer to this regulation. DNA methylation, histone modifications, nucleosome organization, and chromatin-associated co-regulators determine whether hormone-responsive TFs can access target loci [40–43]. In tomato, repeated dehydration produces altered transcriptional responses during subsequent stress, including changes in ABA signaling, stress-responsive TFs, ROS pathways, and chromatin-associated genes [128]; however, direct causal roles for individual epigenetic marks remain unresolved.

For crop improvement, these observations argue against constitutive manipulation of highly connected hubs. Greater precision should come from targeting specific regulatory edges, including tissue-specific or inducible promoters, *cis*-regulatory elements, phosphorylation sites, and protein-interaction interfaces so that desirable stress or immune outputs can be enhanced while preserving growth, fruit development, yield, and recovery.

7. Outstanding Questions and Future Directions

How conserved are hormone–TF networks across horticultural crops? Distinguish truly conserved mechanisms from lineage-specific rewiring through direct genetic and biochemical validation in crop species.

How do domestication, polyploidy, and stress-induced endoreduplication influence hormone–TF networks? Determine how genome duplication and *cis*-regulatory evolution reshape regulatory networks, and whether stress-associated endoreduplication contributes to cellular enlargement, metabolic adjustment, and tolerance to osmotic and oxidative stresses.

How does cellular context determine transcriptional output? Resolve how tissue identity, developmental stage, hormone dose, signal duration, and chromatin state redirect shared regulatory hubs.

How are growth, stress, and immune signals prioritized? Define the molecular rules governing signal hierarchy, trade-offs, and recovery under simultaneous or sequential challenges.

Which hormone–TF interactions are suitable for precision engineering? Prioritize specific regulatory edges that can be modified through tissue-specific, inducible, or *cis*-regulatory strategies with minimal pleiotropy.

How can hormone–TF networks be translated into improved horticultural performance? Evaluate targeted regulatory interventions for enhancing fruit firmness, pigmentation, flavor-related metabolism, shelf life, and postharvest disease resistance while preserving yield and developmental stability across diverse genotypes and environments.

Together, these questions mark the transition from descriptive hormone–TF maps to causal, context-resolved, predictive, and precisely engineerable regulatory networks.

8. Conclusions

Hormone–TF networks provide a unifying framework through which horticultural crops integrate developmental, environmental, and immune signals. Hormonal inputs converge on shared TFs, repressors, co-regulators, and protein-interaction hubs shaped by post-translational regulation, chromatin accessibility, and cellular context. This connectivity enables coordinated control of development and stress adaptation but also creates

pleiotropic effects. Future progress requires moving from descriptive hormone-response maps toward causal, context-resolved regulatory models. Integrating crop-specific genetics, spatial and temporal multi-omics, protein-interaction analysis, and field validation with tissue-specific regulation and *cis*-regulatory editing should enable precise manipulation of hormone–TF networks while preserving growth, reproductive performance, fruit quality, yield, resilience, and sustainability.

Author Contributions: L.H. and H.W. proposed an idea for this review. L.C., A.G., H.W. and L.D. assembled the literature to write the manuscript. L.C., L.D., J.D., J.W., Z.Q., Y.L., J.Y. and L.Z. conducted the investigation, used software, and drew the figures. L.H. secured funding, reviewed and edited the manuscript. All authors have read and agreed to the published version of the manuscript.

Funding: This study was supported by the 'Tianchi Talent' Project of Xinjiang Uygur Autonomous Region, the National Natural Science Foundation of China (32660842), the Breeding of New Melon Variety 'Zidingmi No. 22' Project (2026YD020), and the China Postdoctoral Science Foundation (2023MD734232).

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Data sharing is not applicable. No new data were created or analyzed in this study.

Acknowledgments: We thank Rahat Sharif (SCAU, Guangzhou, China) for critical revision of the manuscript. Regarding the Generative AI usage inquiry, We used Google Gemini and DouBao for language polishing, grammar correction, and text refinement during manuscript preparation. All AI-generated outputs were carefully reviewed, revised, and validated by all authors. The authors take full responsibility for all content in this manuscript.

Conflicts of Interest: The authors declare that they have no conflict of interest.

References

1. Murtaza, G.; Ahmed, Z.; Ullah, S.; Alotaibi, M.S.; Ali, S.; Iqbal, R. Hormone-metabolism crosstalk in plants: An integrated signaling network coordinating growth and stress adaptation. *Plant Signal Behav.* **2026**, *21*, 2710517. [CrossRef] [PubMed]
2. Ullah, S.; Matin, M.N.; Yin, C.; Mas-Ud, M.A.; Khan, A.; Islam, M.S.; Irfanullah; Haq, I.U. Phytohormone-Mediated Regulation of Plant Cold Stress Tolerance: Signaling, Hormonal Crosstalk, and Translational Perspectives. *Int. J. Mol. Sci.* **2026**, *27*, 4085. [CrossRef] [PubMed]
3. Jardim-Messeder, D.; de Souza-Vieira, Y.; Sachetto-Martins, G. Dressed Up to the Nines: The Interplay of Phytohormones Signaling and Redox Metabolism During Plant Response to Drought. *Plants* **2025**, *14*, 208. [CrossRef] [PubMed]
4. Liu, R.; Niimi, H.; Ueda, M.; Takaoka, Y. Coordinately regulated transcription factors EIN3/EIL1 and MYCs in ethylene and jasmonate signaling interact with the same domain of MED25. *Biosci. Biotechnol. Biochem.* **2022**, *86*, 1405–1412. [CrossRef] [PubMed]
5. Dubey, S.M.; Han, S.; Stutzman, N.; Prigge, M.J.; Medvecká, E.; Platre, M.P.; Busch, W.; Fendrych, M.; Estelle, M. The AFB1 auxin receptor controls the cytoplasmic auxin response pathway in *Arabidopsis thaliana*. *Mol. Plant* **2023**, *16*, 1120–1130. [CrossRef] [PubMed]
6. Mäkilä, R.; Wybouw, B.; Smetana, O.; Vainio, L.; Solé-Gil, A.; Lyu, M.; Ye, L.; Wang, X.; Siligato, R.; Jenness, M.K.; et al. Gibberellins promote polar auxin transport to regulate stem cell fate decisions in cambium. *Nat. Plants* **2023**, *9*, 631–644. [CrossRef] [PubMed]
7. Wang, X.; Pan, L.; Wang, Y.; Meng, J.; Deng, L.; Niu, L.; Liu, H.; Ding, Y.; Yao, J.-L.; Nieuwenhuizen, N.J.; et al. PpIAA1 and PpERF4 form a positive feedback loop to regulate peach fruit ripening by integrating auxin and ethylene signals. *Plant Sci.* **2021**, *313*, 111084. [CrossRef] [PubMed]
8. Li, X.; Martín-Pizarro, C.; Zhou, L.; Hou, B.; Wang, Y.; Shen, Y.; Li, B.; Posé, D.; Qin, G. Deciphering the regulatory network of the NAC transcription factor FvRIF, a key regulator of strawberry (*Fragaria vesca*) fruit ripening. *Plant Cell* **2023**, *35*, 4020–4045. [CrossRef] [PubMed]
9. Chen, X.; Gao, J.; Shen, Y. Abscisic acid controls sugar accumulation essential to strawberry fruit ripening via the FaRIPK1-FaTCP7-FaSTP13/FaSPT module. *Plant J.* **2024**, *119*, 1400–1417. [CrossRef] [PubMed]

10. da Silveira Falavigna, V.; Severing, E.; Lai, X.; Estevan, J.; Farrera, I.; Hugouvieux, V.; Revers, L.F.; Zubieta, C.; Coupland, G.; Costes, E.; et al. Unraveling the role of MADS transcription factor complexes in apple tree dormancy. *New Phytol.* **2021**, *232*, 2071–2088. [CrossRef] [PubMed]
11. Serivichyaswat, P.T.; Kareem, A.; Feng, M.; Melnyk, C.W. Auxin signaling in the cambium promotes tissue adhesion and vascular formation during *Arabidopsis* graft healing. *Plant Physiol.* **2024**, *196*, 754–762. [CrossRef] [PubMed]
12. Ferreira-Neto, J.R.C.; de Barros, A.A.G.; de Melo, A.L.T.M.; Amorim, L.L.B.; de Luna Aragão, M.A.; Bezerra-Neto, J.P.; Maciel, L.S.; da Silva, M.D.; da Silva, P.V.G.; Benko-Iseppon, A.M. The Regulatory Army of Plant Defense: Transcription Factors in the War for Plant Immunity. *Int. J. Mol. Sci.* **2026**, *27*, 7315. [CrossRef] [PubMed]
13. Kang, J.; Zhao, S.; Zhong, S.; Shi, H. Light-Controlled Seed Germination: Molecular Integration With Environmental Signals. *Plant Cell Environ.* **2026**, *49*, 6281–6295. [CrossRef] [PubMed]
14. Rienstra, J.; Hernández-García, J.; Weijers, D. To bind or not to bind: How AUXIN RESPONSE FACTORs select their target genes. *J. Exp. Bot.* **2023**, *74*, 6922–6932. [CrossRef] [PubMed]
15. Wang, J.; Fendler, N.L.; Shukla, A.; Wu, S.-Y.; Challa, A.; Lee, J.; Joachimiak, L.A.; Minna, J.D.; Chiang, C.-M.; Vos, S.M.; et al. ARF alters PAF1 complex integrity to selectively repress oncogenic transcription programs upon p53 loss. *Mol. Cell* **2024**, *84*, 4538–4557.e4512. [CrossRef] [PubMed]
16. Hu, J.; Li, X.; Sun, T.P. Four class A AUXIN RESPONSE FACTORs promote tomato fruit growth despite suppressing fruit set. *Nat. Plants* **2023**, *9*, 706–719. [CrossRef] [PubMed]
17. Sharif, R.; Su, L.; Chen, X.; Qi, X. Hormonal interactions underlying parthenocarpic fruit formation in horticultural crops. *Hortic. Res.* **2022**, *9*, uhab024. [CrossRef] [PubMed]
18. Li, Q.-F.; Wang, C.; Jiang, L.; Li, S.; Sun, S.S.M.; He, J.-X. An Interaction Between BZR1 and DELLAs Mediates Direct Signaling Crosstalk Between Brassinosteroids and Gibberellins in *Arabidopsis*. *Sci. Signal.* **2012**, *5*, ra72. [CrossRef] [PubMed]
19. Liu, Z.; Wang, Y.; Guan, P.; Hu, J.; Sun, L. Interaction of VvDELLA2 and VvCEB1 Mediates Expression of Expansion-Related Gene during GA-Induced Enlargement of Grape Fruit. *Int. J. Mol. Sci.* **2023**, *24*, 14870. [CrossRef] [PubMed]
20. Gan, H.; Wang, S.; Yang, Z.; Ma, P. Molecular Decoding of Phytohormone Crosstalk: JA-Mediated Key Regulatory Nodes and Signal Integration. *Plants* **2025**, *14*, 2647. [CrossRef] [PubMed]
21. Vancea, A.I.; Huntington, B.; Steinchen, W.; Savva, C.G.; Shahul Hameed, U.F.; Arold, S.T. Mechanism of cooperative strigolactone perception by the MAX2 ubiquitin ligase-receptor-substrate complex. *Nat. Commun.* **2025**, *16*, 10291. [CrossRef] [PubMed]
22. Kerr, S.C.; Patil, S.B.; de Saint Germain, A.; Pillot, J.P.; Saffar, J.; Ligerot, Y.; Aubert, G.; Citerne, S.; Bellec, Y.; Dun, E.A.; et al. Integration of the SMXL/D53 strigolactone signalling repressors in the model of shoot branching regulation in *Pisum sativum*. *Plant J.* **2021**, *107*, 1756–1770. [CrossRef] [PubMed]
23. Zheng, Z.-L. Cyclin-Dependent Kinases and CTD Phosphatases in Cell Cycle Transcriptional Control: Conservation across Eukaryotic Kingdoms and Uniqueness to Plants. *Cells* **2022**, *11*, 279. [CrossRef] [PubMed]
24. Zhang, W.J.; Zhou, Y.; Zhang, Y.; Su, Y.H.; Xu, T. Protein phosphorylation: A molecular switch in plant signaling. *Cell Rep.* **2023**, *42*, 112729. [CrossRef] [PubMed]
25. Ali, S.; Ullah, S.; Hayat, M.; Ahmad, H.; Shahbaz, Z.; Khan, M.T. Abscisic Acid-Mediated Drought Tolerance in *Populus*: Physiological, Molecular, and Biotechnological Perspectives. *Curr. Issues Mol. Biol.* **2026**, *48*, 946. [CrossRef] [PubMed]
26. Shen, X.; Yin, K.; Wang, Z.; Zhang, Z.; Liu, M.; Luo, S.; Xue, S.; Hu, H. OPEN STOMATA 1 activates SLAC1 anion channel primarily through CPK15 in ABA-induced stomatal closure in *Arabidopsis*. *Proc. Natl. Acad. Sci. USA* **2025**, *122*, e2518134122. [CrossRef] [PubMed]
27. Song, J.; Sun, P.; Kong, W.; Xie, Z.; Li, C.; Liu, J.H. SnRK2.4-mediated phosphorylation of ABF2 regulates ARGININE DECARBOXYLASE expression and putrescine accumulation under drought stress. *New Phytol.* **2023**, *238*, 216–236. [CrossRef] [PubMed]
28. Argueso, C.T.; Kieber, J.J. Cytokinin: From autoclaved DNA to two-component signaling. *Plant Cell* **2024**, *36*, 1429–1450. [CrossRef] [PubMed]
29. Mao, J.; Li, J. Regulation of Three Key Kinases of Brassinosteroid Signaling Pathway. *Int. J. Mol. Sci.* **2020**, *21*, 4340. [CrossRef] [PubMed]
30. Cao, X.; Wei, Y.; Shen, B.; Liu, L.; Mao, J. Interaction of the Transcription Factors BES1/BZR1 in Plant Growth and Stress Response. *Int. J. Mol. Sci.* **2024**, *25*, 6836. [CrossRef] [PubMed]
31. Liu, K.; Li, Y.; Chen, X.; Li, L.; Liu, K.; Zhao, H.; Wang, Y.; Han, S. ERF72 interacts with ARF6 and BZR1 to regulate hypocotyl elongation in *Arabidopsis*. *J. Exp. Bot.* **2018**, *69*, 3933–3947. [CrossRef] [PubMed]
32. Qiao, H.; Shen, Z.; Huang, S.S.; Schmitz, R.J.; Urich, M.A.; Briggs, S.P.; Ecker, J.R. Processing and subcellular trafficking of ER-tethered EIN2 control response to ethylene gas. *Science* **2012**, *338*, 390–393. [CrossRef] [PubMed]
33. Li, W.; Ma, M.; Feng, Y.; Li, H.; Wang, Y.; Ma, Y.; Li, M.; An, F.; Guo, H. EIN2-Directed Translational Regulation of Ethylene Signaling in *Arabidopsis*. *Cell* **2015**, *163*, 670–683. [CrossRef] [PubMed]

34. Deng, L.; Yang, T.; Li, Q.; Chang, Z.; Sun, C.; Jiang, H.; Meng, X.; Huang, T.; Li, C.-B.; Zhong, S.; et al. Tomato MED25 regulates fruit ripening by interacting with EIN3-like transcription factors. *Plant Cell* **2023**, *35*, 1038–1057. [CrossRef] [PubMed]
35. Chen, C.Y.; Shao, Z.; Wang, G.; Zhao, B.; Hardtke, H.A.; Leong, J.; Zhou, T.; Zhang, Y.J.; Qiao, H. Histone acetyltransferase HAF2 associates with pyruvate dehydrogenase complex to control H3K14ac and H3K23ac in ethylene response. *Cell Rep.* **2025**, *44*, 115580. [CrossRef] [PubMed]
36. Zhou, P.; Zavaliev, R.; Xiang, Y.; Dong, X. Seeing is believing: Understanding functions of NPR1 and its paralogs in plant immunity through cellular and structural analyses. *Curr. Opin. Plant Biol.* **2023**, *73*, 102352. [CrossRef] [PubMed]
37. Zhang, S.; Gish, M.; Li, H.; Hinds, T.R.; Mao, H.; Cornell, M.; Silvetti, M.; Klucas, S.; Vorauer, C.; Mundorff, C.C.; et al. Transcriptional activation of plant immunity by salicylic acid. *Nature* **2026**, *659*, 168–176. [CrossRef] [PubMed]
38. Morffy, N.; Faure, L.; Nelson, D.C. Smoke and Hormone Mirrors: Action and Evolution of Karrikin and Strigolactone Signaling. *Trends Genet.* **2016**, *32*, 176–188. [CrossRef] [PubMed]
39. Leydon, A.R.; Wang, W.; Gala, H.P.; Gilmour, S.; Juarez-Solis, S.; Zahler, M.L.; Zemke, J.E.; Zheng, N.; Nemhauser, J.L. Repression by the *Arabidopsis* TOPLESS corepressor requires association with the core mediator complex. *eLife* **2021**, *10*, e66739. [CrossRef] [PubMed]
40. Angidi, S.; Razzaq, K. How WRKY transcription factors fine-tune specificity in plant stress responses: From W-box to regulatory code. *Mol. Biol. Rep.* **2026**, *53*, 1148. [CrossRef] [PubMed]
41. Ray-Jones, H.; Spivakov, M. Transcriptional enhancers and their communication with gene promoters. *Cell. Mol. Life Sci.* **2021**, *78*, 6453–6485. [CrossRef] [PubMed]
42. Kim, S.; Wysocka, J. Deciphering the multi-scale, quantitative cis-regulatory code. *Mol. Cell* **2023**, *83*, 373–392. [CrossRef] [PubMed]
43. Wang, W.; Sung, S. Chromatin sensing: Integration of environmental signals to reprogram plant development through chromatin regulators. *J. Exp. Bot.* **2024**, *75*, 4332–4345. [CrossRef] [PubMed]
44. Sharif, R.; Su, L.; Chen, X.; Qi, X. Involvement of auxin in growth and stress response of cucumber. *Veg. Res.* **2022**, *2*, 13. [CrossRef]
45. Chen, Z.; Liu, J.; Zhimo, V.Y.; Wu, X.; Wisniewski, M. Biochemical and molecular regulation of tomato ripening and disease defense: A trade-off between quality and postharvest integrity. *Food Chem.* **2026**, *508*, 148476. [CrossRef] [PubMed]
46. Li, H.; Chen, Z.; Zhu, W.; Ni, X.; Wang, J.; Fu, L.; Chen, J.; Li, T.; Tang, L.; Yang, Y.; et al. The MaNAP1-MaMADS1 transcription factor module mediates ethylene-regulated peel softening and ripening in banana. *Plant Cell* **2024**, *37*, koae282. [CrossRef] [PubMed]
47. Wu, C.; Cai, D.; Li, J.; Lin, Z.; Wei, W.; Shan, W.; Chen, J.; Lu, W.; Su, X.; Kuang, J. Banana MabHLH28 positively regulates the expression of softening-related genes to mediate fruit ripening independently or via cooperating with MaWRKY49/111. *Hortic. Res.* **2024**, *11*, uhae053. [CrossRef] [PubMed]
48. Foresti, C.; Orduña, L.; Matus, J.T.; Vandelle, E.; Danzi, D.; Bellon, O.; Tornielli, G.B.; Amato, A.; Zenoni, S. NAC61 regulates late- and post-ripening osmotic, oxidative, and biotic stress responses in grapevine. *J. Exp. Bot.* **2024**, *75*, 2330–2350. [CrossRef] [PubMed]
49. Jia, H.; Xu, Y.; Deng, Y.; Xie, Y.; Gao, Z.; Lang, Z.; Niu, Q. Key transcription factors regulate fruit ripening and metabolite accumulation in tomato. *Plant Physiol.* **2024**, *195*, 2256–2273. [CrossRef] [PubMed]
50. Perotti, M.F.; Posé, D.; Martín-Pizarro, C. Non-climacteric fruit development and ripening regulation: 'the phytohormones show'. *J. Exp. Bot.* **2023**, *74*, 6237–6253. [CrossRef] [PubMed]
51. Bianchetti, R.; Ali, A.; Gururani, M. Abscisic acid and ethylene coordinating fruit ripening under abiotic stress. *Plant Sci.* **2024**, *349*, 112243. [CrossRef] [PubMed]
52. Feng, T.; Leng, W.; Yang, Y.; Zhao, H.; Zhang, Z.; Liu, M.; Liu, G. Gibberellins and their interplay with other hormones in the regulation of fruit ripening. *Hortic. Plant J.* **2026**, *12*, 1015–1024. [CrossRef]
53. Zuo, X.; Wang, S.; Liu, X.; Tang, T.; Li, Y.; Tong, L.; Shah, K.; Ma, J.; An, N.; Zhao, C.; et al. FLOWERING LOCUS T1 and TERMINAL FLOWER1 regulatory networks mediate flowering initiation in apple. *Plant Physiol.* **2024**, *195*, 580–597. [CrossRef] [PubMed]
54. Lloret, A.; Quesada-Traver, C.; Conejero, A.; Arbona, V.; Gómez-Mena, C.; Petri, C.; Sánchez-Navarro, J.A.; Zuriaga, E.; Leida, C.; Badenes, M.L.; et al. Regulatory circuits involving bud dormancy factor PpeDAM6. *Hortic. Res.* **2021**, *8*, 261. [CrossRef] [PubMed]
55. Liu, J.; Zhao, Y.; Yuan, Y.; Gao, L.; Liu, C.; Zhang, Y.; Gai, S. ATX1-COMPASS-like complex participates in the bud dormancy release of tree peony by regulating H3K4me3 modification. *Plant Physiol.* **2026**, *202*, kiag295. [CrossRef] [PubMed]
56. Zeng, X.; Wang, N.; Yang, Y.; Yan, R.; Liang, B.; Zhang, T.; Zhang, X.; An, N.; Zhang, D.; Wang, Y.; et al. MdKNOX19 promotes dwarfing and multi-branching in apple by regulating MdGA20ox3 and MdIPT1 expression. *Hortic. Plant J.* **2026**. [CrossRef]
57. Damodaran, S.; Strader, L.C. Factors governing cellular reprogramming competence in *Arabidopsis* adventitious root formation. *Dev. Cell* **2024**, *59*, 2745–2758.e3. [CrossRef] [PubMed]

58. Mao, J.; Niu, C.; Li, K.; Fan, L.; Liu, Z.; Li, S.; Ma, D.; Tahir, M.M.; Xing, L.; Zhao, C.; et al. Cytokinin-responsive MdTCP17 interacts with MdWOX11 to repress adventitious root primordium formation in apple rootstocks. *Plant Cell* **2023**, *35*, 1202–1221. [CrossRef] [PubMed]
59. Pan, J.; Sohail, H.; Sharif, R.; Hu, Q.; Song, J.; Qi, X.; Chen, X.; Xu, X. Cucumber JASMONATE ZIM-DOMAIN 8 interaction with transcription factor MYB6 impairs waterlogging-triggered adventitious rooting. *Plant Physiol.* **2025**, *197*, kiae351. [CrossRef] [PubMed]
60. Selwal, N.; Wani, A.K.; Akhtar, N.; Kaur, M.; Jassal, P.S. Molecular insights of strigolactone biosynthesis, signalling pathways, regulatory roles, and hormonal crosstalks in plant systems. *S. Afr. J. Bot.* **2023**, *160*, 9–22. [CrossRef]
61. Feng, J.; Wang, M.Y.; Chen, X.; Tomes, S.; Tao, J.; Atkinson, R.G.; Nieuwenhuizen, N.J. EIL (ethylene-insensitive 3-like) transcription factors in apple affect both ethylene- and cold response-dependent fruit ripening. *Plant J.* **2025**, *121*, e70059. [CrossRef] [PubMed]
62. Wang, S.; Li, L.-X.; Zhang, Z.; Fang, Y.; Li, D.; Chen, X.-S.; Feng, S.-Q. Ethylene precisely regulates anthocyanin synthesis in apple via a module comprising MdEIL1, MdMYB1, and MdMYB17. *Hortic. Res.* **2022**, *9*, uhac034. [CrossRef] [PubMed]
63. Huang, Y.; Wu, X.; Liu, Z.; Li, A.; Zhang, Z.; Yao, K.; Ding, Z.; Wang, C.; Liao, W. Ripening-related transcription factors during tomato fruit ripening: Crosstalk with ethylene. *Hortic. Plant J.* **2025**, *11*, 963–973. [CrossRef]
64. Li, B.-J.; Shi, Y.-N.; Xiao, Y.-N.; Jia, H.-R.; Yang, X.-F.; Dai, Z.-R.; Sun, Y.-F.; Shou, J.-H.; Jiang, G.-H.; Grierson, D.; et al. AUXIN RESPONSE FACTOR 2 mediates repression of strawberry receptacle ripening via auxin-ABA interplay. *Plant Physiol.* **2024**, *196*, 2638–2653. [CrossRef] [PubMed]
65. Qiao, K.; Yao, X.; Zhou, Z.; Xiong, J.; Fang, K.; Lan, J.; Xu, F.; Deng, X.; Zhang, D.; Lin, H. Mitochondrial alternative oxidase enhanced ABA-mediated drought tolerance in *Solanum lycopersicum*. *J. Plant Physiol.* **2023**, *280*, 153892. [CrossRef] [PubMed]
66. Zhang, T.-T.; Liu, Y.-Q.; Li, X.-W.; Liu, H.-F.; Wang, Y.-X.; Zhang, F.-J.; Wang, X.-F.; You, C.-X.; Lu, X.-Y. The drought-responsive factor MdDREB2A affects root development by directly regulating the transcription of MdARF6. *Environ. Exp. Bot.* **2023**, *213*, 105437. [CrossRef]
67. Zhao, W.; Huang, H.; Wang, J.; Wang, X.; Xu, B.; Yao, X.; Sun, L.; Yang, R.; Wang, J.; Sun, A.; et al. Jasmonic acid enhances osmotic stress responses by MYC2-mediated inhibition of protein phosphatase 2C1 and response regulators 26 transcription factor in tomato. *Plant J.* **2023**, *113*, 546–561. [CrossRef] [PubMed]
68. Zhu, C.; Li, X.; Zhang, M.; Wang, S.; Jing, B.; Hu, C.; Thomas, H.R.; Zhou, Y.; Yu, J.; Hu, Z. ERF.D2 negatively controls drought tolerance through synergistic regulation of abscisic acid and jasmonic acid in tomato. *Plant Biotechnol. J.* **2025**, *23*, 3363–3381. [CrossRef] [PubMed]
69. Zhang, X.; Du, B.; Turupu, M.; Xiao, Y.; Yao, Q.; Gai, S.; Zhang, Q.; Wang, X.; Yan, Y.; Wen, Z.; et al. Strigolactones enhance apple drought resistance via the MsABI5-MsSMXL1-MsNAC022 cascade. *Hortic. Res.* **2025**, *12*, uhaf101. [CrossRef] [PubMed]
70. Shohat, H.; Cheriker, H.; Kilambi, H.V.; Illouz Eliaz, N.; Blum, S.; Amsellem, Z.; Tarkowská, D.; Aharoni, A.; Eshed, Y.; Weiss, D. Inhibition of gibberellin accumulation by water deficiency promotes fast and long-term 'drought avoidance' responses in tomato. *New Phytol.* **2021**, *232*, 1985–1998. [CrossRef] [PubMed]
71. Zhang, Q.; Yuan, W.; Wang, Q.; Cao, Y.; Xu, F.; Dodd, I.C.; Xu, W. ABA regulation of root growth during soil drying and recovery can involve auxin response. *Plant Cell Environ.* **2022**, *45*, 871–883. [CrossRef] [PubMed]
72. Zamir, H.; Shah, D.A.; Rauf, F.; Ahmad, H.; Khan, S.; Jamali, Z.H.; Korai, S.K. Integrating Hormonal, Kinase and Transcription Factor Regulatory Networks in Plant Salinity Stress: Toward a Hierarchical Signal Integration Model. *J. Plant Biol.* **2026**, *69*, 173–194. [CrossRef]
73. Du, N.; Xue, L.; Xue, D.; Dong, X.; Yang, Q.; Shah Jahan, M.; Guo, H.; Fu, R.; Wang, Y.; Piao, F. The transcription factor SlNAP1 increases salt tolerance by modulating ion homeostasis and ROS metabolism in *Solanum lycopersicum*. *Gene* **2023**, *849*, 146906. [CrossRef] [PubMed]
74. Peng, Y.; Cao, H.; Cui, L.; Wang, Y.; Wei, L.; Geng, S.; Yang, L.; Huang, Y.; Bie, Z. CmoNAC1 in pumpkin rootstocks improves salt tolerance of grafted cucumbers by binding to the promoters of CmoRBOHD1, CmoNCED6, CmoAKT1;2 and CmoHKT1;1 to regulate H2O2, ABA signaling and K+/Na+ homeostasis. *Hortic. Res.* **2023**, *10*, uhad157. [CrossRef] [PubMed]
75. Shang, C.; Liu, X.; Chen, G.; Zheng, H.; Khan, A.; Li, G.; Hu, X. SlWRKY80-mediated jasmonic acid pathway positively regulates tomato resistance to saline–alkali stress by enhancing spermidine content and stabilizing Na+/K+ homeostasis. *Hortic. Res.* **2024**, *11*, uhae028. [CrossRef] [PubMed]
76. Wang, X.; Huo, Z.; Ma, L.; Ou, S.; Guo, M. The salt and ABA inducible transcription factor gene, SlAITR3, negatively regulates tomato salt tolerance. *Plant Physiol. Biochem.* **2025**, *222*, 109735. [CrossRef] [PubMed]
77. Takahashi, N.; Ogita, N.; Takahashi, T.; Taniguchi, S.; Tanaka, M.; Seki, M.; Umeda, M. A regulatory module controlling stress-induced cell cycle arrest in *Arabidopsis*. *eLife* **2019**, *8*, e43944. [CrossRef] [PubMed]
78. Ishka, M.R.; Craft, E.; Pineros, M.; Julkowska, M.M. Exogenous Hormone Treatments Reveal Species-Specific Regulation of Individual Components of Root Architecture and Salt Ion Accumulation in Cultivated and Wild Tomatoes. *Plant Direct* **2026**, *10*, e70169. [CrossRef] [PubMed]

79. Yang, J.; Guo, X.; Mei, Q.; Qiu, L.; Chen, P.; Li, W.; Mao, K.; Ma, F. MdbHLH4 negatively regulates apple cold tolerance by inhibiting MdCBF1/3 expression and promoting MdCAX3L-2 expression. *Plant Physiol.* **2023**, *191*, 789–806. [CrossRef] [PubMed]
80. Deng, M.; Zhao, Q.; Pang, H.; Yu, F.; Miao, B.; Yan, J.; Wang, J.; Fu, J.; Song, X.; Ma, F.; et al. ABSCISIC ACID INSENSITIVE 5 confers cold tolerance by regulating MdCBF3 and MdJAZ1/3 in apple. *Plant Physiol.* **2026**, *200*, kiaf612. [CrossRef] [PubMed]
81. Ding, F.; Wang, C.; Xu, N.; Wang, M. The ethylene response factor SlERF.B8 triggers jasmonate biosynthesis to promote cold tolerance in tomato. *Environ. Exp. Bot.* **2022**, *203*, 105073. [CrossRef]
82. Fang, P.; Wang, Y.; Wang, M.; Wang, F.; Chi, C.; Zhou, Y.; Zhou, J.; Shi, K.; Xia, X.; Foyer, C.H.; et al. Crosstalk between Brassinosteroid and Redox Signaling Contributes to the Activation of CBF Expression during Cold Responses in Tomato. *Antioxidants* **2021**, *10*, 509. [CrossRef] [PubMed]
83. Yu, W.; Jia, X.; Shen, L.; Sheng, J. Ethylene perception influences the chilling injury and SlCBF1 gene expression during cold storage of tomato fruit. *Postharvest Biol. Technol.* **2023**, *204*, 112434. [CrossRef]
84. Signorelli, S.; Velappan, Y.; Hermawaty, D.; Feil, R.; Couture, C.; Considine, J.A.; Lunn, J.E.; Considine, M.J. Seasonal changes in the physiology and metabolism of grapevine latent buds. *J. Exp. Bot.* **2026**, *77*, 1756–1772. [CrossRef] [PubMed]
85. He, M.; Wu, J.; Yang, Y.; Cui, Q.; Fan, N.; Chi, C.; Yan, H.; Liu, Y.; Pan, Z.; Zhang, K.; et al. The HsfA1a-BAG5b module mediates thermotolerance through activating autophagy in tomato. *Plant Physiol.* **2025**, *199*, kiaf526. [CrossRef] [PubMed]
86. Li, Y.; Gao, Z.; Lu, J.; Wei, X.; Qi, M.; Yin, Z.; Li, T. SlSnRK2.3 interacts with SlSUI1 to modulate high temperature tolerance via Abscisic acid (ABA) controlling stomatal movement in tomato. *Plant Sci.* **2022**, *321*, 111305. [CrossRef] [PubMed]
87. Chi, C.; Xu, X.; Wang, M.; Zhang, H.; Fang, P.; Zhou, J.; Xia, X.; Shi, K.; Zhou, Y.; Yu, J. Strigolactones positively regulate abscisic acid-dependent heat and cold tolerance in tomato. *Hortic. Res.* **2021**, *8*, 237. [CrossRef] [PubMed]
88. Xu, T.; Ran, T.; Shi, Y.; Yang, F.; Chen, X.; Sobieszczuk-Nowicka, E.; Fotopoulos, V.; Zhou, J. MYC2 interacts with JMJC3 to modulate jasmonate-regulated thermotolerance in tomato. *New Phytol.* **2026**, *249*, 2435–2451. [CrossRef] [PubMed]
89. Liang, Y.; Ma, F.; Huang, S.; Ma, C.; Shi, H.; Meng, F.; Chen, Y.; Dong, H.; Chen, R.; Zhan, X. Multifunctional NAC transcription factor SlJA2L integrates the ethylene pathway to orchestrate thermotolerance and fruit ripening in tomato. *New Phytol.* **2025**, *248*, 2409–2427. [CrossRef] [PubMed]
90. Kunta, S.; Aryee-Atta, A.; Dahan, Y.; Singh, Z.; Aharon, S.; Pasha, A.; Nir, I.; Lati, R.N.; Provart, N.J.; Burko, Y. Phytochrome-interacting factors integrate environmental signals to regulate tomato growth and development. *Plant Physiol.* **2026**, *201*, kiag379. [CrossRef] [PubMed]
91. Spaninks, K.; Offringa, R. Local phytochrome signalling limits root growth in light by repressing auxin biosynthesis. *J. Exp. Bot.* **2023**, *74*, 4642–4653. [CrossRef] [PubMed]
92. Yan, J.; Liu, J.; Yang, S.; Jiang, C.; Liu, Y.; Zhang, N.; Sun, X.; Zhang, Y.; Zhu, K.; Peng, Y.; et al. Light quality regulates plant biomass and fruit quality through a photoreceptor-dependent HY5-LHC/CYCB module in tomato. *Hortic. Res.* **2023**, *10*, uhad219. [CrossRef] [PubMed]
93. Wang, Y.; Wang, L.; Guan, Z.; Chang, H.; Ma, L.; Shen, C.; Qiu, L.; Yan, J.; Zhang, D.; Li, J.; et al. Structural insight into UV-B–activated UVR8 bound to COP1. *Sci. Adv.* **2022**, *8*, eabn3337. [CrossRef] [PubMed]
94. An, J.P.; Wang, X.F.; Zhang, X.W.; Bi, S.Q.; You, C.X.; Hao, Y.J. MdBBX22 regulates UV-B-induced anthocyanin biosynthesis through regulating the function of MdHY5 and is targeted by MdBT2 for 26S proteasome-mediated degradation. *Plant Biotechnol. J.* **2019**, *17*, 2231–2233. [CrossRef] [PubMed]
95. Zhang, Z.; Chen, C.; Lin, H.; Jiang, C.; Zhao, Y.; Guo, Y. The VvHY5-VvMYB24-VvMYBA1 transcription factor cascade regulates the biosynthesis of anthocyanin in grape. *Hortic. Plant J.* **2025**, *11*, 1066–1077. [CrossRef]
96. Wu, C.; Men, C.; Yan, L.; Zhang, J.; Wang, Y.; Chen, M.; Liu, C.; Zheng, L. The ERF transcription factor SlERF7 promotes UV-C-induced biosynthesis of phenolic compounds in tomato. *Sci. Hortic.* **2024**, *338*, 113643. [CrossRef]
97. Tao, J.; Wang, Y.; Fernie, A.R.; Ma, L.; Bai, C.; Liu, S.; Zhou, X.; Zuo, J.; Zheng, Y. Transcriptomic and epigenetic signatures of tomato fruit after postharvest UV-C irradiation are associated with the maintenance of fruit quality. *Plant Biotechnol. J.* **2025**, *23*, 4500–4502. [CrossRef] [PubMed]
98. Li, Y.; Jiang, F.; Niu, L.; Wang, G.; Yin, J.; Song, X.; Ottosen, C.-O.; Rosenqvist, E.; Mittler, R.; Wu, Z.; et al. Synergistic regulation at physiological, transcriptional and metabolic levels in tomato plants subjected to a combination of salt and heat stress. *Plant J.* **2024**, *117*, 1656–1675. [CrossRef] [PubMed]
99. Gaikwad, T.; Breen, S.; Breeze, E.; Stroud, E.; Hussain, R.; Kulasekaran, S.; Kargios, N.; Bennett, F.; de Torres-Zabala, M.; Horsell, D.; et al. Rapid local and systemic jasmonate signalling drives the initiation and establishment of plant systemic immunity. *Nat. Plants* **2026**, *12*, 152–163. [CrossRef] [PubMed]
100. Yokotani, N.; Hasegawa, Y.; Kouzai, Y.; Hirakawa, H.; Isobe, S. Transcriptome analysis of tomato plants following salicylic acid-induced immunity against *Clavibacter michiganensis* ssp. *michiganensis*. *Plant Biotechnol.* **2023**, *40*, 273–282. [CrossRef] [PubMed]
101. Lan, L.; Cao, L.; Zhang, L.; Fu, W.; Luo, C.; Wu, C.; Zeng, X.; Qu, S.; Yu, X.; Deng, W.; et al. A novel mode of WRKY1 regulating PR1-mediated immune balance to defend against powdery mildew in apple. *Mol. Hortic.* **2025**, *5*, 17. [CrossRef] [PubMed]

102. Li, Y.; Wu, F.; Li, C. Jasmonate signaling: Integrating stress responses with developmental regulation in plants. *J. Genet. Genom.* **2025**, *52*, 1490–1506. [CrossRef] [PubMed]
103. Wu, S.; Hu, C.; Zhu, C.; Fan, Y.; Zhou, J.; Xia, X.; Shi, K.; Zhou, Y.; Foyer, C.H.; Yu, J. The MYC2–PUB22–JAZ4 module plays a crucial role in jasmonate signaling in tomato. *Mol. Plant* **2024**, *17*, 598–613. [CrossRef] [PubMed]
104. Li, S.; Huang, Y.; Zhao, Y.; Wu, P.; Guo, S.; Yu, X.; Zhu, B.; Chen, K.; Grierson, D.; Gao, L. Ripening-induced defence signalling in *Botrytis cinerea*-infected tomato fruits involves activation of ERF.F4 by a MYC2-NOR/RIN protein complex. *Plant Biotechnol. J.* **2025**, *23*, 4126–4139. [CrossRef] [PubMed]
105. Yang, T.; Deng, L.; Wang, Q.; Sun, C.; Ali, M.; Wu, F.; Zhai, H.; Xu, Q.; Xin, P.; Cheng, S.; et al. Tomato CYP94C1 inactivates bioactive JA-Ile to attenuate jasmonate-mediated defense during fruit ripening. *Mol. Plant* **2024**, *17*, 509–512. [CrossRef] [PubMed]
106. Deng, H.; Pei, Y.; Xu, X.; Du, X.; Xue, Q.; Gao, Z.; Shu, P.; Wu, Y.; Liu, Z.; Jian, Y.; et al. Ethylene-MPK8-ERF.C1-PR module confers resistance against *Botrytis cinerea* in tomato fruit without compromising ripening. *New Phytol.* **2024**, *242*, 592–609. [CrossRef] [PubMed]
107. Yu, Q.; He, H.; Xian, B.; Zhang, C.; Zhong, X.; Liu, Y.; Zhang, M.; Li, M.; He, Y.; Chen, S.; et al. The wall-associated receptor-like kinase CsWAKL01, positively regulated by the transcription factor CsWRKY53, confers resistance to citrus bacterial canker via regulation of phytohormone signaling. *J. Exp. Bot.* **2024**, *75*, 5805–5818. [CrossRef] [PubMed]
108. Nomoto, M.; Skelly, M.J.; Itaya, T.; Mori, T.; Suzuki, T.; Matsushita, T.; Tokizawa, M.; Kuwata, K.; Mori, H.; Yamamoto, Y.Y.; et al. Suppression of MYC transcription activators by the immune cofactor NPR1 fine-tunes plant immune responses. *Cell Rep.* **2021**, *37*, 110125. [CrossRef] [PubMed]
109. Li, S.; Khoso, M.A.; Wu, J.; Yu, B.; Wagan, S.; Liu, L. Exploring the mechanisms of WRKY transcription factors and regulated pathways in response to abiotic stress. *Plant Stress* **2024**, *12*, 100429. [CrossRef]
110. Ahmad, S.; Sharif, R.; Feng, X.; Zeng, P.; Li, K.; Liu, J.; Tian, L. Jasmonic Acid and Salicylic Acid Pretreatment Differentially Modulates Photosynthesis-Related Transcription, Redox Homeostasis, and Defense Gene Expression in Mulberry Against *Glyphodes pyloalis*. *Horticulturae* **2026**, *12*, 1136. [CrossRef]
111. Aerts, N.; Pereira Mendes, M.; Van Wees, S.C.M. Multiple levels of crosstalk in hormone networks regulating plant defense. *Plant J.* **2021**, *105*, 489–504. [CrossRef] [PubMed]
112. Azaryan, A.; Atighi, M.R.; Shams-Bakhsh, M. Infection of tomato plants by tomato yellow leaf curl virus (TYLCV) potentiates the ethylene and salicylic acid pathways to fend off root-knot nematode (*Meloidogyne incognita*) parasitism. *Plant Physiol. Biochem.* **2024**, *217*, 109271. [CrossRef] [PubMed]
113. Geng, X.; Jin, L.; Shimada, M.; Kim, M.G.; Mackey, D. The phytotoxin coronatine is a multifunctional component of the virulence armament of *Pseudomonas syringae*. *Planta* **2014**, *240*, 1149–1165. [CrossRef] [PubMed]
114. Gahlowt, P.; Tripathi, D.K.; Gupta, R.; Singh, V.P. Tsw: An NLR protein that keeps viruses away. *Plant Cell Rep.* **2023**, *42*, 957–960. [CrossRef] [PubMed]
115. Duan, S.; Jiao, Z.; Lyu, H.; Li, M.; Chen, H. Immune activation drives citrus Huanglongbing. *Trends Plant Sci.* **2026**. [CrossRef] [PubMed]
116. Tzfira, T.; Frankman, L.R.; Vaidya, M.; Citovsky, V. Site-specific integration of *Agrobacterium tumefaciens* T-DNA via double-stranded intermediates. *Plant Physiol.* **2003**, *133*, 1011–1023. [CrossRef] [PubMed]
117. Noureddine, Y.; da Rocha, M.; An, J.; Médina, C.; Mejias, J.; Mulet, K.; Quentin, M.; Abad, P.; Zouine, M.; Favery, B.; et al. AUXIN RESPONSIVE FACTOR8 regulates development of the feeding site induced by root-knot nematodes in tomato. *J. Exp. Bot.* **2023**, *74*, 5752–5766. [CrossRef] [PubMed]
118. Prokchorchik, M.; Choi, S.; Chung, E.-H.; Won, K.; Dangl, J.L.; Sohn, K.H. A host target of a bacterial cysteine protease virulence effector plays a key role in convergent evolution of plant innate immune system receptors. *New Phytol.* **2020**, *225*, 1327–1342. [CrossRef] [PubMed]
119. Pazarlar, S.; Madriz-Ordeñana, K.; Thordal-Christensen, H. *Bacillus cereus* EC9 protects tomato against *Fusarium* wilt through JA/ET-activated immunity. *Front. Plant Sci.* **2022**, *13*, 1090947. [CrossRef] [PubMed]
120. Stringlis, I.A.; Yu, K.; Feussner, K.; de Jonge, R.; Van Bentum, S.; Van Verk, M.C.; Berendsen, R.L.; Bakker, P.A.H.M.; Feussner, I.; Pieterse, C.M.J. MYB72-dependent coumarin exudation shapes root microbiome assembly to promote plant health. *Proc. Natl. Acad. Sci. USA* **2018**, *115*, E5213–E5222. [CrossRef] [PubMed]
121. De Vleesschauwer, D.; Filipe, O.; Hoffman, G.; Seifi, H.S.; Haeck, A.; Canlas, P.; Van Bockhaven, J.; De Waele, E.; Demeestere, K.; Ronald, P.; et al. Target of rapamycin signaling orchestrates growth–defense trade-offs in plants. *New Phytol.* **2018**, *217*, 305–319. [CrossRef] [PubMed]
122. Leibman-Markus, M.; Schneider, A.; Gupta, R.; Marash, I.; Rav-David, D.; Carmeli-Weissberg, M.; Elad, Y.; Bar, M. Immunity priming uncouples the growth–defense trade-off in tomato. *Development* **2023**, *150*, dev201158. [CrossRef] [PubMed]
123. Rahman, S.; Beevi, S.S.; Nishana, F.; Masmoudi, K.; Muliyar Krishna, R.; Kurup, S.S. Gibberellin homeostasis and signaling in plant growth, stress adaptation, and crop improvement. *Curr. Plant Biol.* **2026**, *49*, 100654. [CrossRef]

124. Yan, R.; Zeng, X.; Wang, Y.; Wang, N.; Yang, Y.; Liang, B.; Zhang, X.; Qi, G.; Jia, P. The MdSLR1b-MdKNOX15-MdGA20ox2/MdGA2ox7 module regulates dwarfing by mediating the gibberellin pathway in apple. *Plant J.* **2026**, *127*, e71137. [CrossRef] [PubMed]
125. Xia, X.; Dong, H.; Yin, Y.; Song, X.; Gu, X.; Sang, K.; Zhou, J.; Shi, K.; Zhou, Y.; Foyer, C.H.; et al. Brassinosteroid signaling integrates multiple pathways to release apical dominance in tomato. *Proc. Natl. Acad. Sci. USA* **2021**, *118*, e2004384118. [CrossRef] [PubMed]
126. Zheng, Q.; Zhu, Y.; Wang, Q.; Song, X.; Liu, Y.; Feng, F.; Yu, J.; Zhou, Y.; Xia, X. Brassinosteroid signaling regulates PIN5/10-mediated intracellular auxin homeostasis to promote lateral root development in tomato. *Plant Commun.* **2026**, *7*, 101823. [CrossRef] [PubMed]
127. Su, W.; Ou, X.; Chen, K.; Yao, S.; Song, J.; Wang, H.; Liu, H.; Song, S.; Sharif, R.; Chen, R. The BZR1-GRP5 module regulates leaf growth by altering cell wall metabolism in Flowering Chinese cabbage (*Brassica rapa* ssp. *chinensis* var. *parachinensis*). *Plant Cell Rep.* **2026**, *45*, 178. [CrossRef] [PubMed]
128. Sadder, M.T.; Alsadon, A.A.; Alkharabsheh, B.S.; Musallam, A.; Alnajjar, A.M.; Al-Qadumii, L.W. Dehydration Stress Memory Genes in Tomato (*Solanum lycopersicum* L.). *Int. J. Mol. Sci.* **2026**, *27*, 6187. [CrossRef] [PubMed]

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.