AMERICAN JOURNAL OF BIOLOGY AND NATURAL SCIENCES
https://biojournals.us/index.php/AJBNS
ISSN: 2997-7185

Article

Plant Stress Memory and Priming: Mechanisms of Acquired Resilience

Azhaar Hussein Ali Alshahwani*[1], Rasha Mohammed Salih[2], Sawsan J. Al-Allaf[2]

1. Department of Biology, College of Science, University of Mosul, Mosul, Iraq
2. Department of Forensic Evidence, College of Science, University of Mosul, Mosul, Iraq

*Correspondence: azhsbio47@uomosul.edu.iq[1]

Citation: Alshahwani, A. H. A, Salih, R. M., & Al-Allaf, S. J. Plant Stress Memory and Priming: Mechanisms of Acquired Resilience. American Journal of Biology and Natural Sciences 2026, 3(11), 28-40.

Received: 10th Aug 2026
Revised: 11th Sep 2026
Accepted: 20th Oct 2026
Published: 05th Nov 2026

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Abstract: Plants can retain the physiological and molecular effects of prior stress exposure, thereby modulating their responses to subsequent environmental challenges. Stress priming can lead to a state of modified responsiveness, whereas stress memory refers to the persistence of stress-associated effects that influence subsequent responses. These phenomena have a molecular basis involving interconnected processes at the levels of transcription, epigenetic modifications, hormones, ribonucleic acid (RNA) and metabolism. This study reviews the contributions of transcription factors, chromatin modifications, DNA methylation, small RNAs, phytohormone signalling, reactive oxygen species and metabolic modifications to the formation and expression of plant stress memory. Particular attention is paid to the persistence of stress-related changes, the distinction between molecular correlations and causal mechanisms, and the evidence required to distinguish between intragenerational memory, intergenerational effects and transgenerational inheritance. The review also considers the agricultural importance of seed priming, clonal methods for crop improvement.

Keywords: Abiotic stress; stress memory; priming; epigenetics; transcription factors; phytohormones; reactive oxygen species; metabolism; crop resilience; epigenome editing.

Introduction

Climate change is increasing the frequency, duration and unpredictability of environmental constraints such as drought, heat, salinity and cold episodes that threaten crop productivity. As they cannot escape adverse conditions, plants rely on coordinated sensing, signalling, transcriptional, metabolic and physiological responses to maintain cellular homeostasis and survival [1, 2]. There is increasing evidence that these responses are influenced not only by the current environment, but also by the plant's previous stress history [1, 3, 4]. Stress memory is the ability of a plant to modify its response to subsequent stress based on information acquired during an earlier exposure. A related phenomenon is stress priming, which occurs when an initial mild or transient stimulus enhances tolerance to a subsequent, often more severe, stress [3, 4]. Priming can result from abiotic stresses or from chemical, hormonal, microbial or nutritional treatments. Importantly, priming does not necessarily imply the constant activation of defence pathways. Instead, it may establish a physiological or molecular state in which stress-responsive systems can be activated more rapidly or efficiently when stress recurs.

Stress memory can occur within the lifetime of an individual plant, and under certain conditions it can influence subsequent generations. These categories should be distinguished carefully.

Somatic, or within-generation, memory occurs At the molecular level, stress memory involves interconnected changes in transcription factor activity, chromatin accessibility, DNA methylation, histone modifications, small RNA expression, alternative splicing, hormone signalling, reactive oxygen species (ROS) metabolism and primary and secondary metabolism [3, 5–8]. These processes interact dynamically: stress-induced signalling can modify chromatin; chromatin state can alter transcriptional responsiveness; and metabolites and hormones can feed back into both processes. Therefore, stress memory is better understood as a dynamic regulatory network than as a single molecular mark, as shown in Figure 1. Recent reviews have also emphasised the importance of distinguishing between transient acclimation and true memory, as well as integrating molecular mechanisms with physiological performance.

Understanding this network is becoming increasingly important for crop improvement. While conventional breeding primarily exploits stable genetic variation, stress priming and epigenetic approaches offer opportunities to manipulate environmentally responsive phenotypes without necessarily altering the DNA sequence.

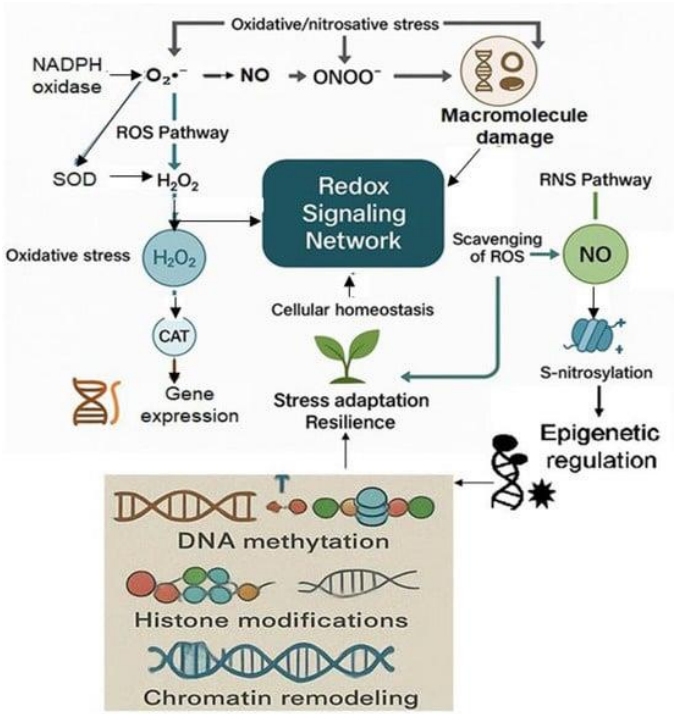


Figure 1. Schematic representation of the unified plant stress memory network [9].

Transcriptional Mechanisms of Stress Memory

Heat Shock Factors and Transcriptional Memory

Heat stress is one of the most well-understood experimental systems for studying plant stress memory. Exposure to high temperatures activates heat shock factors (HSFs), which control the expression of heat shock proteins (HSPs) and other genes that respond to stress. HSFA2 and HSFA3, in particular, play important roles in heat-stress memory in Arabidopsis thaliana [5, 10–12]

Following an initial exposure to heat, some stress-responsive genes may retain an altered state of transcriptional activity or competence during the recovery period. This state does not require continuous high-level transcription, but rather enables selected genes to maintain expression for a limited time or respond more rapidly and strongly when heat stress recurs. Experimental studies have demonstrated that HSFA2 and HSFA3 interact and contribute to the regulation of transcriptional memory after heat exposure [5, 11, 12].

The persistence of these responses is associated with interactions between transcription factors and chromatin-regulatory mechanisms. In particular, histone modifications at selected stress-responsive loci may help to maintain transcriptional competence after the initial stress has ended [5, 12]. However, the contribution of individual transcription factors and chromatin-associated changes

can vary between genes and experimental conditions. Therefore, the detection of a transcription factor or histone mark alone is insufficient to establish whether it causes a persistent physiological benefit.

Heat-stress memory must also be balanced against the need to restore normal growth and development. Prolonged activation of stress-associated pathways can have negative developmental consequences, whereas effective resetting enables plants to recover when environmental conditions improve [13]. Accordingly, heat-stress memory is best understood as a regulated state of altered responsiveness rather than the permanent activation of protective genes. Further investigation of the mechanisms governing memory establishment, maintenance and resetting will help to clarify how plants balance stress resilience with growth and productivity [10, 13].

Other Transcription Factor Families

Other transcription factors besides heat shock factors contribute to the regulation of plant responses to environmental stress. Members of the WRKY, MYB, NAC and bZIP families are involved in transcriptional networks associated with drought, salinity, oxidative stress and temperature fluctuations. These proteins regulate genes that are involved in antioxidant defence, osmotic adjustment, metabolism, and cellular protection. Their activity is influenced by signalling pathways involving calcium ions (Ca^{2+}), reactive oxygen species (ROS), protein phosphorylation and phytohormones [1, 3, 14].

NAC transcription factors are particularly relevant to drought-stress responses. In maize, the ZmNAC111 transcription factor has been investigated as a regulator associated with drought resistance. Experimental work has shown that co-expressing it with ZmVPP1 improves several drought-related physiological traits, including photosynthetic performance and antioxidant activity. Benefits have also been reported for grain yield under water-deficit conditions [15]. This evidence illustrates the potential contribution of transcriptional regulation to stress adaptation. However, a role in drought tolerance does not automatically constitute

Cold Stress and Transcriptional–Chromatin Memory

Exposure to cold temperatures induces substantial transcriptional reprogramming in plants, contributing to cold acclimation and increasing their capacity to withstand subsequent freezing temperatures. While the transcription factors involved in cold-responsive pathways have been extensively studied, chromatin regulation may also influence the activation and subsequent regulation of stress-responsive genes [16]

Among the histone modifications associated with cold responses, trimethylation of histone H3 at lysine 4 (H3K4me3) is generally associated with transcriptional activity, whereas trimethylation at lysine 27 (H3K27me3) is commonly associated with Polycomb-mediated repression. Changes in these marks may influence chroatin organisation and gene responsiveness during cold exposure. However, the effects of these changes are locus-dependent, and the relationship between histone modification and transcription is not always direct [7, 16–18].

Alternative Splicing and Transcriptomic Reprogramming

Stress memory is not limited to transcriptional abundance. Alternative RNA processing can also contribute to stress adaptation. For example, ABA-induced alternative splicing has been shown to contribute to the reprogramming of transcripts associated with drought tolerance in barley [19].

Alternative splicing increases the variety of functions of stress-responsive transcripts, enabling plants to rapidly modify protein composition without requiring entirely new transcriptional programmes. Therefore, RNA-based regulation, including alternative splicing, microRNAs, small interfering RNAs and long non-coding RNAs, represents an important additional layer of stress memory [8].

Epigenetic Mechanisms

DNA Methylation

DNA methylation is one of the major epigenetic mechanisms involved in plant stress responses. Cytosine methylation predominantly occurs in CG, CHG and CHH contexts, and is regulated by methyltransferases, demethylases and the RNA-directed DNA methylation (RdDM) pathway [6, 20–22].

Stress can induce changes in DNA methylation at specific loci, which can potentially alter transcriptional activity and genome stability. Changes in methylation patterns have been associated with drought, salinity, temperature extremes and heavy-metal exposure [22–25].

Histone Modifications

Histone modifications provide an alternative mechanism for altering chromatin accessibility without altering the DNA sequence. Stress-responsive chromatin states can involve changes in H3K4me3, H3K27me3, H3K36me3 and histone acetylation, among other modifications [7, 17, 18].

H3K4me3 is often linked to transcriptional activation or competence, while H3K27me3 is associated with gene repression. The dynamic redistribution of these modifications can establish chromatin states that influence the speed and magnitude of subsequent transcriptional responses [7, 16–18].

Small RNAs and RNA-Directed DNA Methylation

Small RNAs contribute to stress memory by regulating transcription post-transcriptionally and by epigenetic silencing. MicroRNAs can regulate stress-responsive transcripts, interfering RNAs can guide DNA methylation via the RdDM pathway [8, 26].

The RNA layer is important because it connects transcriptional regulation with chromatin modification. Therefore, stress-induced changes in small RNA populations can affect both immediate gene expression and longer-lasting epigenetic states. However, the persistence of these changes and their contribution to stable memory depend heavily on the type of stress, the tissue, the developmental stage and the genetic background [27].

Transgenerational Epigenetic Inheritance

One of the most intriguing questions is whether stress memory can persist across generations. Some forms of stress exposure have been shown to influence the phenotypes of offspring, including heat tolerance and responses to environmental stress [28, 29]. For instance, heat priming in wheat has been linked to enhanced physiological performance in offspring exposed to high temperatures [28].

However, the terminology surrounding inheritance must be used cautiously. Maternal effects, seed provisioning, persistent metabolites, small RNAs and changes in the reproductive environment can all influence offspring phenotypes without constituting stable transgenerational epigenetic inheritance [29–31].

Thus, evidence for true transgenerational inheritance requires rigorous experimental designs involving multiple generations and appropriate controls. Recent reviews emphasise that heritable epigenetic variation can contribute to plant evolution and breeding, but its stability and predictability remain context-dependent [25, 30].

Epigenome Editing

Advances in CRISPR-based technologies have created opportunities to manipulate epigenetic states directly. Catalytically inactive Cas proteins (dCas) can be coupled with transcriptional repressors or activators, as well as with DNA methyltransferases, demethylases or chromatin-modifying domains, in order to alter gene expression without introducing conventional double-strand DNA breaks [32–34].

Such approaches could potentially generate beneficial epialleles at stress-responsive loci. However, achieving stable and predictable inheritance remains a major challenge. The effectiveness of epigenome editing depends on the target locus, chromatin context, cell type, developmental stage and maintenance of the induced epigenetic state [32–34]. Therefore, current evidence supports epigenome editing as a promising research and breeding technology, rather than a mature replacement for conventional genetic improvement.

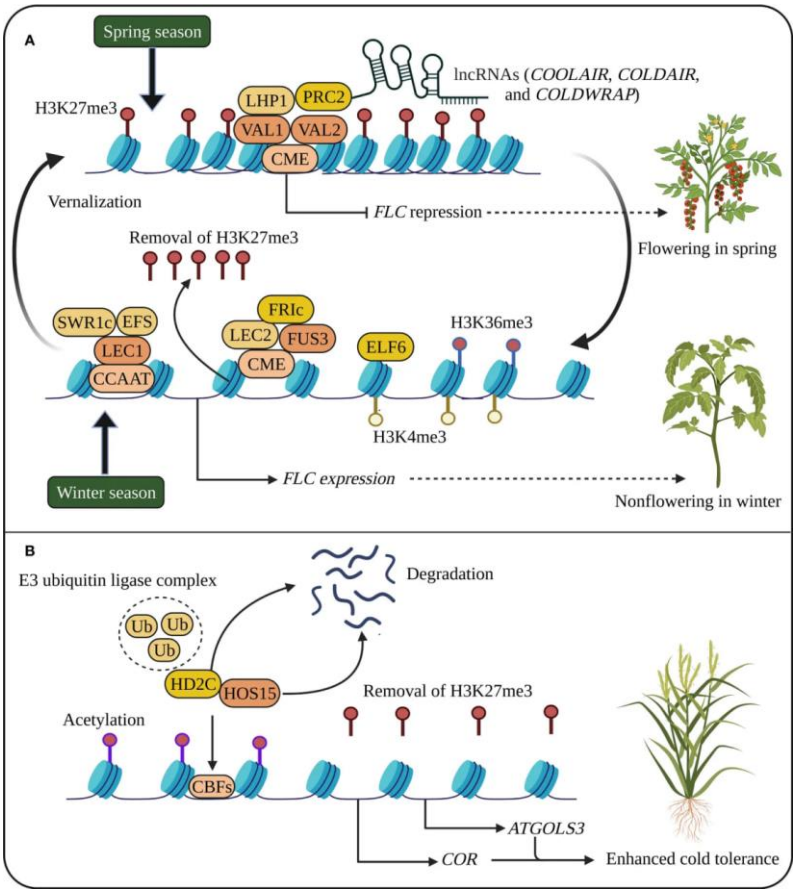


Figure 2. Molecular architecture of epigenetic stress memory [35].

Hormonal Regulation of Stress Memory

Abscisic Acid

ABA is a key regulator of how plants respond to drought, salinity and other abiotic stresses. The canonical ABA pathway involves PYR/PYL receptors, PP2C phosphatases, SnRK2 kinases and downstream targets of transcription and ion transport [36]

ABA signalling has a wider range of effects than merely inducing immediate stress responses. It also interacts with chromatin regulators, transcription factors, reactive oxygen species (ROS), and other hormones, thereby contributing to the establishment and expression of stress memory [3, 36, 37]. For instance, ABA can influence Polycomb-mediated H3K27me3 regulation via VIL1, illustrating the direct link between hormonal signalling and chromatin state [37].

ABA signalling is also closely linked to cellular redox balance. The regulation of SnRK2 proteins can affect NADPH maintenance during prolonged ABA signalling, illustrating the integration of hormone signalling with metabolic and redox processes [38].

ABA also interacts with auxin transport. ABA-dependent regulation of PIN2 trafficking can reduce primary root elongation, demonstrating how stress signalling modifies developmental programmes to balance survival and growth [39].

Jasmonic Acid and Salicylic Acid

JA and SA are key regulators of plant defence and can interact with responses to abiotic stress. Their effects depend strongly on concentration, tissue type and stress type, as well as interactions with other hormones [40, 41]

The cross-talk between ABA, JA, SA, ethylene, auxin and other hormones enables plants to prioritise different physiological strategies. Rather than functioning as independent linear pathways, these hormones form interconnected networks, whereby one signal can enhance or suppress another [40]

Such cross-talk is particularly important for priming, as a primed

Ethylene, Auxin, Gibberellins, and Cytokinins

Ethylene can modify ABA-dependent stomatal regulation, while auxin and gibberellins influence growth responses in response to stress [39, 40, 42]. Similarly, cytokines participate in balancing growth, senescence and stress responses.

This hormonal integration is essential because stress tolerance often necessitates a temporary reduction in growth. However, persistent activation of stress hormones can impose substantial developmental costs. Therefore, stress memory must operate within a regulatory system capable of returning plants to a

Nitric Oxide

Nitric oxide has emerged as an important signaling molecule linking stress perception, redox regulation, hormonal pathways, and priming [43, 44]. NO can modify proteins through redox-dependent post-translational mechanisms and interact with ROS, ABA, and other signaling systems.

Recent work has proposed a role for NO in thermomemory, suggesting that NO-associated signaling can contribute to the persistence and recall of heat responses [45]. More broadly, NO may function as a signaling hub connecting the initial stress signal to transcriptional and metabolic changes.

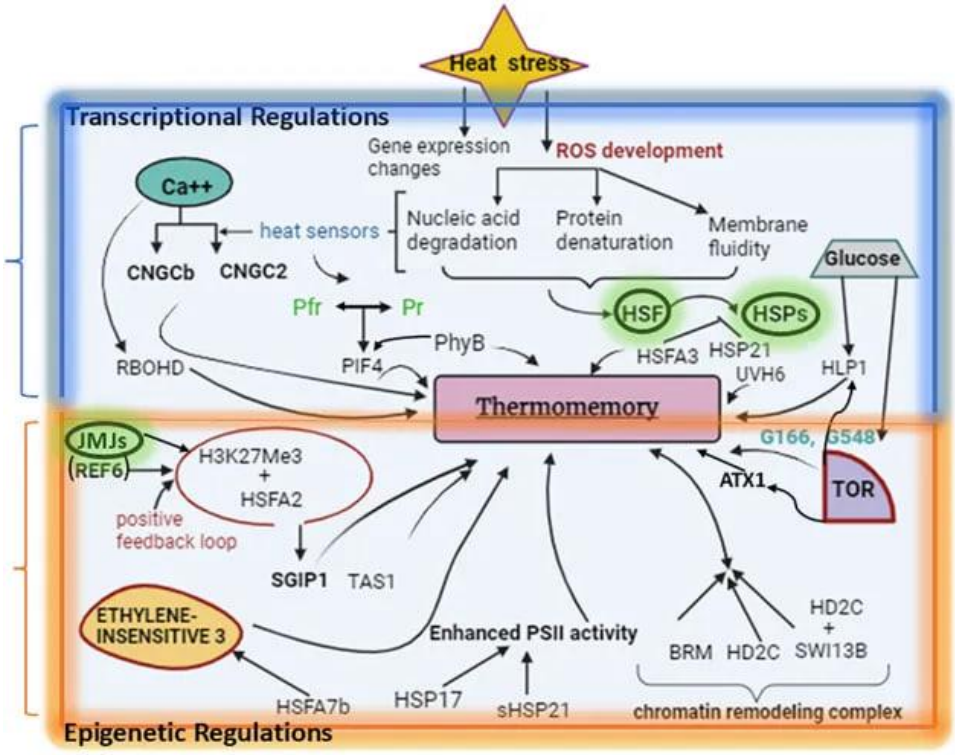


Figure 3. Nitric oxide (NO)-mediated regulatory loop governing heat thermomemory. NO acts as a key signaling hub by stimulating HSFA2 activation, which upregulates the H3K27me3 demethylase REF6. REF6 derepresses *HSFA2* through promoter demethylation, creating a self-sustaining feedback loop integrated with ABA and SA/JA stress signaling [46].

Metabolic Adaptations and Stress Memory

Osmoprotectants

Exposure to stress induces metabolic changes that stabilize proteins, membranes and the cellular osmotic balance. Proline is one of the most well-understood osmoprotectants and plays a role in osmotic adjustment, redox regulation and stress signalling [47, 48]

Proline metabolism is dynamic rather than simply cumulative. Its synthesis and degradation contribute to the redistribution of carbon and nitrogen and can influence the balance of reactive oxygen species (ROS). Consequently, the metabolic state established during initial stress exposure can influence the response to subsequent stress [47–50]

Sugars and polyols also contribute to osmoprotection and signalling. Soluble sugars can stabilise cellular structures and act as signalling molecules, while

Secondary Metabolites

Stress can stimulate the production of phenolic compounds, flavonoids, terpenoids, alkaloids, glucosinolates and other secondary metabolites [51, 52]. These compounds can act as antioxidants, signalling molecules or antimicrobial defences.

Their role in memory likely arises from the interaction between persistent metabolic states and transcriptional regulation. Transcription factors such as WRKY, MYB, NAC and bZIP proteins regulate many biosynthetic pathways, thereby connecting transcriptional memory with metabolic adjustment [13, 51].

Reactive Oxygen Species as Signaling Molecules

ROS were traditionally viewed as primarily damaging by-products of stress. However, it is now clear that they also function as dynamic signalling molecules**.**

Moderate ROS accumulation can activate antioxidant defences, protein phosphorylation, transcription factors and hormone pathways. In contrast, excessive ROS causes oxidative damage. Therefore, stress memory requires the precise regulation of ROS rather than their complete elimination [47, 53].

Redox regulation can also influence chromatin remodelling. Changes in the cellular redox state can affect chromatin-associated proteins and modify transcriptional accessibility, providing a mechanistic link between metabolism and epigenetics [53].

Autophagy and Cellular Recycling

Autophagy plays a role in stress adaptation by recycling damaged proteins and organelles, thereby maintaining cellular homeostasis [54]. As recurrent stress requires the rapid redistribution of cellular resources, autophagy may support the metabolic flexibility necessary for effective stress responses.

However, its role in stress memory is better understood as part of the broader process of cellular resetting and resource management, rather than as an independent memory mechanism. [55]

Maize Drought Stress Memory

Maize is an important model for studying drought memory due to its large, transposable element (TE)-rich genome and C4 photosynthetic system, which provide multiple levels at which stress-associated regulation can occur. Recent research suggests that drought priming in maize may involve histone modifications, DNA methylation, transposable element regulation, proline metabolism, carbon–nitrogen redistribution and photosynthetic adjustments [15].

Metabolomics

provides an opportunity to identify biochemical signatures associated with stress memory. Integrating metabolomics with transcriptomics, epigenomics, proteomics and phenotyping reveals the relationships between molecular changes and whole-plant performance [49, 56, 57].

Such an approach is particularly valuable because a single molecular marker rarely predicts stress tolerance across different environments. Instead, metabolic profiles may provide composite indicators of physiological state and stress history. [58].

Interconnections Between Stress-Memory Mechanisms

Transcriptional–Epigenetic Integration

Transcription factors and epigenetic regulators form reciprocal regulatory loops. For example, a stress-responsive transcription factor can recruit chromatin modifiers, and the chromatin state determines the accessibility of transcription-factor binding sites [5–7, 17, 18, 59]

This feedback mechanism enables stress information to persist beyond the initial signal while retaining the capacity for rapid reactivation.

Hormonal–Epigenetic Crosstalk

Hormonal signals can modify chromatin, and vice versa. ABA provides a clear example of this interaction, as ABA signalling interacts with Polycomb-associated regulation and other chromatin processes [36, 37]

This interaction means that hormonal memory and epigenetic memory are inextricably linked. [60]

Hormonal–Metabolic Integration

Hormones regulate metabolic pathways, while metabolic status influences hormone signalling. For example, ABA regulates stomatal behaviour and carbon assimilation, while cellular energy and redox status can influence the strength and duration of stress signalling.

Similarly, jasmonic acid (JA) and salicylic acid (SA) influence secondary metabolism and the accumulation of defence-related metabolites [40, 41].

ROS as a Central Hub

ROS occupy a particularly important position because they connect environmental sensing, hormone signalling, transcription, metabolism and chromatin regulation [1, 45, 53].

The biological outcome depends on ROS dynamics rather than their absolute concentration. The spatial localisation, amplitude, duration and cellular compartment all influence whether ROS primarily function as signals or cause oxidative damage [61].

The Integrated Stress-Memory Network

Taken together, stress memory can be conceptualised as a hierarchical network.

Stress perception → Ca^{2+}/ROS/NO and hormonal signalling → transcription factor activation → chromatin and RNA regulation → metabolic adjustment → physiological acclimatisation → memory consolidation → rapid response to recurrent stress.

During recovery, resetting mechanisms reduce unnecessary activation while retaining selected components of molecular preparedness [3, 13]. Maintaining this balance between memory and resetting is fundamental to preserving both resilience and developmental plasticity.

7. Applications in Agriculture

7.1 Seed Priming

Seed priming is one of the most practical applications of stress-memory biology. Hydropriming, osmopriming, hormonal priming, chemical priming, chitosan treatment, microbial bio-priming and nanopriming can all improve germination and early stress tolerance [62–65].

The main benefit is that it prepares seedlings for exposure to severe environmental conditions. However, priming responses depend strongly on species, genotype, dose, treatment duration, developmental stage.

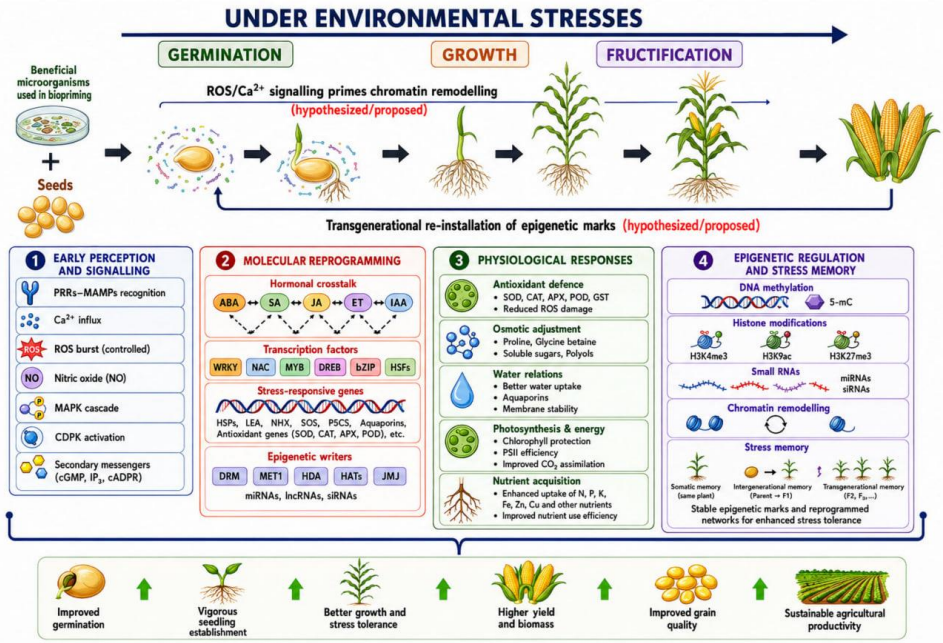


Figure 4. Translation of stress memory and priming mechanisms into climate-resilient agriculture. Schematic summary of practical approaches including seed priming technologies (nano-, bio-, and chemical priming), epigenetic selection models (ERM/GRM integration and epi-RILs), and locus-specific CRISPR-dCas9 epigenome editing for enhanced abiotic stress resilience [66].

Genomic Selection and Epigenetic Breeding

Stress-memory traits could be used to complement conventional genomic selection. Epigenetic variation can provide an additional source of phenotypic variation, particularly in environments where responsiveness substantially contributes to crop performance [25, 67].

Epigenetic relationship matrices, epigenetically divergent recombinant populations and stress-responsive epialleles could be integrated into genomic prediction models. However, as epigenetic states are less stable than DNA sequence polymorphisms, validation across environments and generations is essential. [68].

CRISPR-Based Epigenome Editing

CRISPR/dCas systems offer the chance to manipulate transcription or chromatin at specific locations without the need for conventional sequence editing [32–34]. Targeting susceptibility genes or stress-responsive regulatory regions could enhance stress resilience while minimising undesirable genome-wide effects.

However, the major challenges include target selection, tissue specificity, editing efficiency, epigenetic stability, heritability and regulatory acceptance [32–34, 69, 70].

Multi-Omics and High-Throughput Phenotyping

The complexity of stress memory makes a multi-omics approach particularly attractive. Integrating transcriptomics, epigenomics, metabolomics, proteomics and high-throughput phenotyping can help to identify molecular signatures that are associated with resilience [56, 57].

This approach can also help to distinguish causal mechanisms from stress-associated correlations. In terms of breeding, the ultimate objective should be to identify biomarkers that predict performance under realistic environmental conditions, rather than simply detecting molecular differences under controlled conditions [60].

Challenges and Future Directions

There are several challenges that currently limit the deployment of agriculture.

Firstly, memory is strongly context-dependent. For example, a priming treatment that is beneficial under one combination of temperature, water availability and genotype may have little effect, or even negative consequences, under another.

Secondly, there is a fundamental trade-off between growth and defence. The continuous activation of defence programmes can reduce biomass accumulation, reproduction, or yield [71].

Thirdly, epigenetic states can be unstable. A molecular mark detected after stress may disappear during recovery or be reset during reproduction.

Fourthly, many studies remain laboratory-based. Field conditions expose plants to multiple simultaneous and sequential stresses, so validation in realistic environments is essential.

Finally, the deployment of gene-edited and epigenetically modified crops may be influenced by regulatory frameworks and public acceptance [70].

Therefore, future research should prioritise longitudinal experiments, multi-generational designs where inheritance is claimed, field-scale priming trials, causal manipulation of candidate loci, and integration of molecular phenotyping with yield and quality traits.

Conclusion

Plant stress memory is a dynamic form of acquired resilience whereby previous environmental exposure alters the response to subsequent stress. Rather than being encoded by a single mechanism, stress memory emerges from the coordinated interaction of transcription factors, chromatin modifications, DNA methylation, RNA-mediated regulation, phytohormones, reactive oxygen species (ROS) and metabolism.

Transcriptional regulation provides rapid control of stress-responsive genes, while chromatin and epigenetic mechanisms can maintain selected loci in a state of altered responsiveness. Hormonal signalling, particularly ABA-centred networks, integrates environmental perception with transcriptional and metabolic responses. ROS and NO act as important signalling intermediates, while osmolytes, secondary metabolites and cellular recycling pathways provide biochemical and physiological support.

It is essential to distinguish between within-generation memory, intergenerational effects, and true transgenerational inheritance. While some experimental systems demonstrate inheritance-associated phenotypes, the persistence and causality of epigenetic states are highly context-dependent. Consequently, future research should clearly distinguish between molecular correlation, physiological memory, and demonstrated heritable mechanisms.

From an agricultural perspective, stress priming provides a quick way to exploit these mechanisms. Meanwhile, genomic selection, epigenetic breeding, CRISPR-based epigenome editing and integrated multi-omics offer longer-term opportunities. Rather than searching for a single 'memory gene' or epigenetic mark, the most promising approach is to understand and manipulate the integrated stress-memory network.

A major research priority is determining which molecular changes are causal and which are merely associated with stress exposure and persist sufficiently to influence agronomic performance. Combining controlled mechanistic experiments with field-scale validation is essential for converting the rapidly expanding knowledge of plant stress memory into reliable, climate-resilient technologies.

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