Transactions on Materials, Biotechnology and Life Sciences
ISSN: 2960-1932, eISSN: 2960-2319 | Volume 9
FENBC 2026

A Review of ABA in the Regulation of Plant Growth-Drought Resistance Balance

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Abstract. Abscisic Acid (ABA) is a core phytohormone for plants to respond to drought stress and balance growth and stress resistance. This paper reviews the upstream perception and initiation of the ABA signaling pathway, the SnRK1-TOR energy hub, transcriptional regulation and downstream responses, as well as the crosstalk mechanism between ABA and other phytohormones including auxin and brassinosteroid (BR). It focuses on the regulatory logic of key nodes in each module, and reveals the dynamic regulatory principle of ABA through a multi-level network to maintain growth under non-stress conditions and promote drought resistance under stress conditions. This review provides a theoretical framework for analyzing the molecular mechanism of the plant growth-drought resistance trade-off and crop stress resistance breeding.

Keywords: Abscisic Acid (ABA); Plant Growth; Drought Resistance; Signaling Pathway; Hormone Crosstalk; Growth-Drought Resistance Balance.

1. Introduction

Plants are exposed to multiple abiotic stresses throughout their growth and development, among which drought is the most dominant stress factor restricting plant growth and reproduction. Plants have evolved a dynamic "growth-drought resistance" balance strategy during long-term evolution: they initiate defense responses to improve drought resistance under drought conditions, often accompanied by growth inhibition; under normal water supply, they give priority to growth and inhibit redundant drought resistance pathways to save resources. As the core phytohormone of plant stress response, ABA is the central hub regulating this balance [1]. It is not only widely involved in a variety of stress responses, but also regulates multiple key developmental processes such as seed germination, vegetative growth, and stomatal movement. At present, the multi-level regulatory mechanism of the ABA signaling pathway has been gradually deciphered. This paper systematically sorts out the regulatory logic of the whole chain of the ABA signaling pathway and its crosstalk network with other phytohormones, and focuses on elucidating the core molecular mechanism of the ABA-mediated plant growth-drought resistance balance, providing theoretical support for relevant basic research and crop stress resistance breeding.

2. Core Regulatory Modules and Functional Mechanisms of the ABA Signaling Pathway

2.1. Perception and Initiation Module of the ABA Signaling Pathway

As the upstream core of ABA signaling, the classic "PYL-PP2C-SnRK2" module is the key to the initiation of ABA signaling [1]. There are also negative regulators in this module. For example, Homologous to ABI1 (HAB1) and receptor kinase FERONIA (FER) inhibit the activity of the ABA signaling pathway [2,3], jointly forming a bidirectional regulatory pattern of "positive regulation promoting drought resistance and negative regulation maintaining growth". In *Arabidopsis thaliana*, the PYL family is the initial node of ABA signal perception, located at the most upstream of the ABA signaling pathway, and responsible for accurately capturing intracellular ABA signals [1]. Similarly, the homologous genes of PYL1/4/6 in rice play a core role in the regulation of the balance between growth and stress resistance, and their functions are highly conserved compared with those of PYL1/4/6 in *Arabidopsis*. Rice PYL1/4/6 maintain ABA-mediated drought resistance by moderately

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inhibiting growth [4]. The PYL-ABA complex binds to and inhibits the activity of downstream PP2C phosphatase, thereby relieving the inhibitory effect of PP2C on SnRK2s kinases, releasing the activity of SnRK2s, and initiating the downstream drought resistance-related signaling pathway [1].

At the same time, there is a special negative regulatory branch in the upstream ABA signaling pathway to ensure the normal growth of plants under non-stress conditions. *Arabidopsis* HAB1, as a core negative regulator, is a group A PP2C phosphatase, which can directly bind to SnRK2s and catalyze their dephosphorylation to inhibit their activity and block signal transduction [5]. Notably, ABA-bound PYL receptors can compete with HAB1 for SnRK2s binding. Under stress conditions, the PYL-ABA complex preferentially binds to SnRK2s to relieve inhibition and initiate signaling; under non-stress conditions, HAB1 dominates to maintain the "off" state of the signal [6].

The intensity of ABA signaling depends on both the activation degree of PYL receptors and the activity of SnRK2s kinases. As core positive regulatory kinases, the functions of SnRK2s are highly conserved. The triple mutant of *Arabidopsis* subgroup III SnRK2.2/2.6/2.3 almost completely loses ABA responsiveness, confirming that these kinases are indispensable core factors of the signaling pathway [1,7].

In addition to the basic negative regulation, the *HAB1* gene produces two functionally opposite variants, HAB1.1 and HAB1.2, through alternative splicing regulated by RNA binding motif protein 25 (RBM25). Under non-stress conditions, HAB1.1 is dominant, which efficiently inhibits SnRK2.6 activity to ensure growth; under stress conditions, RBM25 induces the production of HAB1.2 with weak inhibitory ability, relieving the inhibition of SnRK2.6 to activate ABA signaling. An increase in the HAB1.2/HAB1.1 ratio enhances ABA signaling and drought resistance but inhibits growth, while the decrease of the ratio has the opposite effect, thus realizing dynamic environmental adaptation [8].

In addition to PP2C-type negative regulators, the receptor kinase FERONIA (FER) is also involved in the regulation of the balance of ABA signaling. Different from HAB1, FER significantly enhances the activity of ABA Insensitive 2 (ABI2) phosphatase by activating ROP11/ARAC10, indirectly inhibits the activity of SnRK2s, reduces the production of Reactive Oxygen Species (ROS), restores ROS homeostasis, and thus negatively regulates ABA signaling. Loss-of-function mutants of FER and its downstream Guanine nucleotide Exchange Factors (GEFs)-ROP11 module all exhibit an ABA-hypersensitive phenotype, indicating that this pathway can moderately inhibit ABA response under non-stress conditions to ensure normal plant growth [1,9].

In summary, the upstream perception and initiation module of the core ABA signaling realizes the dynamic balance of plant growth and drought resistance through the bidirectional regulatory mode of "positive regulatory axis (PYL-PP2C-SnRK2s) initiating drought resistance and negative regulatory axis (HAB1, FER) maintaining growth", which lays a foundation for subsequent signal transduction and downstream responses.

2.2. Core Hub for Balancing Growth Metabolism and Drought Resistance in the ABA Signaling Pathway

Both plant growth and development and stress adaptation consume a large amount of energy, and ABA signals need to be integrated into the cellular energy network to coordinate growth inhibition and resource conservation. The core of this energy regulation is mediated by a pair of antagonistic factors, SNF1-RELATED PROTEIN KINASE 1 catalytic subunit alpha 1 (SnRK1α1) and Target of Rapamycin (TOR), which regulate catabolic/anabolic metabolism respectively to accommodate stress adaptation and growth and development [1]. ABA regulates the nucleocytoplasmic shuttling of SnRK1α1 through SnRK2s to realize the rapid switch between growth and drought resistance [10]. This transcription-independent regulatory switch enables plants to quickly adjust the growth-drought resistance strategy [10]. At the same time, TOR and ABA pathway form a bidirectional negative regulatory loop: under non-stress conditions, TOR phosphorylates PYL to inhibit ABA signaling and

promote growth [11]; under stress conditions, activated SnRK2s phosphorylate Raptor to inhibit TOR, shifting resources to drought resistance defense [11].

In addition to the classic energy metabolism regulatory pathway, the epigenetic regulation of histone variant Histone H2A.Z (H2A.Z) is also involved in the hub function of ABA-mediated growth and metabolism. The core function of H2A.Z is to coordinate the balance between ABA and auxin in plants, thereby regulating the growth-drought resistance balance. H2A.Z inhibits the expression of SMALL AUXIN UP RNAs (*SAURs*), key downstream genes of the auxin signaling pathway, through selective deposition in the chromatin regions of *SAUR* genes, thereby mediating the ABA-induced inhibition of plant growth and enhancement of drought resistance [12]. As a bidirectional regulatory hub, H2A.Z can not only respond to ABA signals and promote stress adaptation, but also be removed from chromatin when auxin signaling is activated, relieving the inhibition of *SAURs* and restoring plant growth. It ensures that plants can accurately "apply the brake" to suspend growth to save energy under adversity, and quickly "release the brake" to restart growth after rehydration. At the same time, it improves the adaptability to repeated drought through epigenetic memory, which also provides a new regulatory insight into how plants balance growth and drought resistance.

Therefore, ABA realizes the growth-drought resistance balance strategy of "giving priority to drought resistance under stress and giving priority to growth under non-stress" by integrating the SnRK1-TOR energy metabolism hub. At the epigenetic level, the bidirectional regulatory mechanism of H2A.Z further improves this balance-regulatory network, enabling plants to flexibly adjust growth and drought resistance strategies according to environmental changes and realize the optimal allocation of energy and resources.

2.3. Transcriptional Regulation and Downstream Effector Responses of the ABA Signaling Pathway

Upon activation through phosphorylation of the upstream SnRK2s kinases, ABA signaling is further transmitted to the downstream transcriptional regulatory layer, and the transcriptional reprogramming of downstream genes is realized by regulating the activity of a series of transcription factors (TFs). Transcriptional regulation is the core step of ABA signal transduction from intracellular signaling to physiological responses, and also the key intermediate link connecting the energy core hub and downstream physiological effectors [1].

ABA-Insensitive 5 (ABI5) is a key bZIP transcription factor in the ABA signaling pathway. Its expression is up-regulated by ABA, and its core function is to prevent seedlings from entering vegetative growth prematurely under drought by maintaining the quiescent state of post-germination embryos, thereby improving the survival rate. Overexpression of *ABI5* enhances ABA sensitivity and drought tolerance, while the *abi5* mutant shows ABA insensitivity and decreased drought resistance, confirming that ABI5 plays a key regulatory role in the balance between growth arrest and drought resistance after germination [13]. In addition, ABI5 can activate downstream drought resistance genes such as *LEA* to further enhance drought resistance.

ABA-Responsive Element (AREB)/ABRE-binding factors (ABFs) are bZIP transcription factors, which are direct substrates of SnRK2s kinases and are highly phosphorylated in the ABA response. They are key molecules connecting transcriptional regulation and downstream effector responses. Under non-stress conditions, even if AREB/ABFs have basal expression, they cannot effectively activate the expression of downstream drought resistance genes containing ABRE elements, ensuring that plants give priority to growth [14]; under drought stress, SnRK2s are activated, and then phosphorylate specific serine sites of AREB/ABFs, thereby activating the expression of downstream drought resistance-related genes [15]. The activated AREB/ABFs mainly regulate the expression of two types of effector genes: one type consists of Late Embryogenesis Abundant (LEA) protein family genes, which can prevent cells from being damaged by dehydration [16]; the other type includes genes encoding biosynthetic enzymes for osmolytes such as proline [17] and trehalose [18], which can maintain cell turgor and enhance cell drought resistance. In addition, osmoprotectants and protective proteins

produced by downstream effectors can also affect the activity of transcription factors through feedback regulation, thereby optimizing the ABA signaling intensity [19], ensuring that plants can flexibly adjust the balance between growth and drought resistance.

The *Arabidopsis* AP2/ERF transcription factor TINY enhances drought tolerance by promoting ABA-mediated stomatal closure and binding to Dehydration-Responsive Element (DRE) motifs. At the same time, TINY negatively regulates growth by antagonizing BR signaling transcription factor BRI1-EMS-SUPPRESSOR 1 (BES1). BR prevents excessive accumulation of TINY by inhibiting the activity of BRASSINOSTEROID-INSENSITIVE 2 (BIN2), forming a dynamic balance between growth and stress resistance [20]. WRKY DNA-binding protein 46 (WRKY46), as a negative regulator of ABA signaling, acts upstream of ABI4. Its expression is inhibited by ABA to reinforce the inhibition of lateral root growth, but it can also positively regulate lateral root development by maintaining auxin homeostasis [21]. In rice, *Oryza sativa* NAC transcription factor 120 (*OsNAC120*) is located downstream of *Oryza sativa* OSMOTIC STRESS/ABA-ACTIVATED PROTEIN KINASE 9 (*OsSAPK9*). It promotes gibberellin (GA) synthesis and inhibits ABA synthesis under non-stress conditions, and interacts with OsSAPK9 to relieve the inhibition of ABA synthesis under stress to initiate drought resistance responses [22].

Therefore, downstream effector genes realize the growth-drought resistance balance of plants under drought stress through the strategy of positively regulating ABA signaling to promote drought resistance and negatively regulating ABA signaling to maintain growth, ensuring an optimal adaptive strategy that prioritizes survival while accommodating growth in fluctuating environments.

2.4. Crosstalk Between ABA and Other Phytohormones

ABA does not independently regulate the plant growth-drought resistance balance, but forms a precise crosstalk network with a variety of growth-promoting phytohormones through multiple key nodes [23]. Among them, Regulatory-Associated Protein of TOR 1B (RAPTOR1B) is the core node of crosstalk between ABA, auxin and BR signals, which can integrate hormone and metabolic signals to realize the dynamic switch of "promoting growth under non-stress and initiating drought resistance under stress" [11,24,25]; BIN2, a key GSK3 (glycogen synthase kinase 3) kinase mediating the crosstalk between BR and ABA, can perform bidirectional antagonistic regulation to balance growth and drought resistance responses [26,27]; ABI5 is the core of crosstalk between ABA and GA, and the module formed by ABI5 and DELLA protein determines the resource allocation strategy of seed germination and plant growth by regulating GA-ABA homeostasis [23,28-32]; Tryptophan Synthase β Subunit 1 (TSB1) mediates the crosstalk between ABA and auxin at the metabolic level, and coordinates the balance between growth and drought resistance by regulating tryptophan (Trp) and ABA homeostasis [33].

In summary, these key crosstalk nodes form a multi-level regulatory network that balances growth and drought resistance by integrating different hormone signals. Through the crosstalk with different growth-promoting phytohormones, ABA realizes the dynamic switch between drought resistance defense under stress and growth and development under non-stress conditions, ensuring that plants can maximize adaptability and achieve survival and reproduction in a complex environment.

3. Conclusion and Outlook

As the core phytohormone of plants in response to drought stress, ABA realizes the precise regulation of plant growth-drought resistance balance through its complex signaling pathway network. The bidirectional regulatory pattern of the upstream perception and initiation module, the decision of "promoting drought resistance under stress and promoting growth under non-stress" of the core hub of growth and metabolism, as well as the precise transmission and execution of signals in transcriptional regulation and downstream effector responses, together constitute the multi-level signaling pathway network of ABA. The crosstalk between ABA and auxin, BR, GA and other

phytohormones further improves the balance regulatory network of growth and drought resistance, enabling plants to flexibly adjust physiological strategies according to environmental changes.

At present, there have been many studies on the molecular mechanism by which ABA regulates the balance between plant growth and drought resistance, but there are still some problems to be further explored, for example, more key nodes of crosstalk between ABA and other phytohormones and their regulatory logic. In addition, with the increasingly urgent demand for crop stress resistance breeding, how to apply the regulatory mechanism of the ABA signaling pathway to crop breeding to cultivate crop varieties with both high yield and drought resistance is also an important research direction in the future.

Future research can combine molecular biology, bioinformatics, gene editing and other technologies to further analyze the interaction network of key genes and proteins in the ABA signaling pathway, identify new regulatory nodes, and clarify the synergistic mechanisms between different regulatory modules. At the same time, it is essential to strengthen the application of ABA regulatory mechanisms in crop breeding, precisely regulate the expression of genes related to the ABA signaling pathway through gene editing, transgenesis and other means, realize the synergistic improvement of crop growth and drought resistance, and provide theoretical and technical support for coping with drought stress caused by global climate change and ensuring food security.

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