REVIEW ARTICLE

Melatonin-Mediated Activation of the MKK5–MPK3/6 Cascade: A Molecular Framework for Integrated Drought Stress Tolerance in Plants

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Abstract

Drought stress is one of the most devastating abiotic stresses threatening global food security. Melatonin (N-acetyl-5-methoxytryptamine) and Mitogen-Activated Protein Kinase Kinase 5 (MKK5) are independently recognized as central regulators of plant stress adaptation; however, the molecular intersection of their signaling axes remains poorly characterized. This review proposes and examines a coherent molecular framework wherein melatonin, perceived via the phytomelatonin receptor PMTR1/CAND2, may converge on MKK5-containing MPK3/6 signaling modules as part of a broader MKK4/5/7/9 MAPK response network to orchestrate an integrated drought stress response. Key convergence points include the ABA-AIK1-MKK5-MPK6 module, reactive oxygen species (ROS) homeostasis, cooperative stomatal regulation, and the shared transcriptional targeting of WRKY and DREB factors. Understanding this melatonin–MKK5 axis opens new avenues for engineering drought-resilient crops in the context of accelerating climate change.

Keywords: melatonin, MKK5, MAPK cascade, PMTR1/CAND2, drought stress.

1 Introduction

Global climate change is intensifying the frequency and severity of drought events, posing a mounting threat to plant productivity and global food security under water-limited conditions [1]. In response to water deficit, plants activate multilayered molecular responses orchestrated by hormonal pathways, kinase cascades, transcription factor networks, and antioxidant defense systems. Among signaling hubs, the Mitogen-Activated Protein Kinase (MAPK) cascade, composed of hierarchically arranged MAPKKK, MAPKK, and MAPK tiers, is an evolutionarily conserved signal transduction module that converts diverse environmental stimuli into appropriate cellular responses [2]. Within this cascade, MKK5 functions as a MAPK kinase that relays upstream signals to MPK3 and MPK6 [3], while AIK1 acts upstream of the MKK5-MPK6 module in Arabidopsis ABA

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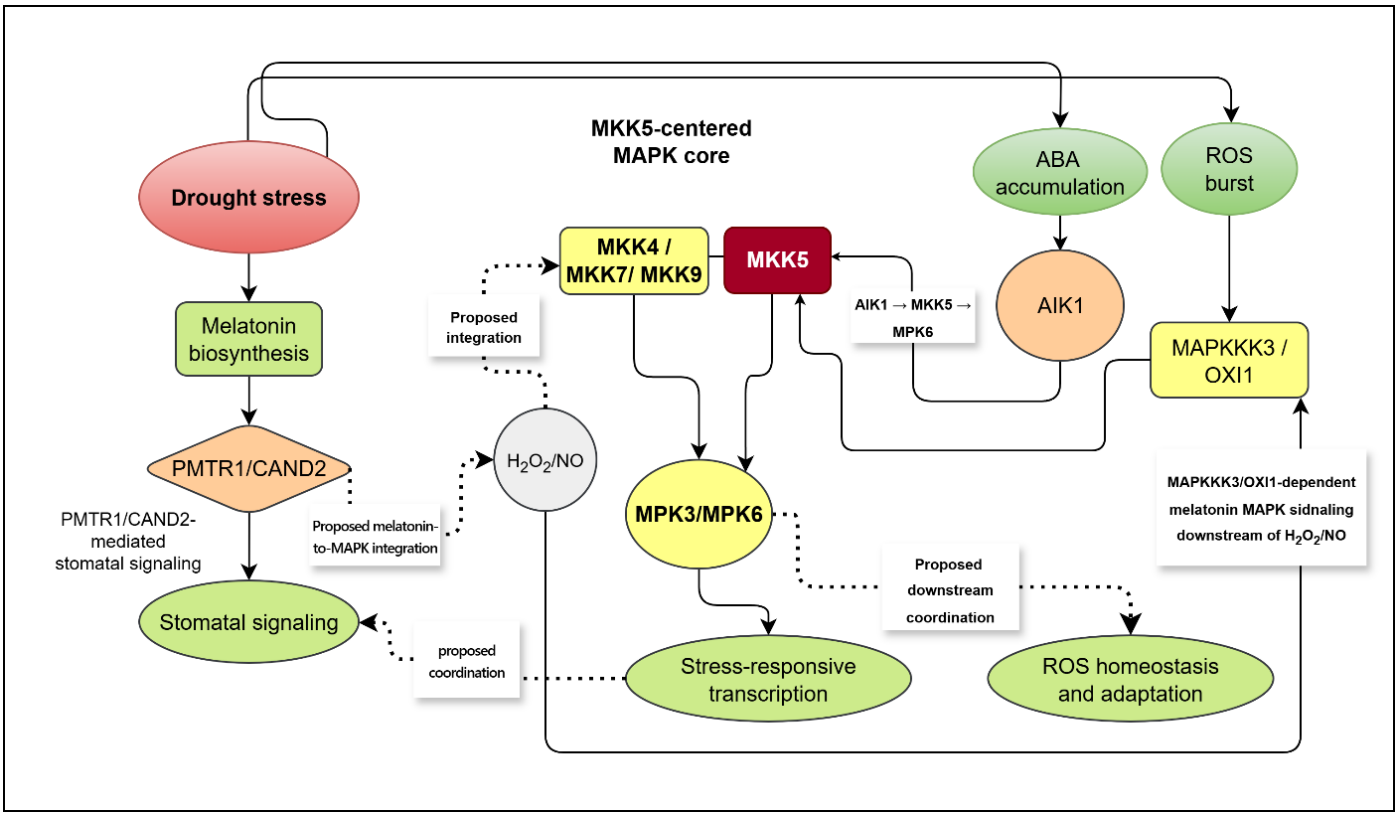

Figure 1. Proposed integrative model of melatonin, ABA, ROS, and MKK5-centered MAPK signaling during drought stress. Drought stress is depicted as a common upstream trigger promoting melatonin biosynthesis, ABA accumulation, and ROS burst, while melatonin signaling proceeds through PMTR1/CAND2 to regulate stomatal signaling directly. Within the central MKK5-centered MAPK core, MKK5 is highlighted as a distinct focal node alongside the MKK4/MKK7/MKK9 branch, with both routes converging on MPK3/MPK6 and then on stress-responsive transcription. The ABA-associated activation of AIK1 and a parallel ROS-related route converging through MAPKKK3/OXI1 toward the MAPK core. Dashed arrows indicate proposed integration steps, including melatonin-to-MAPK coupling through the H_2O_2/NO node and downstream coordination linking MAPK-dependent transcription with stomatal signaling and ROS homeostasis/adaptation, which are presented as working hypotheses rather than as interactions directly demonstrated within a single drought-specific experimental system.

responses [4]. In parallel, the MKK4/5-MPK3/6 modules regulate drought-associated genes such as NCED3 and RD29A and participate in an ABA-MKK4/5-MPK3/6-ABI5 feedback framework during drought stress [3]. Concurrently, melatonin (N-acetyl-5-methoxytryptamine) has emerged as a versatile phytohormone synthesized from tryptophan through the sequential enzymatic action of TDC, T5H, SNAT, and ASMT/COMT [5]. Endogenous melatonin levels rise rapidly during drought stress, and overexpression of melatonin biosynthesis genes confers improved tolerance in multiple plant species [6]. Melatonin signals are transduced through the phytomelatonin receptor PMTR1/CAND2, a GPCR-like plasma membrane protein first identified in Arabidopsis thaliana that exhibits saturable and specific binding ^{125}I-melatonin with a dissociation constant (Kd) of 0.73 $\pm$ 0.10 nmol/L [8]. Melatonin activates MPK3 and MPK6 in Arabidopsis, and this response requires MKK4, MKK5, MKK7, and MKK9 in a defense-signaling context [2]. Independently, PMTR1/CAND2 mediates melatonin-induced stomatal closure through GPA1-dependent H_2O_2 and Ca^{2+} signaling in guard cells [8], while MAPKKK3 and OXI1 function upstream of melatonin-mediated MPK3/MPK6 activation downstream of H_2O_2 and nitric oxide (NO) [9]. Taken together, these studies support a proposed PMTR1–ROS–MAPKKK3/OXI1–MKK5 signaling connection, but they do not yet prove a direct linear drought pathway. Despite these independent advances, both melatonin and MKK5 converge on overlapping downstream effectors, WRKY transcription factors, ROS-scavenging enzymes, ABI5, and stomatal regulators, yet the mechanistic integration of these two signaling arms has not been explicitly addressed. The present review synthesizes current evidence to propose a unified molecular

model in which melatonin engages MKK5-centric MAPK pathways to consolidate drought tolerance, and discusses the implications for biotechnological crop improvement (Figure 1).

2 Melatonin Biosynthesis and Drought-Induced Upregulation

2.1 Enzymatic Pathway from Tryptophan to Melatonin

Melatonin biosynthesis in plants proceeds from tryptophan via two major enzymatic pathways operating in distinct subcellular compartments [5]. The primary pathway is: tryptophan → tryptamine (by TDC) → serotonin (by T5H, localized to the endoplasmic reticulum) → N-acetylserotonin (by SNAT, in chloroplasts and cytoplasm) → melatonin (by ASMT or COMT) [5]. A secondary pathway operates when plants accumulate large pools of serotonin—as occurs during senescence and severe drought—and proceeds via 5-methoxytryptamine as an intermediate using COMT activity [5]. The dual cytoplasmic and chloroplastic localization of melatonin biosynthesis provides compartment-specific pools that can differentially modulate nuclear and organellar signaling pathways [5].

2.2 Drought-Induced Transcriptional Activation of Biosynthesis Genes

In rice, key melatonin biosynthetic genes with circadian regulation are associated with altered expression under abiotic stresses including drought, salt, and cold [12]. In Arabidopsis, osmotic stress likewise upregulates melatonin-related signaling components, supporting stress-responsive induction of the melatonin pathway under water-deficit-like conditions [10]. In apple (Malus), drought stress transcriptionally activates MdTDC1, MdAANAT2, MdT5H4, and MdASMT1, establishing an endogenous positive-feedback loop between melatonin accumulation and drought gene expression [7]. Exogenous melatonin applied to rice seedlings further upregulates TDC2 and ASMT1 expression, amplifying endogenous melatonin levels and enhancing antioxidant enzyme activities including SOD, POD, CAT, and APX [1].

2.3 PMTR1/CAND2: The Melatonin Receptor Linking Perception to MAPK Cascades

The phytomelatonin receptor PMTR1/CAND2 was discovered in Arabidopsis thaliana as the first receptor for melatonin in plants, exhibiting saturable binding ^{125}I-melatonin with a Kd of 0.73 $\pm$ 0.10 nmol/L, confirming high-affinity and specific receptor–ligand interaction [8]. PMTR1 is a membrane protein that interacts with the G-protein α subunit GPA1, and cand2 mutants are completely insensitive to melatonin-induced stomatal closure, demonstrating that PMTR1 is obligatory for melatonin's guard cell effects [8]. PMTR1 is required for melatonin-conferred osmotic stress tolerance, as cand2 mutants show greater ROS accumulation, reduced CAT1, CAT2, CAT3, and SOD1 expression, and lower catalase and SOD activities under osmotic stress [10]. Evidence for a direct drought role is stronger in maize, where ZmPMTR1 improves tolerance to osmotic and drought stress [11]. In maize, ZmPMTR1 overexpression promotes stomatal closure, reduces water loss, and confers enhanced drought tolerance, confirming conservation of PMTR1 function across monocots [11]. Downstream of PMTR1, melatonin-induced MPK3/MPK6 activation requires MKK4, MKK5, MKK7, and MKK9, indicating that MKK5 is one important component of the melatonin-responsive MAPK module rather than a uniquely established transducer acting alone [2, 3].

3 Molecular Convergence of Melatonin and MKK5 in Drought Signaling

3.1 The MAPKKK3/OXI1–MKK5–MPK3/6 Module Activated by Melatonin

Melatonin activates MPK3 and MPK6 rapidly and transiently within minutes of treatment in Arabidopsis and tobacco [2]. Four MKKs (MKK4, MKK5, MKK7, and MKK9) are responsible for mediating melatonin-induced MPK3/MPK6 activation, indicating that MKK5 is one of four obligate intermediates in this cascade [2, 3]. The most upstream melatonin-responsive MAPK kinase is MAPKKK3 together with OXI1 (oxidative signal-inducible 1), with H_2O_2 and NO acting as co-required second messengers; melatonin functions downstream of the ROS/NO burst to amplify defense gene expression including PR1 and ICS1 [9]. A working model consistent with the available evidence is that melatonin perception, PMTR1-dependent ROS/Ca^{2+} signaling, and MAPKKK3/OXI1-dependent MAPK activation may converge on MKK5-containing MPK3/MPK6 modules; however, the direct linear sequence PMTR1 → MAPKKK3/OXI1 → MKK5 has not yet been experimentally demonstrated in a drought-specific system and should therefore be treated as a proposed framework rather than an

established pathway [3, 9].

3.2 Convergence at the ABA–MKK5–ABI5 Regulatory Node

One of the most significant convergence points between melatonin and MKK5 is the ABA-MKK4/5-MPK3/6-ABI5 regulatory feedback loop during drought. Under water deficit, ABA accumulation activates SnRK2, which dissociates PP2C to enable ABI5 expression; subsequently, ABA-activated AIK1 (MKKK20) phosphorylates MKK5, which phosphorylates MPK6 to regulate stomatal responses via the AIK1-MKK5-MPK6 module [4]. MPK3, phosphorylated downstream of MKK5, further phosphorylates ABI5 specifically at Ser314, establishing a reinforcing ABA-MKK5-MPK3-ABI5 feedback loop that sustains drought-responsive transcription. Melatonin intersects this axis by selectively downregulating MdNCED3 (ABA biosynthesis gene) while upregulating MdCYP707A1 and MdCYP707A2 (ABA catabolism genes), thereby reducing ABA levels and modulating the upstream signal that activates AIK1-MKK5 signaling in drought-stressed apple plants [6, 7]. Melatonin clearly modulates ABA metabolism by repressing MdNCED3 and inducing MdCYP707A1/A2 in drought-stressed Malus plants [7].

3.3 Shared Downstream Transcription Factor Targets: WRKY and DREB

Both melatonin-activated MAPK signaling and MKK5-centered drought signaling converge on transcriptional reprogramming involving WRKY- and DREB-class factors. Lee et al. [2] found that melatonin activates MAPK-dependent defense signaling,

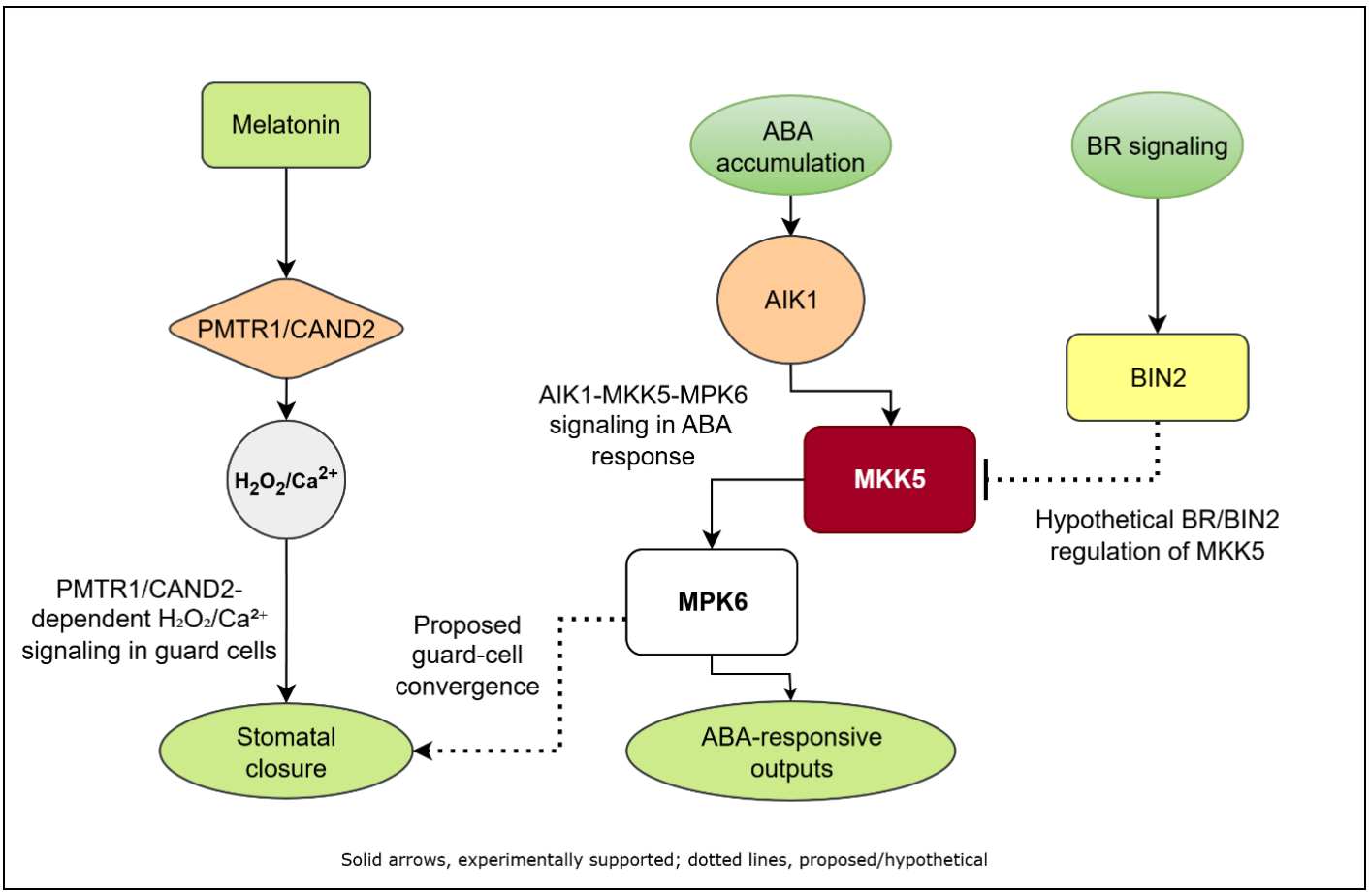

Figure 2. Proposed guard-cell model for convergence between melatonin signaling and MKK5-associated ABA responses during drought stress. Melatonin is perceived by PMTR1/CAND2 and triggers H_2O_2/Ca^{2+} signaling that promotes stomatal closure in guard cells. In parallel, ABA accumulation activates AIK1, which feeds into the MKK5–MPK6 module to regulate ABA-responsive outputs. MKK5 is highlighted as the central convergence node because it links established ABA signaling with the putative melatonin-associated MAPK branch discussed in this review. The dotted connection toward stomatal closure indicates a proposed site of guard-cell convergence rather than a directly validated linear pathway. The BR/BIN2 branch is shown as a provisional inhibitory layer at the MKK5 node, reflecting a hypothesis derived from cross-source synthesis that still requires direct experimental validation.

leading to downstream transcriptional outputs like WRKY-associated responses in Arabidopsis.

4 Melatonin–MKK5 Cooperation in Stomatal Guard Cell Regulation

4.1 Dual Regulation of Guard Cell Aperture

Stomatal regulation is a critical drought-adaptive mechanism, and both melatonin and MKK5 independently regulate guard cell aperture through complementary mechanisms [6]. Melatonin promotes stomatal closure via PMTR1-GPA1-H_2O_2-Ca^{2+} signaling in guard cells, and cand2 mutant guard cells fail to close in response to melatonin treatment [8]. Concurrently, MKK5 controls stomatal activity via the AIK1-MKK5-MPK6 module [4] and through the YODA-MKK4/5-MPK3/6 cascade that regulates the transcription factors SPEECHLESS (SPCH), MUTE, and FAMA, which determine stomatal cell fate and development. Because melatonin-induced signaling can engage MAPKKK3/OXI1-dependent MAPK activation, and ABA-activated AIK1 also feeds into MKK5, guard cells represent a plausible site of convergence among melatonin, ABA, and ROS signaling during drought; however, the complete integrated MKK5-centered module remains a model rather than a single experimentally verified pathway [4, 6, 8, 9](Figure 2).

5 BIN2-Mediated Post-Translational Regulation and BR-Melatonin Interplay

5.1 A Putative Regulatory Layer at the MKK5 Node

Post-translational regulation may provide an additional control layer over the proposed melatonin–MKK5 drought-signaling framework. BIN2 can be considered a plausible negative regulatory checkpoint at the MKK5 node, potentially influencing the intensity or duration of downstream MKK5-dependent responses. This potential connection is significant as it provides a mechanistic link where the stress signaling of melatonin could interact with brassinosteroid pathways and other hormonal crosstalk during drought adaptation [3, 6].

5.2 Status of Experimental Validation

At present, however, a direct melatonin–BR–BIN2–MKK5 regulatory sequence has not been experimentally demonstrated in a drought-specific system. Therefore, this putative BIN2-centered regulatory layer should be presented as a working hypothesis rather than as an established linear pathway. Future studies combining melatonin treatment with BIN2 genetic perturbation and MKK5 phosphorylation analysis will be required to determine whether melatonin modifies this post-translational checkpoint during drought stress.

6 Melatonin–MKK5 Synergy in ROS Homeostasis

6.1 Complementary ROS Regulation Mechanisms

Reactive oxygen species (ROS) function as both damage-inducing agents and second messengers in drought-stressed plants, and both melatonin and MKK5 regulate ROS homeostasis through complementary mechanisms. MKK5 activates the MEKK1-MKK4/MKK5-MPK3/MPK6-FSD1/2 pathway, which elevates iron superoxide dismutase (FeSOD) activity and reduces superoxide accumulation. MKK5 also enhances expression of CSD1 and CSD2 (copper/zinc SODs) through MPK6 under oxidative stress, and regulates H_2O_2-induced expression of GST and HSP through the ANP1-MKK4/5-MPK3/6 module. Simultaneously, melatonin acts as a direct antioxidant scavenging H_2O_2 in Malus species [7] and enhances enzymatic antioxidant activities (SOD, POD, CAT, and APX) during drought in rice seedlings [1].

6.2 H_2O_2 Threshold Control and Prevention of Cell Death

Mechanistically, PMTR1-mediated melatonin signaling activates MAPK cascades via H_2O_2 generated by NADPH oxidase downstream of GPA1 [8], creating a positive feedback where controlled H_2O_2 pulses activate MAPKKK3/OXI1-MKK5 signaling [9]. However, prolonged MKK5 activation can generate excessive H_2O_2 leading to cell death in Arabidopsis. The antioxidant capacity of Melatonin, through both direct H_2O_2 scavenging and enhancement of SOD, POD, CAT, and APX activities, is consistent with a buffering role against excessive oxidative outputs associated with stress signaling, although direct threshold control over MKK5 activity has not yet been demonstrated [1, 6, 7].

7 Biotechnological Implications for Drought-Resilient Crop Engineering

7.1 Genetic Strategies Targeting the Melatonin–MKK5 Axis

Understanding the melatonin–MKK5 interface presents multiple genetic engineering entry points for crop improvement. Overexpression

Table 1. Comparative analysis of melatonin-mediated and MKK5-mediated drought stress response components and their molecular intersection.

Signaling component	Evidence from MKK5-centered signaling	Evidence from melatonin signaling	Integrated interpretation	Evidence status
Receptor and upstream input	MKK5 functions downstream of upstream MAPK kinases in stress signaling and is activated in the AIK1-MKK5-MPK6 ABA module [4]	PMTR1/CAND2 is a phytomelatonin receptor required for melatonin-triggered stomatal signaling and osmotic stress tolerance [8]	Melatonin perception and ABA signaling provide distinct upstream inputs that may converge on MKK5-containing MAPK modules [10]	[4, 8, 10]
MAPK cascade architecture	MKK5 participates in MPK3/MPK6 activation in stress-related MAPK cascades [4]	Melatonin-induced MPK3/MPK6 activation requires MKK4, MKK5, MKK7, and MKK9 rather than MKK5 alone [2]	Melatonin can engage MAPK signaling that includes MKK5, but a uniquely MKK5-specific melatonin branch has not been established [2]	Directly supported for MKK4/5/7/9-dependent MAPK activation; MKK5 preference remains unproven [2, 3].
MAPKKK-level linkage	The MKK5 within broader upstream MAPKKK-controlled stress pathways	MAPKKK3 and OXI1 act upstream of melatonin-mediated MPK3/MPK6 activation downstream of H_2O_2 and NO [9]	A MAPKKK3/OXI1-to-MKK5 connection is plausible but has not been directly demonstrated as a linear drought-specific pathway [9]	Partial support; integrative step is proposed [2, 9]
ABA crosstalk	AIK1 activates the MKK5-MPK6 cascade in ABA responses [4]	Melatonin modulates ABA metabolism by repressing *MdNCED3* and inducing *MdCYP707A1/A2* under drought [7]	Melatonin may influence the ABA input feeding into MKK5-associated signaling by reshaping ABA accumulation and catabolism [7]	Supported for ABA metabolism and AIK1-MKK5 separately; their functional coupling is inferred [4, 7]
Osmotic/water-deficit tolerance	MKK5-associated cascades are discussed in drought and osmotic stress adaptation	PMTR1/CAND2 is required for melatonin-conferred osmotic stress tolerance in Arabidopsis, and *ZmPMTR1* improves osmotic and drought tolerance in maize [10]	Melatonin signaling clearly contributes to water-deficit adaptation, but direct proof that this depends specifically on MKK5 in drought remains incomplete [10]	Strong support for PMTR1 in osmotic/drought tolerance; MKK5-specific dependency remains incomplete [2, 10, 11]
Guard-cell regulation	AIK1-MKK5-MPK6 regulates ABA responses, and MKK4/5-associated stomatal developmental pathways [4]	PMTR1/CAND2 mediates melatonin-induced stomatal closure through GPA1-dependent H_2O_2 and Ca^{2+} signaling [8]	Guard cells are the most plausible cellular site of melatonin–MKK5 convergence, but the full merged pathway remains a model rather than a single experimentally verified module [8]	Supported for each arm individually; integrated guard-cell module is proposed [4, 8]
ROS regulation	MKK5-centered cascades regulate ROS-responsive outputs and antioxidant-related genes	Melatonin acts as an antioxidant and enhances SOD, POD, CAT, and APX activities in drought-stressed rice seedlings [7]	Melatonin likely buffers oxidative outputs associated with MKK5-linked stress signaling, helping maintain adaptive rather than damaging ROS levels [7]	Supported for antioxidant action; direct ROS-threshold control over MKK5 remains hypothetical [1, 3, 7]
Transcriptional outputs	The MKK5-associated signaling to WRKY-, DREB-, and ABI5-related drought outputs.	Melatonin activates MAPK-dependent transcriptional defense outputs and broader stress-responsive gene expression [2]	Convergence at transcriptional reprogramming is likely, but specific WRKY59/DREB2 integration with melatonin is not yet directly demonstrated [2]	General convergence supported; specific node-level integration remains inferential [2]
Post-translational control	BIN2-mediated inhibition of MKK5	The manuscript proposes that melatonin may affect this layer through brassinosteroid-related crosstalk	A melatonin-BR-BIN2-MKK5 circuit should be treated as a hypothesis until directly tested	Hypothesis only in current manuscript
Biosynthesis and co-induction under stress	Drought-responsive roles of MKK5	Melatonin biosynthetic genes such as *TDC*, *T5H*, *SNAT1*, and *ASMT1* respond to abiotic stress [12]	Stress-driven co-activation of melatonin production and MKK5-linked signaling is plausible, but direct co-induction has not been experimentally resolved [12]	Hypothesis only in current manuscript

of melatonin biosynthetic genes, particularly SNAT and COMT, elevates endogenous melatonin levels and confers improved tolerance in transgenic plants; because elevated melatonin can signal through PMTR1/CAND2 and engage MAPK pathways that include MKK5-containing modules, this strategy may prime part of the stress-signaling network rather than a uniquely MKK5-specific route [2]. Conversely, engineering constitutively active forms of MKK5 (e.g., MKK5DD-T215D/S221D) can bypass upstream requirements and directly activate downstream drought gene expression. However, the risk of MKK5DD-induced H_2O_2-mediated cell death must be mitigated, and co-expression with melatonin biosynthetic genes could provide the antioxidant buffering needed to maintain MKK5 activity within a safe range. CRISPR-Cas9 editing of BIN2, which inhibits MKK5 at Thr-234, combined with overexpression of SNAT or ASMT, represents a synergistic dual-gene strategy to simultaneously enhance MKK5 kinase activity and provide antioxidant protection [3].

7.2 Exogenous Melatonin as a MAPK-Activating Agronomic Tool

Exogenous melatonin application at 100–200 μM has consistently improved drought tolerance across diverse species including wheat, rice, maize, tomato, apple, and kiwifruit. Given that melatonin-induced MAPK activation can involve MKK4/5 in Arabidopsis, the agronomic benefits of exogenous melatonin are consistent with possible engagement of endogenous MKK5-containing cascades, but direct demonstration of an MKK5-specific mechanism in drought-treated crops is still lacking [2, 3, 6, 11]. In maize, ZmPMTR1 overexpression reduces water loss and improves drought tolerance, supporting a role for phytomelatonin perception in water-deficit adaptation but not, by itself, proving a direct PMTR1-to-MKK5 drought signaling sequence [9, 11]. Furthermore, melatonin pre-treatment in rice dramatically upregulates antioxidant enzyme genes and improves relative water content, melatonin pre-treatment in rice upregulates antioxidant enzyme genes and improves relative water content under drought [1], findings that are consistent with improved oxidative stress management but do not by themselves demonstrate direct activation of specific MKK5-FSD1/2 or MKK5-CSD1/2 branches. Future agronomy trials should correlate melatonin treatment efficacy with MKK5 transcript levels and MPK3/MPK6 phosphorylation status to validate the melatonin-MKK5 mechanism in field conditions [1].

8 Future Perspectives

The melatonin–MKK5 signaling axis described in this review constitutes a compelling but incompletely characterized nexus in plant drought adaptation. Several critical knowledge gaps require resolution by future research. First, direct biochemical evidence for the specificity of PMTR1-MAPKKK3-mediated activation of MKK5 relative to other MKKs remains to be established through phosphoproteomics and kinase specificity assays [9]. Second, the spatiotemporal dynamics of melatonin-induced MKK5 activation specifically within guard cells during drought require investigation using FRET-based kinase activity biosensors in live cells. Third, the proposed melatonin-BR-BIN2-MKK5 regulatory circuit remains hypothetical and requires direct validation using BIN2 genetic perturbation, exogenous melatonin treatment, and MKK5 phosphorylation analysis [3, 4]. Fourth, single-cell transcriptomics and phosphoproteomics during co-application of melatonin and drought stress would comprehensively map the shared and distinct downstream targets of melatonin and MKK5. Fifth, characterizing MKK5 homologs across horticultural and cereal crops will clarify which elements of the proposed PMTR1–MKK5 axis are conserved, particularly the experimentally supported versus inferred nodes (Table 1). Combined with CRISPR-based precision editing, melatonin priming strategies, and advanced agronomic delivery systems, the melatonin–MKK5 axis holds significant promise for engineering the next generation of drought-resilient crops capable of sustaining yield under intensifying climate stress.

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Conflicts of Interest

The authors declare no conflicts of interest.

AI Use Statement

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Ethical Approval and Consent to Participate

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