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Review

Epigenetic Regulation for Heat Stress Adaptation in Plants: New Horizons for Crop Improvement under Climate Change

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Abstract: Global warming poses a significant threat to plant ecosystems and agricultural productivity, primarily through heat stress (HS), which disrupts photosynthesis, respiration, and overall plant metabolism. Epigenetic modifications, including DNA methylation, histone modifications, and RNA modifications, enable plants to dynamically and heritably adjust gene expression in response to environmental stressors. These mechanisms not only help plants survive immediate stress but also confer stress memory, enhancing their resilience to future HS events. This review explores the mechanisms underlying plant thermotolerance, emphasizing the critical role of epigenetic regulation in adapting to HS. It also highlights how DNA methylation modulates stress-responsive genes, histone modifications facilitate transcriptional memory, and RNA modifications influence mRNA stability and translation. Recent advancements in genome editing technologies, such as CRISPR-Cas9, have enabled precise modifications of epigenetic traits, offering new avenues for breeding climate-resilient crops. The integration of these modern tools with traditional breeding methods holds significant promise for developing crops with enhanced thermotolerance. Despite the potential, challenges such as the stability and heritability of epigenetic marks and the complex interplay between different epigenetic modifications need to be addressed. Future research should focus on elucidating these interactions and identifying reliable epigenetic markers for selection. By leveraging the insights gained from epigenetic studies, we can develop innovative breeding strategies to improve crop resilience and ensure sustainable agricultural productivity in the face of global warming. This review underscores the importance of epigenetic regulation in plant adaptation to heat stress and its potential to revolutionize crop breeding, offering a pathway to secure food production and sustainability under changing climatic conditions.

Keywords: global warming; heat stress; thermotolerance; epigenetic heat memory

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1. Global Warming and Its Impact on Plants

Global warming, driven primarily by increased greenhouse gas emissions, has resulted in significant climate changes, including more frequent and extreme high temperatures. Plants, being sessile organisms, are directly influenced by these changes, making them vulnerable to the adverse effects of global warming, and these changes have had devastating effects on crop production [1,2]. Climate change is being increasingly recognized as one of the greatest challenges facing humanity and all other life on Earth. The global average temperature has already risen by approximately 0.74 °C and is projected to increase [3]. Climate models predict that global surface temperatures could rise by as much as 5.5 °C by the end of the century, depending on efforts to mitigate CO_2 and other harmful emissions [4]. This rise in temperature is largely due to urbanization, fossil fuel burning, and high carbon dioxide emissions, which trap heat in the atmosphere and cause global warming. The consequences of climate change include shifts in seasonal patterns, extreme weather events, and increased temperatures, all of which pose significant threats to agricultural productivity and food security [5,6]. This increase will likely be accompanied by more frequent extreme weather events, such as floods and droughts, further impacting crop productivity. As temperatures increase, the geographic distribution of plant species is expected to change, impacting growth in relation to the day and night temperatures, light intensity, and biotic stress, among other factors. It is estimated that a 1 °C rise in temperature could decrease the yields of maize, rice, wheat, and soybeans by between 3.1% and 7.4% [7]. The intensity of temperature-induced stress is further intensified when environmental stressors, such as heat and drought, are combined [8]. For instance, during the intense 2012 heatwave in the U.S. corn belt, coupled with low rainfall, maize and soybean yields fell by around 22% and 17%, respectively [9]. Similarly, extreme temperatures and decreased precipitation in Northern and Eastern Europe in 2018 led to significant reductions in wheat and barley yields [10].

One of the most significant effects of increased temperatures is heat stress (HS), an abiotic factor that impacts plant growth, development, and yield. HS diminishes plant productivity by influencing their morphological, physiological, phenological, biochemical, and molecular responses. HS adversely affects the photosynthetic activity, respiration, protein synthesis, membrane stability, and hormone regulation in plants and reduces the water content, thereby impairing cell division and growth [11]. These disruptions can lead to poor seed germination, stunted growth, reduced fruit set, and plant death [12]. Photosynthesis, the process by which plants convert light energy into chemical energy, is highly sensitive to temperature fluctuations. High temperatures can disrupt photosynthetic enzymes, reduce the chlorophyll content, and damage the photosynthetic apparatus, thereby decreasing the plant's ability to synthesize food efficiently [13]. Additionally, high temperatures can cause oxidative stress, leading to the production of reactive oxygen species (ROS) that damage cellular structures and macromolecules [14]. Respiration, another critical physiological process, is also affected by heat stress. Elevated temperatures increase the rate of respiration, leading to higher carbohydrate consumption and decreased energy availability for growth and development [15]. This imbalance between photosynthesis and respiration under heat stress conditions can severely impact plant growth and yield.

Moreover, heat stress affects various aspects of plant metabolism. High temperatures can denature proteins and inhibit their synthesis, which is essential for maintaining cellular functions and stress responses [16]. Membrane stability is compromised under heat stress due to increased fluidity and permeability, leading to ion leakage and loss of cellular homeostasis [17]. Hormonal changes induced by heat stress can disrupt growth and development, affecting processes such as flowering, fruit set, and seed development [18]. Understanding the molecular mechanisms involved in the heat stress response (HSR) of plants is essential for developing crops with improved thermotolerance [19]. Research aimed at breeding high-yielding crop varieties can sometimes compromise their resistance to both biotic and abiotic stress. Under heat stress conditions, these crops may struggle to survive, leading to decreased crop production and posing a threat to food security [12].

Understanding plant responses to heat stress and developing strategies for thermotolerance are crucial for ensuring food security and ecosystem stability. As global temperatures continue to rise, it becomes imperative to enhance the thermotolerance of plants through various approaches, including breeding, genetic engineering, and biotechnological interventions. This review aims to explore the mechanisms underlying plant thermotolerance, with a particular focus on the role of epigenetic regulation in adapting to heat stress. By elucidating these mechanisms, we can develop innovative strategies to mitigate the adverse effects of global warming on plant productivity and sustainability.

2. Thermotolerance in Plants

Thermotolerance refers to the ability of plants to endure high temperatures, which is essential for survival and productivity amidst global warming. This trait is intricate, involving various physiological, biochemical, and molecular mechanisms. Despite the harmful effects of HS, most plants can adapt to moderate heat. Depending on the HS temperature regimes and the developmental stages of the plants, there are different types of thermotolerance that plants can achieve through inherent resistance and acquired thermotolerance (AT) [20,21]. Three categories of thermotolerance are typically observed in *Arabidopsis*: basal thermotolerance to HS at temperatures ranging from 40 to 45 °C, AT following a short period of nonlethal temperature or priming HS, and thermotolerance to prolonged exposure to moderately high temperatures between 30 and 38 °C [20].

Plant thermotolerance is divided into basal and acquired thermotolerance. Basal thermotolerance refers to the inherent ability of plants to survive high temperatures, while acquired thermotolerance refers to the ability to withstand lethal high temperatures after a period of acclimatization and long-term adaptation through evolutionary process spanning multiple generations. At the molecular level, key responses to heat stress include the induction of heat shock transcription factors (HSFs). These factors target the gene expression of heat shock proteins (HSPs) and ROS-scavenging enzymes, which play crucial roles in protecting plants from heat-induced damage [19,22]. Basal thermotolerance represents the plant's innate ability to cope with moderate heat stress without prior exposure. This form of thermotolerance is essential for plants growing in environments with naturally fluctuating temperatures. It involves constitutive mechanisms that include the maintenance of cellular homeostasis, membrane stability, and efficient functioning of metabolic processes. The integrity of cellular membranes is crucial for thermotolerance, as it prevents leakage of ions and other solutes, maintaining cellular turgor and function under heat stress [23]. Acquired thermotolerance represents a dynamic adaptive mechanism exhibited by plants subsequent to their exposure to non-lethal, elevated temperatures. This response prepares them to resiliently endure subsequent, more intense heat stress episodes. The intricate process involves the orchestrated induction of specialized HSPs, the activation of robust antioxidant systems, and the modulation of signaling pathways, all of which contribute to the plant's enhanced tolerance against heat stress [24].

2.1. Mechanism of Stress Memory

During stress events, plants acclimate to protect themselves from damage. This acclimation process has been studied extensively, but recent research highlights the importance of mild stress exposure, known as a "priming cue", which triggers a state of acclimation called the "primed state". Maintaining this primed state over time, referred to as "stress memory", enables plants to better handle future stress events that occur after a stress-free period [25–27]. In the primed state, plants become "sensitized" to subsequent stress incidents, leading to faster, stronger, or modified responses compared to unprimed plants. Stress memory can be somatic, lasting only one generation, or extend to future generations as intergenerational or transgenerational memory. When stress memory is transgenerational or when somatic memory is stable across multiple cell divisions, it aligns with the classical definition of epigenetic inheritance [28]. Even non-dividing cells can exhibit

epigenetic phenomena [29], suggesting that their stress memory may also be governed by epigenetic regulation [26,30].

2.2. Physiological and Developmental Responses to Heat Stress

Crop species and genotypes display a remarkable diversity in their optimal growth temperatures and heat tolerance throughout their life cycles. Amidst the natural variations in daily and seasonal temperatures, crops have evolved intricate mechanisms to adapt their growth patterns in response to temperature fluctuations. This adaptation, known as thermomorphogenesis, encompasses accelerated shoot and root development, as well as the timely transition to flowering. Nevertheless, exposure to heat shock poses a significant stress to plants, disrupting vital processes like respiration, photosynthesis, water and nutrient absorption, immune response, membrane integrity, protein function, and hormone and antioxidant metabolism. Notably, heat stress frequently results in sterility, particularly male sterility, leading to substantial yield reductions in crops like maize, rice, and wheat, which are staples in human diets [31].

Given the paramount importance of these crops to global food security, it is imperative to deepen our understanding of how temperature modulates their growth and development. Furthermore, the development of integrated strategies aimed at cultivating heat-tolerant crop varieties is crucial to safeguard food production amidst rising temperatures [21].

Plants employ conserved stress response mechanisms to cope with adversity; however, extreme heat stress can trigger ferroptosis pathways, leading to severe cellular damage or even death. This stress elicits a complex series of morphological, physiological, and biochemical changes, which are governed by the plant's genetic predisposition [32,33]. In an attempt to acclimate to heat stress, plants may redirect resources, but this adaptation often comes at a cost, as it may initiate programmed cell death in certain tissues, manifesting as leaf shedding, cessation of floral and fruit development, or even plant mortality in extreme cases [34–37].

Moreover, heat stress triggers the expression of heat shock genes, which encode heat shock proteins essential for managing the stress response. The regulation of these genes is influenced by both environmental cues and developmental signals, highlighting their pivotal role in critical stages of male gametogenesis and embryogenesis [38]. Additionally, heat stress exacerbates oxidative stress by promoting the overproduction of ROS, further compounding the overall stress impact on plants [39].

2.3. Molecular Mechanisms of Heat Stress Response

A small family of heat-induced transcription factors HsfA1s (heat shock transcription factors) plays a crucial role as the master regulators for both drought and heat stress responses (Figure 1) [40]. Mutations in three of the five *HsfA1* genes (*HsfA1a*, *HsfA1b*, and *HsfA1d*) significantly impair the HS response and plant survival under HS conditions [40–42]. These transcription factors activate HSPs and ROS-scavenging enzymes, which protect against HS-induced protein unfolding and oxidative stress. HS also leads to global translation inhibition, resulting in the formation of stress granules (SGs). These are membraneless organelles comprising untranslated mRNAs, RNA-binding proteins, translation initiation factors, and other components [43–46]. The formation of SGs in response to various stress conditions is a common phenomenon observed in many eukaryotes [47,48].

At the molecular level, a key response to HS involves the induction of TF HSFs. These HSFs regulate the expression of HSPs and ROS-scavenging enzymes, both of which play crucial roles in developing thermotolerance [19,22]. For instance, in rice, acquired thermotolerance is linked to the activity of OsHSP101 and OsHSA32 [1], while in tomatoes, HSP40 has been shown to safeguard melatonin synthesis, thus modulating abiotic stress tolerance under HS [49]. Additionally, plants activate a protective mechanism known as the unfolded protein response (UPR) to manage misfolded proteins in the endoplasmic reticulum during heat stress. In maize, this response is mediated by the transcription factor bZIP60, which, upon heat-induced splicing, enhances the expression of critical HSFs

essential for the HS response [50,51]. Similarly, in rice, the transcription factors OsbZIP74 and the membrane-associated NAC protein OsNTL3 are components of a UPR-related regulatory network that contributes to heat tolerance [52]. These HSF- and HSP-driven HSRs are vital for plants to adapt to high temperatures. Understanding these molecular mechanisms is crucial for breeding and developing heat-tolerant crop varieties.

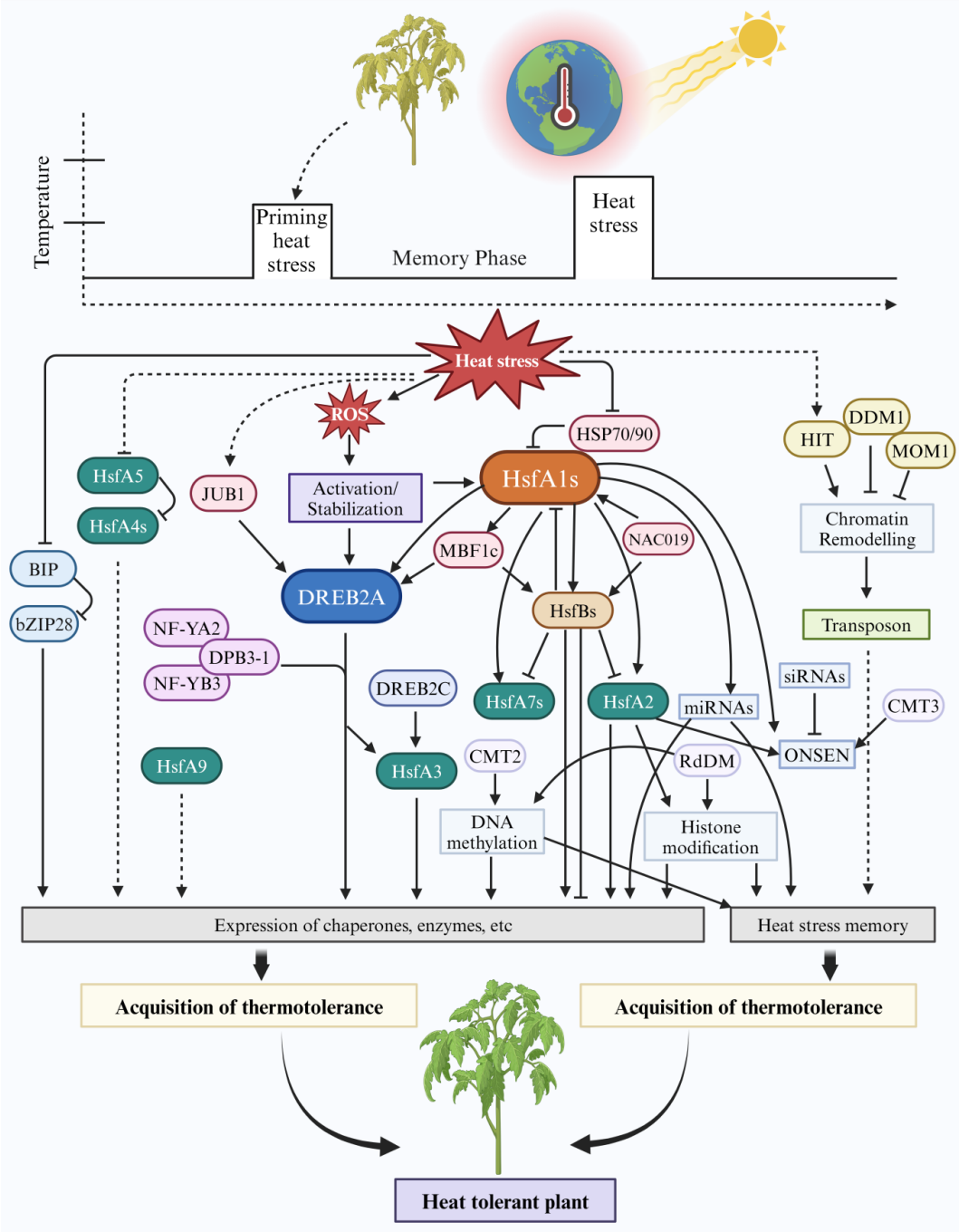

Figure 1. Epigenetic regulation pathways of heat shock response (HSR) in plants. HEAT SHOCK TRANSCRIPTION FACTOR A1s (HsfA1s), HEAT SHOCK PROTEIN 70 (HSP70) and HEAT SHOCK PROTEIN 90 (HSP90), DEHYDRATION-RESPONSIVE ELEMENT BINDING 2A (DREB2A), MULTIPROTEIN BRIDGING FACTOR 1C (MBF1C), JUNGBRUNNEN1 (JUB1), DNA POLYMERASE II SUBUNIT B3-1 (DPB3-1), NUCLEAR FACTOR Y, SUBUNIT A2 (NF-YA2), NUCLEAR FACTOR Y, SUBUNIT B3 (NF-YB3), DECREASED DNA METHYLATION 1 (DDM1), MORPHEUS MOLECULE 1 (MOM1).

Moderate HS enables plants to develop thermotolerance, allowing them to withstand potentially lethal high temperatures [53]. This capability, known as acquired thermotolerance, can persist for several days once plants return to non-stress temperatures, forming what is termed HS memory. Although the molecular processes that confer thermotolerance are well documented, the mechanisms underlying HS memory are not as clear. The acquisition of thermotolerance typically involves the activation of HSFs that trigger the production of HSPs to protect cellular proteins from denaturation [54]. This response to HS is common among plants, animals, and fungi [55]. In mammals, HSF1, beyond its role in heat stress, is also implicated in aging and various diseases [2].

In plants like *Arabidopsis thaliana*, there are over 20 specialized HSF copies, with eight specifically engaged in heat stress responses [3,42,54,56,57]. One notable HSF, HSFA2, is crucial for maintaining, but not acquiring, thermotolerance after its initial heat induction [56]. Similar roles have been identified for HSA32 [58] and miR156 [6]. Microarray studies have identified several HS memory-related genes, such as small HSPs (HSP21, HSP22.0, HSP18.2) and ASCORBATE PEROXIDASE 2 (APX2), which remain elevated for at least three days post-HS [6]. In contrast, non-memory genes, like HSP70 and HSP101, peak shortly after exposure to HS and then quickly decrease. The necessity of HSFA2 for maintaining the high expression levels of HS memory-related genes, but not for their initial induction, suggests that it directly targets these genes [56,58].

Thermotolerance in plants is a multifaceted trait involving both basal and acquired mechanisms. The interplay between HSPs, antioxidant systems, and signaling pathways is central to the plant's ability to cope with HS. Understanding these mechanisms is crucial for developing strategies to enhance thermotolerance in crops, thereby improving their resilience to global warming.

3. Epigenetic Regulation for Adaptation to HS

Plants have developed advanced epigenetic mechanisms to quickly adapt to heat stress and maintain transgenerational memory of heat-induced changes in post-transcriptional gene silencing (PTGS) [7]. Epigenetic regulation is crucial in plant development and the response to environmental challenges, allowing the plant epigenome to dynamically respond to various stimuli [59]. Under heat stress, epigenetic regulation involves DNA methylation, histone modification, and chromatin remodeling, with histone modification being particularly important in modulating transcriptional activity through chromatin configuration (see some examples of genes and epigenetic modifiers listed in Table S1) [60]. These processes often involve transcription factors (TFs) and are part of a multi-layered regulatory system [19]. Environmental changes induce epigenetic modifications that help plants remember past interactions, which is vital for future stress resistance [61]. Epigenetic memory in plants can be classified into three distinct types: cellular, transcriptional, and transgenerational. Cellular memory refers to the heritable transcriptional states that arise from developmental signals. Transcriptional memory entails the heritable changes in gene expression that enhance responsiveness to environmental cues. Lastly, transgenerational memory encompasses changes in gene expression that are inheritable through meiosis, enabling plants to respond to stress based on ancestral experiences [62–64].

Research suggests that plants are capable of retaining the memory of stress experiences across multiple generations [65,66]. Generally, epigenetic modifications induced by stress revert to their baseline once the stress diminishes; however, certain modifications remain stable and are passed down as epigenetic stress memory through both mitotic and meiotic divisions. Additionally, some modifications are reactivated during processes such as sexual reproduction or embryogenesis [61,67]. Stress memory varies in persistence and transmission, with somatic stress memory being immediate and temporary, while transgenerational memory transfers to subsequent generations without stress presence [8,26]. Mechanisms that contribute to somatic stress memory include nucleosome remodeling, histone PTMs, RNA interference, and DNA methylation [68–70]. In *Arabidopsis*, the stress-induced removal of the histone variant H2A.Z at elevated temperatures facilitates the binding and

activation of PIF4 to the FT gene, a key component of the thermomorphogenesis response. This process is supported by the interaction of the INO80 chromatin-remodeling complex with PIF4 [71,72]. Despite being initially described as a thermosensor, evidence suggests H2A.Z lacks true thermosensor properties [73].

Certain chromatin-related mechanisms are thought to underpin somatic stress memory, including nucleosome remodeling, an abundance of histone PTMs, RNA interference, and DNA methylation. Additionally, transgenerational stress memory involves changes in genome structure, inheritance of epialleles or chromatin states, and influences from either sperm or maternal contributions. During plant sexual reproduction, the transmission of epialleles across generations is usually hindered by active chromatin resetting processes [8,74,75]. Despite this resetting mechanism, plants demonstrate a higher frequency of epiallele transmission through sexual reproduction compared to mammals [8]. This increased rate is linked to the reprogramming of DNA methylation that primarily occurs in the vegetative nucleus of pollen, companion cells, and the central cell of the ovule—these are the cells that merge during fertilization to create the zygote [74]. Significant epigenetic alterations, such as histone exchange [76] and DNA demethylation [77], occur during gametogenesis in plants. Moreover, the robust capacity for vegetative reproduction in plants may also promote the potential for transgenerational inheritance, even in sexually reproducing species.

RNA-directed DNA methylation (RdDM) is a distinct pathway in plants, where noncoding RNAs guide the methylation of DNA and the establishment of H3K9me2 histone methylation, assisting plants in managing heat stress. Mutations in the components of the RdDM pathway lead to increased heat sensitivity in plants [78,79]. Additionally, histone modifications significantly influence the regulation of AT. For example, the heat shock factor HSFA2 is involved in promoting H3K4 hyper-trimethylation, while the JMJ family of demethylases facilitates H3K27 demethylation. Similarly, ASF1 influences thermotolerance through the removal of H3K56ac, and the complex involving FGT1-BRM/CHR11/CHR17 plays a crucial role in nucleosome positioning, collectively affecting AT in plants [80]. HSFA2 is a key regulator of AT [9,56], maintaining heat shock protein (HSP) gene expression and recruiting histone methyltransferases to memory loci, leading to sustained heat memory gene expression [81].

Epigenetic regulation allows plants to adapt to heat stress by altering gene expression without changing the DNA sequence, enabling rapid and reversible responses to environmental stressors. This regulation involves DNA methylation, histone modifications, RNA modifications, and other epigenetic marks, all contributing to gene expression control in response to heat stress (Figure 2).

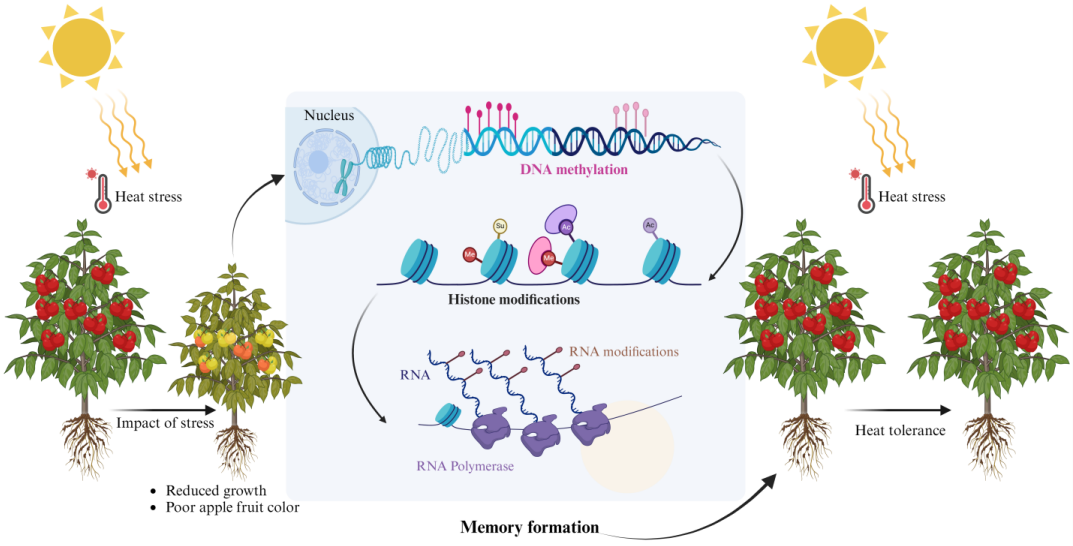

Figure 2. Epigenetic memory of heat stress in plants.

3.1. DNA Methylation

High temperature stress significantly impacts crop growth and development globally, inducing a range of morphological, physiological, and biochemical changes in plants. To mitigate the damage caused by heat, nearly all organisms have developed signaling pathways that monitor ambient temperature fluctuations and accordingly adjust their metabolic and cellular functions [82]. Recent advances have highlighted the crucial role of epigenetic regulation, particularly DNA methylation, in thermal responses [83,84]. Studies across different plant species have provided valuable insights into how DNA methylation contributes to stress tolerance. The relationship between DNA methylation and heat stress reveals that low DNA methylation levels can lead to flexible gene expression and plasticity, as observed in seagrasses [10]. In *Pinus halepensis*, elevated temperatures during somatic embryogenesis have been shown to impact DNA methylation and alter the expression of stress-related genes [11]. Similarly, *Arabidopsis* displays specific adjustments in its DNA methylation and demethylation mechanisms when exposed to high temperatures, facilitating the precise addition and removal of DNA methylation [85]. Active demethylation plays a critical role in responding to heat stress [86]. The significance of DNA methylation in regulating gene expression, which is vital for heat acclimation, is becoming more recognized [35,87,88]. For example, in *Brassica napus*, differences in DNA methylation between heat-sensitive and heat-tolerant genotypes underscore its importance in responding to environmental stress [89]. Additionally, DNA methylation by the RdDM pathway, which affects various heat-sensitive genes, also play a crucial role in regulating DNA methylation critical for heat tolerance in *Arabidopsis* [79]. Heat stress-induced changes in gene expression, such as those seen in tobacco BY-2 cells, correlate with altered DNA methylation states, suggesting that DNA methylation modulates gene expression during heat [79,90].

Studies indicate that overall methylation levels in plants under heat stress are generally lower compared to control plants, as seen in *Populus simonii* and *Brassica napus* [91,92]. Heat stress affects cytosine methylation across various genes, with heat-sensitive genotypes showing higher methylation levels and more methylation events compared to heat-resistant genotypes, which exhibit more demethylation events [89]. In *Arabidopsis*, exposure to heat stress primarily leads to DNA demethylation in gene coding regions rather than in intergenic areas [86].

Additionally, TEs that are activated under heat stress contribute to the plant's stress response. This is exemplified by the activation of the LTR-copia-type retrotransposon *ONSEN* in *Arabidopsis* when subjected to heat stress [13]. The enzymes DNA methylases and demethylases play a crucial role in regulating the dynamic status of DNA methylation under heat stress conditions, ensuring that the genome's response to environmental changes is both rapid and precise [93–95]. In *Brassica napus*, 22 DNA methylase genes and 6 DNA demethylase genes have been identified, with expression analyses suggesting their involvement in the heat/salt stress response [96]. In *Arabidopsis*, the genetic regulation of epigenetic modifications, such as those involving CMT2, plays a role in natural adaptation to temperature [97]. DNA methylation and demethylation processes are crucial for proper seed germination under heat stress, with genes like *ROS1* affecting the expression of germination-related genes [98].

While the connection between DNA methylation and heat stress memory is not completely understood, it is recognized that DNA methylation by the RdDM pathway controls multiple heat stress-responsive genes and is critical for both basal heat tolerance and transgenerational memory in *Arabidopsis* [79,99]. In *Brassica napus*, genotypes that are sensitive to heat show increased DNA methylation levels, suggesting that DNA methylation is dynamically responsive to heat stress [89]. In *Arabidopsis*, heat-induced DNA demethylation is associated with intergenerational stress memory, where differentially methylated regions (DMRs) enriched with transposons play substantial roles [86,100]. This heat-accelerated methylation is key to either silencing or activating transposons, thereby influencing stress memory. For example, in *Arabidopsis*, the DNA methyltransferase CHROMOMETHYLASE3 (CMT3) activates the retrotransposon *ONSEN*, which plays a role in transcriptional memory

under heat stress conditions [13,101]. DNA methylation by the RdDM pathway suppresses chromatin modifications and aids in stress memory by preventing transpositional activities in heat-stressed progeny [14,68]. Furthermore, heat-induced demethylation in maize suggests that DNA demethylation controls heat responses via RNA splicing pathways [102]. DNA methylation is a dynamic and reversible epigenetic modification that plays a crucial role in plant adaptation to heat stress. By modulating the expression of stress-responsive genes, DNA methylation helps plants to rapidly adjust their physiological and biochemical processes in response to changing temperatures, enhancing their thermotolerance and overall survival under adverse environmental conditions. The interplay between gene expression, transcriptomic plasticity, and DNA methylation under heat stress remains underexplored and warrants further investigation.

3.2. Histone Modifications and Stress Memory in Plants

Stress memory in plants is a complex phenomenon regulated at multiple levels, ranging from metabolites to chromatin structure. Modifications in chromatin structure, particularly through histone modifications, are thought to mediate changes in gene expression patterns associated with stress memory [81]. Chromatin, which consists of DNA and proteins, is essential in regulating gene expression through mechanisms such as nucleosome positioning, incorporation of histone variants, and histone posttranslational modifications [103,104]. These modifications can enhance or inhibit transcription by modifying chromatin accessibility or by interacting with specific protein complexes. Transcriptional memory, a phenomenon observed in various organisms, including yeast, mammals, and plants, describes how gene activation can occur more rapidly and robustly upon subsequent stimuli after an initial exposure and a period of latency or suppression [67,105,106].

In plants, transcriptional memory is increasingly recognized as a mechanism enabling them to remember past environmental stressors to prepare for future incidents. For instance, HS memory in plants has been well-characterized at both the whole plant and molecular levels [6,15,56,105,107]. Studies have highlighted the role of histone modifications in HS memory, particularly through interactions with heat shock factor A2 (HSFA2). HSFA2 is essential for maintaining the induction of HS memory-associated genes by directly binding to these loci shortly after HS exposure. Elevated levels of H3K4me3 and H3K4me2 were identified as marks distinguishing HS memory-related genes from other HS-inducible genes, persisting even after HSFA2 binding declined, thus facilitating a robust response upon subsequent HS exposure [81]. Thus, HS memory comprises two primary components: the prolonged activation of HS memory-associated genes and a varied response to subsequent HS events. Both aspects are linked to increased levels of H3K4me3 and H3K4me2 and rely on the functionality of HSFA2. Overall, the research highlights H3K4 methylation as a key marker for HS memory, emphasizing its reliance on a transiently binding transcription factor [81].

Heat acclimation, critical for stress adaptation, involves specific patterns of gene expression regulated by dynamic histone modifications under heat stress conditions [16,108]. These modifications include rapid changes during stress and gradual adjustments in response to the heat stimulus [17]. The histone variant H2A.Z, which incorporates H2A, H2A.Bbd, H2A.X, and H2A.Z, is crucial for temperature sensing via nucleosome occupancy. In *Arabidopsis*, the SWR1 chromatin remodeling complex mediates the insertion of H2A.Z into nucleosomes, replacing H2A in an ATP-dependent manner, with ARP6 encoding SWR1, playing a critical role in histone modification and temperature response [109]. In *Brachypodium*, mutations in H2A.Z impair heat acclimation, underscoring its role in temperature response [110]. Additionally, under heat stress, the levels of histone acetylation, specifically H3K9ac and H4K5ac, have been observed to increase in maize, underscoring the significance of acetylation and methylation in facilitating chromatin de-condensation [111]. In *Arabidopsis*, the chromatin protein BRUSHY1 (BRU1) plays a critical role in the epigenetic inheritance of chromatin states, aiding in the sustained transcriptional activation of heat stress memory genes [112]. Furthermore, the histone acetyltransferase GCN5 is essential for

maintaining heat tolerance in *Arabidopsis*, where its suppression leads to reduced heat tolerance, whereas its overexpression in gcn5 mutants boosts stress memory and reestablishes stress tolerance [113]. Histone deacetylases (HDACs), including HD2C, HDA6, HDA9, HDA15, and HDA19, play diverse roles in regulating stress responses. For instance, in *Arabidopsis*, the interaction of HDA9 with POWERDRESS (PWR), a SANT-domain protein, enhances heat tolerance [18], conversely, HDA15 diminishes heat tolerance through its interaction with the transcription factor long Hypocotyl in Far Red1, demonstrating the varied impacts of HDACs on heat stress adaptation [114].

Histone methylations also play crucial roles in heat stress responses and memory. A notable histone modification is the trimethylation of lysine 4 on histone H3 (H3K4me3), which is associated with active gene expression. Elevated levels of H3K4me3 facilitate rapid gene activation in response to environmental stimuli, preventing gene silencing [19]. In *Arabidopsis*, H3K4me3 correlates with active transcription, whereas H3K4me2 has a negative correlation with active transcription in rice. These modifications are essential for regulating genes related to development and stress response. Elevated H3K4me3 and H3K4me2 levels are associated with the hyper induction of heat stress memory genes under continuous heat stress [21,81,115]. Reduced H3K27me3 at HSP17.6C and HSP22 genes, mediated by JMJC histone demethylases, increases HS memory by maintaining H3K4me3 levels at the HSP21 gene, balancing H3K27me3 and H3K4me3 levels for stress memory [22]. Additionally, decreased H3K9me2 is crucial for regulating Fertilization-Independent Endosperm1 (OsFIE1), a member of PRC2, in seed development under heat stress in *Arabidopsis* [116]. Histone SUMOylation, mediated by HDACs, is vital for transcriptional regulation in heat acclimation. Heat stress in *Arabidopsis* decreases histone SUMOylation (H2B) while increasing GCN5, indicating a complex interplay among histone modifiers [17]. Despite advancements, the precise roles and mechanisms of these modifications in stress memory remain underexplored.

Recent studies have shown that methylation of H3K4 is crucial for the sustained expression of HS-inducible genes and their hyper-induction during repeated HS exposures [81]. This modification enables rapid reactivation of genes during recurring stress, acting as a form of HS memory. H3K4 hypermethylation is associated with HsfA2 binding, suggesting that HsfA2 recruits histone methyltransferases to HS memory loci. This mechanism appears conserved across species, as human HSF1 can also modulate chromatin in response to heat stress [117]. In *Arabidopsis*, the histone chaperone ANTI-SILENCING FUNCTION 1 (ASF1) is crucial for HS-inducible gene expression, facilitating nucleosome removal and H3K56 acetylation under HS conditions [118]. Plants possess sophisticated epigenetic mechanisms to respond to heat stress quickly [83]. Heat exposure activates normally silenced transgenes and repetitive elements through transcriptional gene silencing (TGS) and releases post-transcriptional gene silencing (PTGS) by inhibiting siRNA biogenesis [16,101,119,120]. Heat stress reduces trans-acting siRNAs (tasiRNAs) from eight *Arabidopsis* TAS loci, upregulating HEAT-INDUCED TAS1 TARGET (HTT) genes that enhance thermotolerance by interacting with Hsp70–14 [121]. Importantly, DNA methylation is not involved in the release of gene silencing triggered by heat stress [83].

Heat stress responses are typically transient, though some somatic stress memory can last several days [26]. Factors like Decrease in DNA methylation1 (DDM1) and Morpheus' Molecule 1 (MOM1) inhibit the transgenerational transmission of heat-induced epigenetic states [23]. Additionally, the small interfering RNAs (siRNAs) pathway prevents the transgenerational retrotransposition of ONSEN [101]. However, certain responses to prolonged heat stress exhibit transgenerational memory detectable in subsequent stress-free generations [120,122,123]. For example, offspring of extreme heat-stressed *Arabidopsis* plants tend to bolt earlier [122], and heat stress-mediated release of reporter gene silencing can be transmitted to non-stressed progeny, limited to two generations [120]. H3K4me3 is widely recognized as being associated with active chromatin states [124]. This particular histone modification is also connected to transcriptional memory, particularly at genes responsive to abiotic stress. For example, upon priming with dehydration, H3K4me3

accumulates at type II dehydration memory genes and is maintained during the memory phase [30]. Similar patterns of H3K4me2/3 accumulation were observed in plants primed with jasmonic acid (JA) [125] or HS [81,126]. These observations suggest that increased H3K4 methylation serves as a marker for recently activated genes, priming them for more robust reactivation in response to subsequent stress events.

In plants that have experienced repeated dehydration events, RNA polymerase II (Ser5P Pol II), which becomes stalled, shows an increased presence at memory-specific genes during the memory phase. This adaptation results in a higher rate of transcription during subsequent stress events compared to the initial stress exposure [30]. Increased occupancy of the TATA-binding protein (TBP) at memory gene promoters during recovery phases suggests the maintenance of the transcriptional machinery at these sites, facilitating faster reactivation upon recurrent stress [125]. This indicates that H3K4me3 may create a chromatin environment conducive to transcriptional machinery accessibility. The COMPASS-like complex, responsible for depositing H3K4me3, regulates various aspects of plant development [25]. Upon induction of the unfolded protein response (UPR), this complex is recruited by the bZIP28/60 transcription factors to deposit H3K4me3 at specific genes [127]. Given the UPR's commonality in abiotic stress responses [128], similar mechanisms might be involved in HS and dehydration memory genes. Alternatively, different stress-specific transcription factors might recruit the COMPASS-like complex. In yeast, COMPASS and Mediator complexes are crucial for the transcriptional memory of the INO1 gene, involving increased H3K4me levels upon activation [129,130].

Enzymes responsible for H3K4me deposition at memory genes include the H3K4me3 methyltransferase ARABIDOPSIS TRITHORAX1 (ATX1), essential for dehydration stress response [131]. However, other factors likely provide memory specificity, as stress-induced transcription factors play key roles in transcriptional memory. For instance, HS-dependent H3K4me deposition relies on HSFA2, with additional factors dependent on HSFA1 also involved [81,126]. The promoter of the type II memory gene ASCORBATE PEROXIDASE 2 (APX2) is sufficient for transcriptional memory, indicating that TF binding and *cis*-regulatory motifs are critical [126]. HSFs, acting in trimeric complexes, may also contribute to HS memory specificity [54]. ABA-dependent dehydration memory genes require MYC2 for transcriptional memory, involving the recruitment of the Mediator complex [125]. This complex integrates TF binding with RNA Pol II activity, affecting various plant development and stress responses [132]. However, the relationship between MYC2–MED25 binding and H3K4me3 deposition at dehydration memory genes remains to be elucidated.

The interplay between different histone modifications is critical for the regulation of gene expression under heat stress. These modifications do not act in isolation but rather in a coordinated manner to fine-tune the chromatin landscape and ensure an appropriate transcriptional response. For instance, the combined action of histone acetylation and methylation can create a permissive chromatin state for the activation of heat-responsive genes, while repressive marks ensure that non-essential or potentially harmful genes remain silenced. Understanding the precise roles of these histone modifications and their dynamic changes under heat stress provides valuable insights into the molecular mechanisms of plant thermotolerance. This knowledge can be leveraged to develop strategies for enhancing crop resilience to heat stress through epigenetic breeding and biotechnological interventions.

3.3. RNA Modification

RNA molecules undergo various post-transcriptional modifications that significantly influence their stability, translation, and overall function. These modifications are crucial for fine-tuning gene expression in response to environmental stressors, including heat stress (HS). RNA modification plays a crucial role in the regulation of plant responses to environmental stress, particularly HS. Regulatory RNA molecules, including microRNAs (miRNAs), small interfering RNAs (siRNAs), long noncoding RNAs (lncRNAs), and circular

RNAs (circRNAs), significantly impact the expression of transcription factors (TFs) and genes related to stress responses.

MicroRNAs are small noncoding RNAs that target messenger RNA (mRNA) for degradation or translational repression, thereby downregulating gene expression. For example, miR398, which functions downstream of HSFA7b and HSFA1s during HS, is suppressed under oxidative stress, leading to the accumulation of the COPPER/ZINC SUPEROXIDE DISMUTASE 1 (Cu/ZnSOD1) and Cu/ZnSOD2 genes. These genes encode enzymes that detoxify superoxide radicals, thus enhancing tolerance to oxidative stress [133]. Similarly, miR156, another miRNA induced by heat stress, represses SQUAMOSA-PROMOTER BINDING-LIKE (SPL) TFs, including SPL2, SPL9, and SPL11. This repression maintains the expression of HSFA2, contributing to heat stress memory [6]. When plants are exposed to heat, miR156 plays a crucial role in maintaining the expression of HSFA2 even after the HS subsides, thereby enhancing AT and strengthening the plants' defensive capabilities against future heat challenges. Additionally, miR159 and miR396 specifically target MYB and WRKY transcription factors, respectively, which are key in conferring thermotolerance in plants [134]. Furthermore, recent studies have revealed that miR165/166 and its target, PHABULOSA (PHB), constitute a regulatory complex for HSFA1 that operates at both the transcriptional and translational levels in response to HS. Under typical conditions, PHB acts to suppress the transcription of HSFA1 and plays a broad role in regulating heat-responsive genes. During episodes of heat stress, miR165/166 levels increase, leading to the suppression of PHB. This reduction in PHB levels allows for the activation of HSFA1, which in turn triggers the expression of genes involved in the heat stress response [135].

SiRNAs also play a vital role in the heat stress response. The retrotransposon ONSEN, targeted by HSFA1, is regulated through a siRNA-mediated pathway. The suppression of ONSEN by siRNA leads to increased expression of heat-induced genes. HSFA1s further promote thermotolerance by binding to promoters and activating the transcription of HEAT-INDUCED TAS1 TARGET 1 (HTT1) and HTT2 genes. This action is supported by trans-acting siRNA (TAS1) and natural antisense transcript siRNA (nat-siRNA), which contribute to heat resistance by negatively regulating HTT1 and HTT2 [136]. CircRNAs are unique, single-stranded RNAs that form a closed loop by joining their ends head-to-tail. These molecules have been identified as playing a significant role in regulating the plant response to HS, working in synergy with plant hormone signaling pathways [137]. This interaction enhances the plant's ability to cope with thermal stress by modulating various stress response mechanisms.

MicroRNAs are known to regulate plant stress responses through various mechanisms [26,138–142]. They are approximately ~21-nucleotide-long single-stranded RNA molecules that direct effector proteins, predominantly ARGONAUTE1 (AGO1), to complementary mRNAs, resulting in mRNA cleavage or translational inhibition [143,144]. These miRNAs originate from primary transcripts transcribed by RNA polymerase II, which fold into hairpin structures and are processed by DICER-LIKE1 (DCL1) to produce active miRNAs [145]. They are critical for plant growth, development, and stress response modulation, including growth and development adjustments after stress exposure [138]. A notable example is miR156, which is involved in the adaptation to recurring heat stress in *Arabidopsis*. miR398 also plays a significant role by negatively regulating several ROS-scavenging enzymes during heat stress, resulting in ROS accumulation, which activates HsfA1 and establishes a positive feedback loop. Conversely, miR156 helps maintain the expression of HS-inducible genes during recovery from heat stress, contributing to heat stress memory [6,105]. miR156 specifically targets SPL proteins, leading to the downregulation of genes that are induced by HS. Essential for sustaining the expression of HsfA2 and heat HSPs post-HS, miR156 plays a crucial role in the heat stress response. This interaction between miR156 and SPL is considered to be a key component of HS memory, helping plants to enhance their resilience against future heat stress events.

The role of AGO1 and DCL1 in the miRNA pathway has been further elucidated through studies on mutants. The reduced heat acclimation capacity observed in ago1 and

dcl1 mutants highlights the miRNA pathway's importance in heat acclimation. Transcriptome analyses of these mutants have identified differentially expressed pri-miRNAs, with miR156 showing the most robust accumulation in response to acclimation. This miRNA, by post-transcriptionally downregulating SPL genes, triggers the sustained induction of heat memory genes [146–148]. The regulation of plant heat stress responses by various noncoding RNAs, especially miRNAs, underscores their crucial role in enhancing plant resilience to environmental stress. Understanding these regulatory networks offers valuable insights into plant stress biology and potential strategies for improving crop tolerance to adverse conditions.

RNA modifications play a crucial role in the regulation of gene expression in response to heat stress. By modulating the stability, splicing, and translation of mRNAs encoding stress-responsive proteins, these modifications contribute to the plant's ability to cope with elevated temperatures. Understanding the mechanisms and functions of RNA modifications in the heat stress response provides valuable insights into plant adaptation and resilience, offering potential strategies for improving crop performance under global warming conditions.

4. Breeding Application of Heat Stress-Induced Epigenetic Modifiers

Ensuring sustainable agricultural production in the face of changing climatic conditions requires the adoption of climate-resilient plant breeding strategies. These strategies are designed to develop crop varieties that can withstand variable temperatures. Modern tools and techniques, including molecular markers, have proven invaluable in enhancing plant performance under both cold and heat stress, offering a more efficient and cost-effective alternative to traditional breeding methods. In the last two decades, epigenetics has emerged as a significant field in crop improvement, enhancing plant resilience and survival by bolstering stress tolerance and memory. Technologies that enable the manipulation of epigenetic processes in plants hold the potential to revolutionize crop development.

Recent studies highlight the significant impact of epigenetics in developing climate-resistant crops and transforming plant breeding practices. Breeders can utilize insights from epigenetic pathways to increase stress tolerance, boost crop yields, and improve plant adaptability to changing environmental conditions [149,150]. Epigenetic memory in plants allows them to remember past stress events and respond more effectively to future challenges. Breeding programs can harness this memory to enhance plant resilience to environmental stressors, like drought, heat, and disease. This can be accomplished through controlled stress exposure or via epigenetic modifications, resulting in plants that not only survive but thrive in adverse conditions [151,152]. The persistence of epigenetic changes across generations is essential for providing future plant populations with enhanced mechanisms to cope with stress. By selecting and breeding plants with desirable traits, breeders can develop varieties that not only possess increased resistance to environmental stressors but also inherit stress memory to subsequent generations, thereby boosting their adaptability [153]. This approach not only meets immediate agricultural needs but also contributes to the long-term sustainability and resilience of crop production.

The emergence of genome editing technologies like CRISPR-Cas9 has revolutionized the ability to precisely modify epigenetic traits. This advancement, known as epigenome editing, enables breeders to specifically target and control the expression of stress-sensitive genes. By altering DNA methylation patterns or histone modifications at crucial stress-related loci, plants' stress tolerance can be significantly enhanced [30,136,154,155]. Regulating flowering time via epigenetic mechanisms is also vital for breeding plants that can adapt their flowering periods to thrive in diverse climates, thus improving their reproductive success [156]. Research into naturally occurring epigenetic changes in wild plant populations that confer climate resilience could further aid the development of climate-resistant crops. Selective breeding techniques can introduce these adaptive changes into cultivated plant species, increasing their resilience to climate change [157]. The exploration of epigenetic diversity within plant germplasm collections is beneficial for identifying

candidates with desirable traits for stress tolerance, which can then be enhanced through breeding programs [149,158]. These epigenetic variations can be incorporated into elite breeding lines using crossbreeding or biotechnological approaches, and plants with favorable epigenetic traits can undergo further selection and propagation to develop stable, heat-tolerant varieties [159].

Incorporating epigenetics into conventional breeding methods offers a new dimension to crop improvement. However, it is important to consider ethical issues and the potential for unforeseen effects of epigenetic modifications. The application of epigenetic techniques in plant breeding is increasingly seen as a way to ensure food production and long-term sustainability in the context of climate change [160,161]. Research has shown that epigenetic information can define complex traits, such as fruit maturity in tomatoes [32] and somaclonal variation in palms [33]. Transgenerational epigenetic changes allow for the inheritance of epigenetic marks across generations, contributing to natural phenotypic variation that helps plants adapt to environmental changes [162–164]. Furthermore, generating alternative phenotypes or mutants through epigenetic changes, known as epimutations, represents a potential strategy for enhancing crop traits [165]. Epimutations can arise due to environmental stress affecting the genome [166], and such mutations, whether occurring spontaneously or through genetic variation, have been identified in various crops [167]. Identifying and utilizing epimutations that enhance plant adaptability to stress can profoundly improve crop resilience in future generations [34], making the use of epialleles or epimutations a promising approach for advancing crop development.

5. Conclusions and Perspectives

Gaining a profound understanding of the epigenetic regulation of heat stress responses in plants sheds light on the intricate mechanisms underlying thermotolerance. Leveraging this knowledge can propel the development of innovative breeding strategies, aimed at cultivating heat-resilient crop varieties, which are paramount in mitigating the impacts of global warming. Integrating epigenetic modifications into plant breeding holds immense promise for bolstering crop resilience and safeguarding sustainable agricultural productivity amidst shifting climatic conditions. Epigenetic modifications, encompassing DNA methylation, histone alterations, and RNA modifications, serve as pivotal regulators of plant heat stress responses. These modifications dynamically and heritably influence gene expression, empowering plants to adapt to and retain memories of stress encounters. Specifically, DNA methylation fine-tunes the expression of stress-responsive genes, histone modifications foster transcriptional memory, while RNA modifications fine-tune the stability and translation of stress-associated mRNAs. Collectively, these epigenetic mechanisms reinforce plant thermotolerance, empowering them to withstand elevated temperatures with greater resilience.

The implementation of heat stress-induced epigenetic modifiers in breeding programs holds tremendous promise for cultivating crops with unparalleled climate resilience. By strategically manipulating epigenetic marks through traditional breeding techniques, epigenome editing advancements, and harnessing naturally occurring epigenetic variations, breeders can cultivate crop varieties with enhanced stress tolerance and superior adaptive abilities. Advanced technologies, such as CRISPR-Cas9, offer precision in modifying epigenetic traits, precisely targeting stress-sensitive genes to bolster plant resilience.

While the prospects are exciting, there remain pivotal challenges to be addressed. Chief among them is the need for a deeper understanding of the stability and heritability of epigenetic marks across generations, as certain modifications may exhibit reversibility or be influenced by environmental cues. Furthermore, the intricate interplay between diverse epigenetic marks and their combined impact on gene expression necessitates extensive research. Comprehensive studies are imperative to identify reliable epigenetic markers for selective breeding and to elucidate the underlying mechanisms governing epigenetic regulation of thermotolerance. Addressing these challenges will pave the way for the

successful development of heat-resilient crops, ensuring global food security amidst the threat of climate change.

Future research endeavors should prioritize elucidating the intricate interplay among various epigenetic modifications and their cumulative effects on gene expression patterns and plant stress responses. By seamlessly integrating epigenetics with traditional breeding methodologies and cutting-edge biotechnological tools, we can unlock immense potential for bolstering crop resilience. Furthermore, delving into the natural epigenetic diversity present within wild plant populations presents a treasure trove of invaluable insights and genetic resources that can significantly contribute to the breeding of climate-resilient crops.

In essence, epigenetic regulation emerges as a potent lever for enhancing plant thermotolerance and safeguarding food security amidst the backdrop of global warming. By harnessing the dynamic and reversible nature of epigenetic alterations, we can cultivate crops that are more resilient to the myriad challenges posed by climate change. Consequently, sustained research efforts and innovative breakthroughs in this field are paramount for achieving sustainable agricultural productivity and addressing the food demands of an ever-growing global populace.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/agronomy14092105/s1, Table S1. Genes and Epigenetic Modifications and Their Role in Heat Stress Response.

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