

Review

Flowering Under Heat: Linking Phenological Adaptation, Reproductive Resilience, and Yield Stability in Plants

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Abstract

The reproductive stage is a critical time in a plant's life history when dealing with heat stress as the specific processes of meiosis, gametogenesis, anthesis, pollination, fertilization, and early seed development all occur within comparatively narrow temperature limits. Much of the importance of flowering is tied directly to temperature, both for the timing of reproductive transition and the ability for male and female reproductive tissues to survive exposure to damaging heat. Ambient-temperature sensing is linked to flowering via regulatory modules that involve phytochrome *B*, *EARLY FLOWERING 3 (ELF3)*, *PHYTOCHROME INTERACTING FACTOR 4 (PIF4)*, *FLOWERING LOCUS T (FT)*, *FLOWERING LOCUS M (FLM)*, *SHORT VEGETATIVE PHASE (SVP)*, and the light-circadian components. Conversely, when temperature is harmful, protective responses involve other cellular mechanisms such as activation of heat-shock transcription factors (HSFs), heat-shock proteins (HSPs), endoplasmic-reticulum protein quality control, calcium and reactive oxygen species (ROS) signaling, antioxidant systems, hormone regulation, metabolic reprogramming, autophagy and DNA-repair pathways. Male reproductive development is often very sensitive, especially at meiosis, during formation of the tetrad, microspore development, during the maturation of pollen, and during the growth of the pollen tubes, although injury to pistils, ovules and the post-fertilization tissues may solely have an effect on the restriction of fertilization and seed set. Phenological heat escape and intrinsic reproductive thermotolerance are genetically separable but can be complementary aspects of adaptation, as revealed by natural allelic variation, QTL mapping, genomic prediction and marker assisted selection. This review summarizes molecular, genetic and physiological evidence, to propose that, to ensure stable yields at high temperatures, there is a need to coordinate optimization of reproductive timing, cell and development thermotolerance, and post-fertilization sink stability.

Keywords: heat stress; flowering time; reproductive thermotolerance; pollen; pistil; phenological adaptation; heat-shock factors; reactive oxygen species; genetic variation; yield stability



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1. Introduction

Plant reproductive processes are highly vulnerable to high temperatures (supra-optimal temperature) and account for distinct developmental processes that are critical for crop productivity, especially under increasing temperature and more frequent extreme heat exposure [1–4]. A uniform treatment applied to the whole plant, like heat stress, is different from reproductive heat stress because the damage caused is related to the duration of heat stress as well as the time of day, genotype and developmental stage [5,6]. Although vegetative tissues may appear relatively unaffected following brief heat exposure, reproductive tissues can experience irreversible fertility loss when heat coincides with meiosis, microspore development, anthesis, or fertilization [7–9]. Exposure to temperatures above genotype-specific thresholds in rice can substantially reduce spikelet fertility, with the extent of fertility loss increasing with both temperature intensity and duration [6,10–12]. Heat sensitivity can be detected in tomatoes only during pre-anthesis and early post-anthesis windows, but not just from the day of flower opening [9,13]. Similar stage dependency exists in sorghum, soybean, common bean, maize, and crops of the Brassica species [7,14–17].

Environmental sensing and reproductive competence are two primary conditions that come together at flowering, which is the time point when temperature plays a role in triggering the plant to flower, as well as determining whether reproductive tissues are affected by heat stress after flowering [18–20]. Developmental timing is mediated by temperature-driven and photoperiod/circadian-driven response pathways, which interact with each other within the plant [21–24]. Apart from PIF4-mediated direct activation of FT at higher ambient temperature, the regulation of floral transition by FLM–SVP-dependent repression and by temperature-sensitive FLM RNA processing supplements these mechanisms in *Arabidopsis thaliana* [19,20,25–27]. In addition, Phytochrome B and ELF3 have also been shown to have direct or indirect temperature-sensing ability, and flowering is part of a larger network that connects light, circadian phase and temperature [22,23,28]. In addition, the release of the gene-regulating mechanism FLM alternative splicing further demonstrates the integration of the photoreceptor and RNA-processing pathways in thermosensory flowering [29].

Temperature exposure can be minimized during flowering time through phenological plasticity, which may shift the flower production timing during predictable periods of terminal stress [18,30]. This escape mechanism is directly applicable to wheat breeding: introgression of early-anthesis alleles in wheat with markers may decrease exposure to terminal heat and keep desirable grain traits [30]. However, phenological heat escape represents an avoidance strategy rather than intrinsic cellular thermotolerance [7,31]. At the other extreme, a genotype exposed to heat might be able to maintain its fertility through mechanisms including gametophytic, pistil, proteostasis, redox and/or DNA-repair systems [12,32,33]. Genetic analysis should therefore differentiate between how much exposure is altered from any effect and how it is responded to in order to prevent misinterpretation of cellular heat tolerance [30,34,35].

When reproductive tissues are subjected to aberrant temperature, fertility requires multiple processes that, although out of phase in their development, proceed successfully under normal circumstances. These include the process of chromosome pairing and segregation, the differentiation of the tapetum, the formation of the pollen wall, carbohydrate and lipid provisioning, development of the anthers, receptivity of the stigma, pollen germination, pollen-tube growth, competence of the ovules, fertilization, and development of the embryo within the seed or ovule [36–40]. Heat causes these processes to become disrupted, resulting in the creation of oxidative imbalance, changes in calcium signaling, impaired functioning of membranes, hormone disruption, metabolic changes, and defects in genome maintenance [33,41–44]. The following are all examples of responses that are not independent;

ROS and calcium are both developmental and heat signals in pollen, whereas protein quality control needs to maintain developmental programs and not only keep cells alive [45–48].

Thus, in this review, we concentrate on three facets that share mechanistic connections in their response to heat during flowering—reproductive physiology, molecular regulation, and genetic architecture. The main idea is that yield resilience is the result of a combination of the appropriate phenological positioning and intrinsic protection of male and female reproductive development, and enough metabolic and postfertilization stability to convert successful fertilization into the production of seeds or fruits [3,18,49]. The integrated framework linking thermal environment, phenological regulation, reproductive vulnerability, protective mechanisms, and final yield stability is summarized in Figure 1.

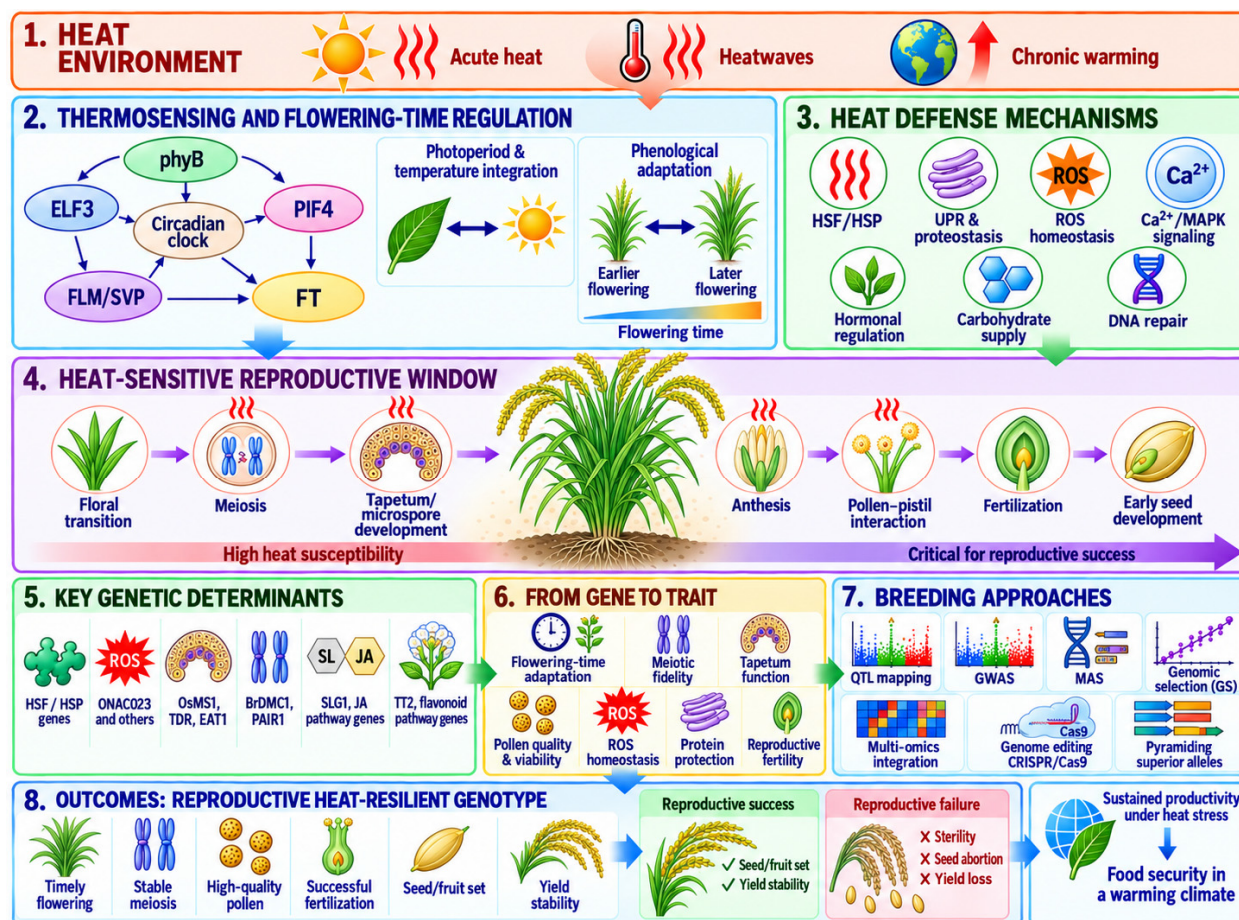


Figure 1. Comprehensive model connecting heat environment, thermosensing, reproductive susceptibility, heat defense mechanisms and grain stability in plants. The rationale of reproductive heat resistance at the flowering stage is shown. Acute heatwaves and chronic warming are presented as environmental triggers that cause the regulation of thermosensing and flowering time via phytochrome B (phyB), EARLY FLOWERING 3 (elf3), PHYTOCHROME INTERACTING FACTOR 4 (pi4), FLOWERING LOCUS T (ft), FLOWERING LOCUS M/SHORT VEGETATIVE PHASE (flm/svp), and circadian regulation. These pathways affect phenological adaptation by determining when flowering will begin and therefore the timing of contact between reproductive tissues and high temperatures. The floral transition stage, meiosis, stage of tapetum and microspore development, anthesis, pollen–pistil interaction, fertilization, and early seed development are all sensitive to heat. Several protective mechanisms are needed to ensure reproduction during heat: HSF/HSP-mediated thermoprotection, unfolded protein response (UPR) and proteostasis, reactive oxygen species (ROS) homeostasis, Ca^{2+} /MAPK signaling, hormonal regulation, carbohydrate supply, and DNA repair. These mechanisms can affect reproductive success, seed or fruit set, and/or stability of yield or reproductive

failure, sterility, seed abortion, and/or loss in yield. The bottom summary panel highlights that reproductive heat resistance depends on three aspects: phenological optimization, intrinsic reproductive thermotolerance and physiological stability. Generated with the assistance of ChatGPT (OpenAI, San Francisco, CA, USA; GPT-5.6 Sol) and subsequently edited by the authors. The authors reviewed and verified the final figure for scientific accuracy.

2. Physiological Basis of Reproductive Heat Sensitivity and Resilience

Reproductive heat sensitivity is highly stage-dependent since flowering involves a series of cellular events that each have unique metabolic and structural demands [4,50,51]. Therefore, the same temperature regime can affect different stages of floral organ development, meiosis, tetrad formation, etc., in pollen grains, pollen maturation, or anthesis, pollination, fertilization, and the establishment of seeds in different ways [9,40,52,53]. This is why, for instance, heat stress applied several days prior to anthesis has a stronger impact on pollen fertility than the same stress applied to mature pollen, and why other whole-plant phenotyping approaches that do not accurately stage plant development can mask the impact on pollen fertility [7,31,54].

Male meiosis is a sensitively timed process in which events such as homolog pairing, recombination, spindle function, chromosome segregation, and cytokinesis occur at precise times and in certain steps [11,55,56]. Meiotic couplings in *Arabidopsis* are also thermoresponsive, as revealed in the enhanced frequency of crossovers and their distribution when temperature is elevated [11,55]. At higher temperatures, more severe heat can cause activation/exposure to a specialized checkpoint at the pachytene stage and lead to chromosome defects/meiotic progression defects [56]. In recent studies, *Brassica rapa* has been used to link the roles of homologous recombination and DNA repair to maintenance of male fertility under heat stress, with *BrDMC1* identified as a gene involved in determining pollen meiotic tolerance [33]. These results go beyond the classical heat shock factor/heat shock protein pathway, demonstrating that genome maintenance during meiosis is also a thermotolerance trait.

Tetrads and early microspores are also very sensitive. A transient heat stress at the tetrad stages in maize causes damage to anthers, slows the degeneration of the cell layers surrounding the anther, stunts starch production in the anther, disrupts mature pollen germination, and leads to male sterility, while the vegetative growth of the plant seems to recover after the heat stress [7]. For common bean, the differences in (disruption of) microsporogenesis between heat-sensitive and heat-tolerant genotypes suggest that this developmental sensitivity is to a great extent genetically controlled [16]. The susceptibility of male reproductive tissues is also stage-specific, with *HsfA2*-dependent protection highlighted during stage-specific times such as meiosis and microspore formation [31]. The present study utilizes cell-type-specific proteomic analysis to show that pollen mother cells and microspores also exhibit different heat responses; further, even nearby developmental stages bear different responses.

Programmed degeneration of the tapetum at the right time is an important factor in this vulnerability, requiring the transfer of metabolites, lipids, enzymes and pollen-wall precursors to the developing microspores. Coordinated involvement of sporophytic anther tissues and developing gametophyte in male fertility is highlighted by perturbing *bHLH142* genes in rice marked by tapetal degeneration, anther-wall development, pollen maturation, metabolic gene expression and dehiscence [37]. A naturally occurring temperature-sensitive allele of *OsMS1* also affects the development of the tapetum and the formation of the pollen exine and could be a source of thermosensitive genic male sterility TGS [57]. Heat stress may not only damage pollen but also cause sterility by disrupting the developmental program that supports anther tissues [36,57].

Pollen has to get quite a lot of its metabolic materials ready for maturation. As microspores mature, they require carbohydrates, lipids, amino-acids and proteins—which

are needed for development—from neighboring tissues, and mature pollen must store enough reserves for germination and rapid tube growth [50,58]. For maize, heat exposure at the tetrad stage causes permanent alterations in starch production and the metabolic profile of mature male pollen [7]. The general metabolic profile of various developmental stages of tomato pollen undergoes significant changes towards maturity, and these changes are also temperature-dependent and different among the various pollen developmental stages and genotypes [58]. Another potential effect of brief extreme heat on blueberries is the alteration of pollen proteins, carbohydrates, and amino acids in a stage-dependent manner, and this is linked to lowered subsequent reproductive performance [59]. From the results, it can be seen that the thermotolerance of pollen is not only about survival; it is also about maintaining metabolic quality.

However, the viability of pollen is not a complete measure of reproductive capacity. The fertilization process relies on the hydration, germination and elongation of the pollen tube, which must be polarized; the tube must traverse the pistil, and the ovule must be targeted for fertilization; the sperm must be delivered; and all of these processes involve moving pollen tubes and sperm across cell membranes. These are processes that begin with cytoskeletal dynamics and are regulated by the presence of calcium gradients, ROS, cell wall remodeling, and energy metabolism [45–47]. Even with high relative viability staining, thermal stress can affect the tubes' ability to grow and cause yield differences among cultivars to exist in the absence of distance observations [60]. Redox homeostasis, mediated by flavanols, enhances tomato pollen germination and elongation under high-temperature conditions, highlighting its essential role after the release of the pollen [47]. Furthermore, the high temperatures induced by higher pollen tube performance are correlated with high fruit and seed yields of the thermotolerant germplasm of tomato crops [32]. Pollen formation occurs before fertilization, and the preliminary stages of this process must be carried out within a reproductive tolerance framework.

The female reproductive tissues also produce factors that determine heat resistance. The stigma, style, ovary, ovule and embryo sac are of importance in pollen capture, hydration, guiding the pollen tube, fertilization and early seed development; however, these parts could be defective, independently from pollen [49,61]. The reciprocal cross and transcriptomic experiments in *B. napus* reveal that short-term heat negatively affects pistil support for pollen adhesion, germination and tube progression while significantly inducing large-scale transcriptional reprogramming of the female tissues [38]. Based on direct comparison in *B. napus*, the female and male organs may be equally sensitive or more sensitive in the case of the female [62]. In rice, the females are more thermotolerant than males, and damage to the female reproductive organs due to heat during gametogenesis results in a severe impact on yield even if the male organs remain only partially affected. Stress during gametogenesis has a severe impact on yield even if male organs are not damaged completely [39]. Heat-sensitive pistils are later damaged and are more likely to suffer oxidative injury and are less responsive and/or less likely to form seed [12].

It is known that redox homeostasis is a key physiological cross-point for normal reproduction and heat injury. The production of these ROS is controlled for pollen germination and growth of the pollen tube, but excess production of these ROS causes damage to membranes, proteins, nucleic acids and organelles [47,51]. ROS and the products of lipid peroxidation occur in the anthers at higher levels during heat stress in rice, and differences in the responses of the superoxide dismutase isozyme are correlated with differences in pollen viability and in floret fertility [43]. Among reproductive tissues, heat-tolerant tissues demonstrate higher antioxidant behavior compared to susceptible tissues, and oxygenation ability is in accordance with reproductive survival [12]. Ca^{2+} entry through cyclic nucleotide-gated channels plays a role in thermosensing, and pollen-

specific channels are essential for its stress tolerance and polarized reproductive growth, both at the signaling level [41,45,46]. As these ions and radicals are also developmental signaling factors, resilience is more about “control” (both the spatial and the temporal) than “suppression” [63,64].

Another physiological basis for reproductive thermotolerance is protein homeostasis. It can be assumed that the development of anthers is quickly activated when heat stress occurs, leading to the recruitment of HSF/HSP networks. The anthers of *B. napus* displayed a significant augmentation within minutes after heat stress of endoplasmic-reticulum protein-processing and unfolded-protein-response pathways [44]. ERdj3B is a translocase that functions in quality control to promote anther development at high temperatures, directly linking ER proteostasis with male fertility in *Arabidopsis* [48]. Other key findings were that higher genetic susceptibility to heat stress resulted in greater resistance to autophagy and the ubiquitin–proteasome process, which indicates the removal and recycling of damaged cellular components and hence reproductive survival [12]. Therefore, the mechanism of reproductive thermotolerance relies on proper coordination between protein stabilization, refolding mechanisms, protein quality control and degradation of proteins within the organ.

These cellular processes are correlated with developmental decisions by hormonal homeostasis. During early reproductive development of rice, heat induces changes in the levels of certain hormones in the plants, including a reduction in active cytokinins, auxin and GA levels, and an increase in ABA; all these changes are accompanied by impairment of anther dehiscence, reduction in pollen vigor, decreased spikelet fertility, and reduced grain yield [42]. Partial rescue with cytokinin treatment assists a functional role of hormonal imbalance instead of just the correlative advantage [42]. Conversely, oxidative signaling by stress is regulated by ABA, ethylene, salicylic acid, and calcium, and as such, the pathways are intricately linked together [63].

Even after gamete fusion is successful, post-fertilization development remains vulnerable. Transient thermal exposure in earlier reproductive stages of *B. napus* decreases the ability to form seeds and pods [8], and extended exposure to high temperature leads to changes in developmental programs and hormonal processes of embryos and seeds [40]. Reproductive heat affects canola yield and fatty-acid composition of the seeds, with evidence of changes to flower opening after high night temperature during the flowering stage and changes in yield and seed fatty-acid composition after high reproductive heat during pod filling [65]. Moreover, the transcription profiles of heat-resistant and heat-sensitive rapeseed cultivars through time further reveal that reactions to heat stress involve other gene families, such as heat shock protein, DNA repair, antioxidant, and developmental-related functions, which are also active starting from flowering up to young-pod stages [66]. Reproductive resilience, then, should apply to the entire pathway from initial gametogenesis through to successful sink establishment and go beyond the fertilization point. The stage-specific effects of reproductive heat stress reported across representative crop and model species are summarized in Table 1, highlighting both conserved reproductive vulnerabilities and species-specific responses to elevated temperature.

Table 1. Stage-specific reproductive responses to heat stress in representative plant species.

Plant Species	Heat Stress on Plant Part/Stage	Major Effect Reported
<i>Arabidopsis</i> (<i>Arabidopsis thaliana</i>)	Male meiosis; 30–32 °C heat treatment	Heat altered crossover distribution, impaired synapsis, generated univalents and abnormal recombination, disrupted meiotic cell-wall formation and produced dyads/triads through meiotic restitution [55].

Table 1. Cont.

Plant Species	Heat Stress on Plant Part/Stage	Major Effect Reported
Barley (<i>Hordeum vulgare</i>)	Pollen mother-cell meiosis and pollen mitosis during floret development	Heat caused abnormal anther development, reduced pollen number, viability and starch accumulation, and lowered seed set; sensitivity varied strongly among cultivars and developmental stages [67].
	Pollen mother cells and developing microspores	High temperature disturbed early male gametophyte development and promoted pollen sterility, establishing the PMC/microspore period as a major thermal-sensitive window [68].
Maize (<i>Zea mays</i>)	Tetrad stage of male gametophyte development	A transient heat episode disrupted anther/pollen metabolism, reduced starch accumulation and enzyme activity, impaired mature pollen germination, and ultimately caused male sterility [7].
Rapeseed/canola (<i>Brassica napus</i>)	Mature pollen and pistil (stigma + style) during flowering; 40 °C short-term heat	Heat reduced pollen viability and germination but also directly impaired the ability of the pistil to support pollen germination and tube growth; reciprocal crosses showed reduced seed set and yield from both male- and female-side injury [38].
Rice (<i>Oryza sativa</i>)	Developing anthers exposed to short acute heat	Heat caused extremely rapid transcriptional reprogramming, strongly activating HSF/HSP and unfolded-protein-response/proteostasis pathways, demonstrating the importance of rapid protein-quality control during reproductive heat stress [44].
	Flowering/anthesis; spikelets exposed to high daytime temperature	Spikelet fertility declined strongly above the critical temperature range; genotypes differed in sensitivity, demonstrating that anthesis is a major reproductive thermal bottleneck [5].
	Early reproductive development, including panicle/anther development	Heat altered auxin, cytokinin, GA and ABA homeostasis, reduced anther dehiscence and pollen vigor, and decreased spikelet fertility and grain yield [42].
	Developing anthers during meiosis	Heat increased ROS and lipid-peroxidation damage; differences in SOD activity were associated with variation in pollen viability and floret fertility among genotypes [43].
Sorghum (<i>Sorghum bicolor</i>)	Reproductive development around panicle emergence, sporogenesis and flowering	High temperature decreased pollen viability and floret fertility, resulting in reduced seed set and grain yield; reproductive stages were considerably more sensitive than later developmental periods [69].
Soybean (<i>Glycine max</i>)	Flowering; developing anthers and pollen	Heat reduced pollen germination, altered pollen anatomy, produced a thicker/deformed exine and disrupted tapetal development, ultimately reducing pod set [15].
Tomato (<i>Solanum lycopersicum</i>)	Developing anthers and pollen under prolonged elevated temperature	Heat disrupted the normal transient accumulation of starch in developing pollen and altered soluble carbohydrate concentrations in anthers, contributing to reduced pollen viability [70].
	Male reproductive tissues, particularly meiosis and microspore formation	HsfA2 was required for effective reproductive thermotolerance; suppression particularly reduced pollen viability and germination when heat occurred during early pollen development [31].
Wheat (<i>Triticum aestivum</i>)	Floral development, especially meiosis, pollen development and anthesis	Heat disrupted pollen development and ovary function; reproductive-stage stress reduced floret fertility and subsequent grain number. Classic studies also showed abnormal sporogenesis and heat-induced male sterility [71].
	Pre-anthesis through anthesis and early grain formation	Floret fertility was especially sensitive to heat around reproductive development, whereas later heat increasingly affected individual grain weight; duration and temperature threshold strongly determined the magnitude of yield loss [72].

3. Molecular Regulation of Flowering and Reproductive Heat Resilience

Flowering responses to temperature occur through developmental thermosensing pathways, whereas reproductive protection under damaging heat involves stress-response mechanisms [18,19,31]. However, a considerable fraction of the molecular evidence for thermosensory flowering relies on moderate ambient warming, while flowering as a result of a heat injury usually requires temperatures high enough to induce a proteotoxic, oxidative, genomic and metabolic stress [20,44,56]. One of the big challenges, then, is to understand how developmental temperature sensing relates to protective stress signaling when plants are acclimating to “shifting from warm” to “damaging heat”.

Phytochrome B is an important crossing point for environmental integration since its state of signaling depends on both light and temperature. In Arabidopsis, phytochromes serve as thermosensors and daytime temperature perception via phyB is mediated by the PIF4 pathway, which is activated by HEMERA [22,28]. ELF3 is another thermosensory mechanism, with its prion-like domain that has temperature-dependent behavior and natural variation in the domain that affects thermal responsiveness [23]. The results indicate the use of more than one thermosensor in plants, distributed rather than concentrated in a single site.

PIF4 is one of the most important molecular connections between temperature and floral transition. The activation of FT by PIF4 is improved under warmer conditions in the surrounding environment, and PIF4 interacts with the FT promoter under conditions that promote a faster flowering time [19]. This pathway is not the only one in place. PIF4 and ELF3 are able to contribute to the thermoresponsive flowering pathway in a partial, independent manner from each other; the photoperiodic and thermosensory pathways converge in CONSTANS, under specific day lengths [21,24]. The interactions give a mechanistic explanation as to how the same temperatures can elicit very different flowering responses in different light environments, and at different times of day (circadian timing).

The FLM-SVP module is a supplement that delivers a temperature-dependent repressor system. FLM transcript modulation and abundance of FLM variants binding to SVP are temperature-dependent, potentially causing modifications to the repression of floral integrators [20,25]. At the same time, nonsense-mediated decay helps regulate the levels of FLM transcripts, and CDKG2 affects thermosensitive FLM alternative splicing [26,27]. Under temperature variation, this interaction between CRY2 and CIS1 is also observed, which is involved in coupled photoreceptor signaling and FLM splicing [29]. These studies demonstrate that regulation of thermosensory flowering not only occurs at the level of transcription factor activity but also involves the processing of mRNA and mRNA surveillance.

HSFs orchestrate a key transcriptional defense program once the temperature is “devastating”. Plant HSFs are divided into different functional classes that control heat responses in early events, amplification of stress signals, acquired thermotolerance, and recovery [73,74]. Arabidopsis HsfA1 factors function as significant positive regulators in heat-shock responsive transcription, while HsfA2 is required for prolonged acquired thermotolerance and is involved in developmentally specific stress responses [75–77]. This hierarchy of heat stress is also reproduced in tomato, in which the high silence of HsfA2 drastically affects pollen viability during heat stress at the meiosis and/or microspore development stage, leaving the subsequent growth of the microgametophyte unaffected [31]. The classic pollen observation that pollen shows a heat-shock response further bolsters this idea that gametophytic proteostasis is a direct component of reproductive heat tolerance [78]; gametophytic heat tolerance is also linked to reproductive longevity in pollen and leaf.

These networks can turn on in reproductive tissues very quickly. The number of HSFs and family members of HSP100, HSP90, HSP70, HSP60, and small-HSP are significantly induced in developing *B. napus* anthers within minutes under high-temperature condi-

tions [51]. The same experiment shows that ER unfolded-protein response (UPR) and ER protein processing pathways are activated swiftly in parallel, suggesting that the onset of cytosolic chaperones and ER quality control pathways occurs simultaneously [44]. ERdj3B is involved in anther development at high temperatures in Arabidopsis, and it has been shown that ER-associated quality control is not a generic process in leaves, but essential for reproductive processes [48]. Another connection between ER stress and thermotolerance is the reproductive-stage bZIP17 pathway, which is involved in heat tolerance in the reproductive stage of Arabidopsis [79].

These transcriptional networks are downstream and parallel to calcium signaling. Cyclin nucleotide-gated channels are involved in thermal sensing and acquired thermotolerance, and CNGC14 and CNGC16 function to impart heat and chilling tolerance in rice [41,80]. In pollen, CNGC16 plays a role in reproductive stress tolerance, and a related cyclic nucleotide-gated channel is instrumental in the polarized growth of the pollen-tube [45,46]. There is also a calcium signaling interaction between ROS and transcription responses; for instance, the calcium is required in an annexin-mediated way for the MYB30-dependent ROS and heat responses [64]. These findings are relevant since they draw a parallel between general heat sensing and the same calcium machinery that is crucial for successful reproduction.

The regulation of ROS is also embedded and not just protective. Although high oxidative stresses cause anther damage in rice, leading to decreased fertility, low ROS signaling induces pollen-tube renewal [43,47]. The high antioxidant and autophagic activity of anthers and pistils are responsible for the high thermotolerance in rapeseed, while excessive ROS production during germination and tube elongation is responsible for the low thermotolerance in tomato [12,47]. ONAC023 of rice is also induced by stress signals, and under this condition ROS homeostasis also regulates other processes such as protein function, water transport, and alternative splicing, suggesting that the higher-order transcription factors could link multiple protective modules.

Heat also impacts reproductive development, beyond just canonical stress pathways, by disrupting key developmental genomes. In rice, the bHLH142 gene regulates the development of the anther wall, metabolism, pollen maturation and dehiscence [37]. A natural conditional variant of OsMS1, also known as their temperature-sensitive variant, regulates tapetal cells ATP-formation and pollen-wall development [57], and BrDMC1 is involved in the regulation of meiotic thermotolerance of the recombination machinery in Brassica rapa [33]. HSF2a is also involved in gametophyte development and regulated by an antisense long non-coding heat-induced RNA that shows point-in-time interactions of heat signaling with developmental regulation of reproduction in plants [81]. In recent years, a tomato flower-bud expression atlas was established and a lncRNA-ERF regulatory axis was identified that is linked to basal reproductive thermotolerance [82]. All this evidence suggests that a combination of stress response genes and developmental regulators can shape reproductive heat resilience at the interface.

This network interpretation is backed up by transcriptome-wide studies. Heat exposure induces extensive expression changes in the reproductive stage in rice panicles [83], and comparisons between heat-tolerant and heat-attenuated rice varieties show that they have different transcription programs in anthesis [84]. Genotype-dependent mechanisms involved in higher temperature tolerance in rice are identified using integrated physiological and molecular approach [85]. A comparison of *B. napus* pollen/pistil specific transcriptional responses in response to short heat stress shows that both tissues undergo heat-stress-specific changes in transcript abundance [38], and minute-scale profiling of developing anther shows rapid activation of heat stress factors/heat shock proteins and UPRs [44]. When comparing the genomic profiles of the same hybrid flower or young

pod with each other over time, DNA-repair, anti-oxidative, and developmental differences emerge and distinguish resilient cultivars from those that are sensitive [66]. These findings show that both the time and tissue specificity and the duration of molecular responses, as well as magnitude, may be as critical as the intensity.

Vegetative thermotolerance genes and molecular pathways may be present in the landscape of the genetic context in which reproductive resilience is enacted, but their importance needs to be demonstrated and cannot be assumed. Natural alleles of one of the subunits of the alpha-complex of the 20S proteasome contribute to thermotolerance and climatic adaptation in African rice [86]; alleles of SLG1 confer high-temperature tolerance in indica rice [87]; natural variation in the gene TT2 and in a genetic module involved in protecting the chloroplast contribute to thermotolerance and adaptation to climatic conditions in rice [88,89]. These thermotolerance sites serve as valuable illustrations of naturally occurring mechanisms of thermotolerance, but breeding for more stable yields at the flowering stage will necessitate testing whether this same protection extends to the reproductive organs and final fertility. Collectively, these pathways form an interconnected molecular network in which thermosensing and early stress signaling converge with proteostasis, redox regulation, hormonal control, carbohydrate metabolism, and genome maintenance to protect male and female reproductive development under heat stress (Figure 2).

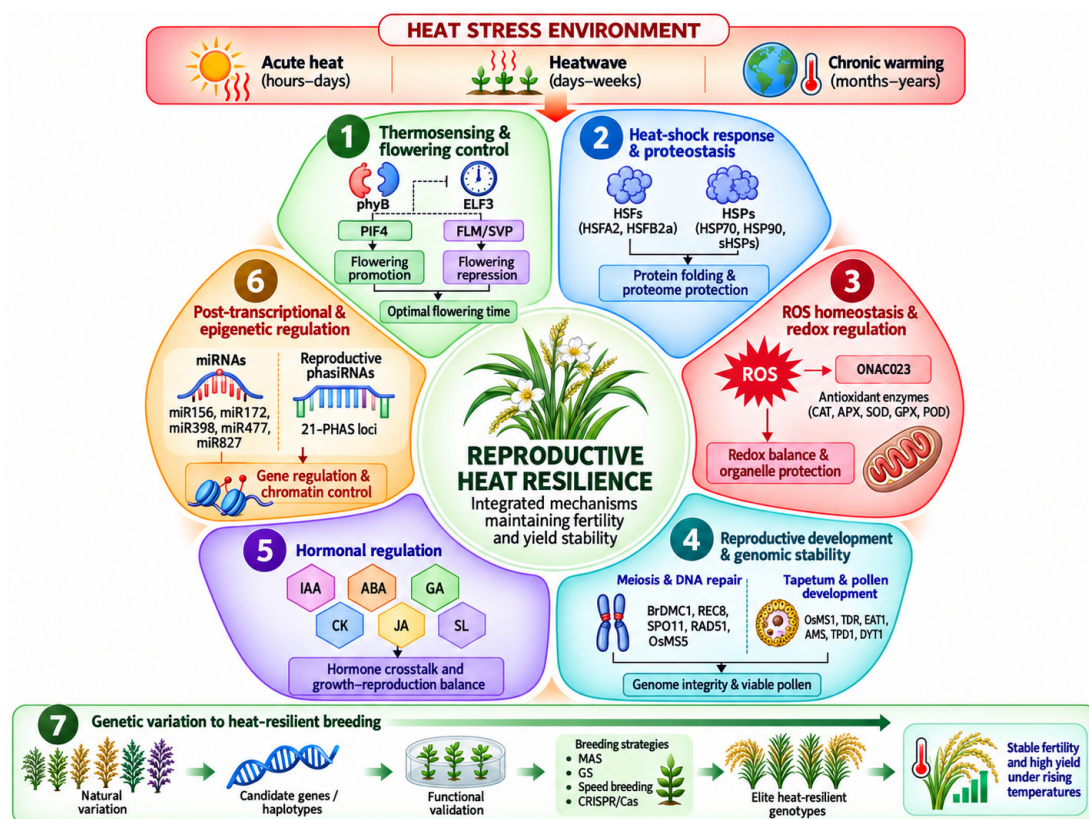


Figure 2. Molecular and genetic mechanisms supporting reproductive thermotolerance under heat stress. Temperature perception and early signaling through thermosensory, Ca^{2+} , ROS, and MAPK pathways activate interconnected protective systems involving HSF/HSP-mediated thermoprotection, unfolded protein response and proteostasis, antioxidant regulation, hormone signaling, carbohydrate metabolism, and DNA repair. These processes protect heat-sensitive male functions, including meiosis, tapetal development, pollen maturation and pollen-tube growth, as well as female functions involving stigma receptivity, style support, ovule viability, and fertilization. Their coordinated activity contributes to maintenance of fertility and subsequent seed or fruit formation under heat stress. Generated with the assistance of ChatGPT (OpenAI, San Francisco, CA, USA; GPT-5.6 Sol) and subsequently edited by the authors. The authors reviewed and verified the final figure for scientific accuracy.

4. Genetic Architecture of Flowering and Reproductive Heat Resilience

Flowering time, gametophyte development, cellular protection, pistil function, seed development and final yield are genetically complex traits that can segregate in part exclusively [34,35]. However, genetic studies without information on flowering time can be misunderstood as showing apparent tolerance for loci that only shift flowering time when stress is applied [30]. Reproductive traits without final yield in contrast can reveal physiological protection that does not necessarily relate to agronomic resilience [32,60]. A pragmatic gene-by-gene framework is therefore established for phenological exposure, intrinsic reproductive tolerance and yield conversion.

The first layer is natural variation in thermosensory flowering. The first layer is natural variation in flowering in response to thermal conditions. There are molecular pathways that can converge on allelic variation (PIF4–FT, FLM–SVP, phyB, ELF3, CRY2 and associated circadian and photoperiodic regulators), which impacts the temperature control of flowering [19,20,22,23,29]. Not only do thermosensing domains vary in being cold or sensitive (as shown above), but temperature responsiveness can also be genetically variable in ELF3, which is evident from the presence of natural variation in the ELF3 prion-like domain [23]. However, in crops, flowering networks have been modified through domestication, vernalization requirements, photoperiod adaptability and polyploidy, and the validity of mechanisms forming part of the model systems cannot be assumed but needs to be tested experimentally [18,30].

Phenology genes can be used for heat escape in marker-assisted breeding. A marker-assisted breeding study [30] demonstrated that the introgression of early-anthesis and high-kernel-weight QTLs in bread wheat resulted in shifting the flowering time and improving performance under terminal heat condition. It is a successful adaptation but it is mainly an exposure change, and, it does not demonstrate heat tolerance of pollen, pistils or meiocytes [30]. Using the introgression lines of *Triticum durum* x *Aegilops speltoides*, the thermotolerance of multiple heading-, maturity-, and yield-related traits have been found to tolerate heat. This shows that wild relatives such as valuable loci can expand the genetic basis of thermotolerance [34]. Stable QTLs across heat environments are especially desired as they can be drivers of resilience in more than one season.

The second layer in the genome is for those loci that directly affect reproductive development under temperature stress. For example, in *OsMS1*, it has been found that a natural allelic variant causes temperature-dependent male sterility by inhibiting the development of tapetum and the formation of exine in pollen grains [57]. *BrDMC1* shows that meiotic variation in recombination and DNA restoration tools can impact meiotic survival under heat [33]. *Onac023* also reflects a new class of regulatory strategy: the activity of specific downstream pathways related to ROS homeostasis, protein functions, water transport and RNA processing upon stress-induced nuclear translocation led to enhanced rice heat tolerance at the reproductive stages [90]. These illustrations demonstrate various kinds of reproductive thermotolerance: developmental, genome maintenance, and stress regulatory.

Numerous physiological components are also part of the genetic diversity in whole-crop performance under heat. There are variations in the heat sensitivity of anthesis, spikelet fertility, proteomic responses, hormone homeostasis, and transcriptomic regulation among rice cultivars [10,42,84,91]. While night temperature and grain filling response are influenced by natural genetic diversity, higher night temperature lowers rice yield [92], and high day or night temperature affects yield formation by affecting the reproductive and grain development processes [93]. High grain-filling temperatures affect the grain quality through starch metabolism, amino-acid metabolism and gene expression [94–96]. For these post-fertilization properties to be an important component of the genetics of

reproductive ability, they should also be included in genetic definitions of reproductive resilience (assuming that a stable seed set alone is not enough for yield stability).

Given that heat response is polygenetic, genomic prediction is appealing for this problem. For tomato, genomic prediction under high temperature was meaningful for yield prediction and soluble-solids and resulted in useful regions of the genome involved with thermotolerance-related performance [35]. This strategy will catch many small-effect loci that affect flowering, pollen function, fruit set, metabolism and yield, all of which are developing in a developmentally relevant heat regime. The lack of correlation between pollen viability and yield in the different tomato cultivars tested clearly supports the argument against using any single intermediate phenotype as a predictor of ultimate yield [60].

There is cross-species evidence and support for trait-specific genetic architectures. In heat-sensitive and tolerant common bean genotypes, differences exist in the processes of microsporogenesis [16], differentiation of pollen anatomy, and semen fertility [15], and there are differences in threshold for the pod set and pollen fertility at the seed stage in grain sorghum [17], indicating differences in genotype. Brassica species have a high reproductive heat response [14] and canola pollen germination has a genotype- and temperature-dependent response curve [97]. These findings contradict a common heat-tolerant phenotype so as to give more support for the species-specific identification of the limiting process to yield.

5. Linking Phenological Adaptation, Reproductive Thermotolerance, and Yield Stability

The main biological question is represented as two consecutive decisions with a developmental consequence. Flowering pathways are thermosensory, meaning that they regulate reproduction in relation to the seasonal temperature [19,20,23]. Second, the molecular and physiological interaction between damaging heat and meiocytes, anthers, pollen, pistils and ovules determines whether they are functional or not [31,33,38,39]. Third, the successful fertilization has to be turned into viable embryos, seeds, grains or fruits by continued thermal exposure [8,40,66]. Yield stability under heat therefore depends on the coordinated contribution of phenological adaptation, maintenance of reproductive function during damaging temperatures, and successful post-fertilization development. The relative contribution of these processes is expected to vary with genotype, developmental stage, heat intensity and duration, and environmental context.

Heat escape works where there is extreme temperature that is predictable during a given season. Earlier anthesis can shift wheat reproduction away from the terminal heat and boost wheat grain performance [30]. However, an escape-only approach is restricted by the variability of climate because of the possibility of heatwaves occurring earlier than anticipated or lasting longer than the altered reproductive window. Transient heat during the tetrad-stage in maize reveals how an unexpected short heat can cause permanent loss of fertility [7], and transient heat in the reproductive stage in *B. napus* leads to a decrease in the fecundity of later developing florets and pod production [8,53]. Intrinsic reproductive protection thus acts as a protection insurance if phenological avoidance is not successful.

Preserved reproductive function should be the basis for intrinsic thermotolerance, not cell survival. There is a need for recombination and chromosome segregation to occur in the meiocyte [33,55], a tapetum for supporting pollen development and degradation at the right time [37,57], tube growth in pollen grains [32,47], and a receptive pistil to facilitate pollen–pistil interactions [38,62]. The results, showing that pollen viability does not always correspond to yield in tomato, highlight the need for multiple screening endpoints [60].

This is also true following fertilization. High temperatures during embryo and seed development can lead to a decrease in yield even after flowering and fertilization are complete [40]. In rice, high night temperature during grain filling decreases yield [92], and high temperature during grain filling results in disruption of starch metabolism and grain quality [94–96]. In the case of canola, high night temperatures during flowering and pod filling increase yield and seed composition [65]. Consequently, there are several checkpoints in the physiological pathway from floral initiation to production of marketable yield that are sensitive to heat stress.

Taken together, these observations support a conceptual rather than mathematical framework in which reproductive heat resilience emerges from the coordinated contribution of three partially separable dimensions: phenological optimization, intrinsic reproductive thermotolerance, and post-fertilization physiological stability. Their relative importance is context-dependent and may vary among species, genotypes, developmental stages, and thermal environments. A limitation in one dimension can become yield-limiting under a particular heat scenario; for example, earlier flowering cannot prevent sterility if an unexpected heatwave coincides with meiosis, pollen thermotolerance cannot compensate for severe heat injury to the pistil, and successful fertilization cannot ensure yield when subsequent embryo or seed development is impaired [7,38,40]. These dimensions should therefore be regarded as interacting biological processes rather than components of a fixed algebraic relationship. Robust reproductive adaptation is expected to require coordinated improvement of flowering-time adaptation, reproductive tissue protection, and post-fertilization sink stability [30,66,90].

6. Translating Molecular and Genetic Knowledge into Crop Improvement

The first step in breeding for reproductive heat resilience is to correctly phenotype the traits involved. The date of flowering or heading should be noted to differentiate between escape and intrinsic tolerance [30,34]. The stage-specific evaluation of the meiosis or gametogenesis, anther dehiscence, pollen viability and viability of germination, pollen-tube performance, stigma receptivity, fertilization, seed or fruit set, and final yield should all be performed in reproductive screening [32,54,61]. Screening efficiency can be achieved by implementing high-throughput pollen protocols that could reduce labor and costs, but the collection of pollen should continue to be correlated with the whole-plant reproductive outcome [54,60].

Developing stages are also vital. In tomato, dependence on HsfA2 varies during male reproductive development [31]; in maize, persistent sterility occurs if the heat occurs during the tetrad stage [7]; in the case of rapeseed, if heat occurs during gametophytic development, the fecundity of later stages is reduced [53]. Heat applied to less well-defined developmental periods can then create a mixture of mechanistically different phenotypes and diminish the power for mapping. This suggests that accurate staging needs to be incorporated into QTL, GWAS, genomic selection and gene-editing experiments.

Rather, marker-assisted breeding can still be used, provided large-effect loci are available or data on phenology exist [30]. Genomic selection has the potential to identify polygenic variation scattered across the genome that is important for final performance under heat stress, while heat-adaptive alleles may be introgressed into elite backgrounds from wild relatives [34]. The best breeding designs involve cross-combining optimum flowering time with meiosis protection, pollen, pistil function and post-fertilization development protection rather than just early flowering.

To translate these mechanistic findings into breeding decisions, candidate genes and loci should be matched to the specific component of heat resilience that they influence. Thermosensory flowering regulators such as PIF4-FT, FLM-SVP, ELF3, and phyB are principally

relevant to phenological optimization and may provide targets for selection of favorable natural alleles or, following crop-specific validation, precise modification of regulatory regions [19,20,22,23]. In contrast, HSF/HSP components, ONAC023, OsMS1, and BrDMC1 are more directly associated with intrinsic thermotolerance through proteostasis and stress regulation, tapetal and pollen development, or meiotic genome stability [31,33,48,57,90]. Natural heat-adaptive loci such as SLG1 and TT2 provide additional candidates for marker-assisted introgression or genomic-selection models [82,83]; however, because much of their current evidence concerns broader plant thermotolerance, their effects on reproductive-stage fertility and yield stability should be directly validated before they are classified as reproductive thermotolerance loci. Similarly, stable heat-responsive QTLs identified in wheat and wild-relative introgression material can support marker-assisted breeding when their effects are reproducible across environments [30,34], whereas highly polygenic variation affecting final yield under heat is more appropriately captured through genomic selection [35].

Considering reproductive heat resilience as a multi-omics attribute is of particular value, as this is produced by interactions among transcription, protein quality control, metabolism, redox regulation and development. We have seen unique tolerant and sensitive programs in rice [84] and in rapeseed [66], identifying cell-type specific heat responses in the male reproductive cells of tomato [98] and stage-specific changes in the metabolome of tomato and blueberry pollen [58,59], respectively. These datasets can be mapped out with genetics to differentiate generic stress responses from those that could predict fertility and yield.

Mechanistic resolution should be further improved with the use of single cell and spatial approaches. Meiocyte, microspores, tapetum, endothecium, epidermis and connective tissues are present in whole-anther transcriptome and stigma, style, ovary and ovules in whole-pistil transcriptome [38,98]. It is likely that the first step of thermal failure is unnoticed in bulk measurements, given the recent proteomic and genetic thermal specificity and cell-type specificity that were discovered [31,33,98]. Spatial resolution of these answers should lead to the establishment of more cell-specific biomarkers and regulatory targets with breeding value.

7. Future Perspectives and Conclusions

Despite substantial progress in identifying the physiological and molecular determinants of reproductive heat responses, several fundamental questions remain unresolved. Addressing these questions will be essential for moving from descriptive heat-stress phenotypes toward mechanistically informed and environmentally robust crop improvement.

A major unresolved issue is how thermosensory flowering networks operating under moderate temperature interact with cellular protection pathways activated by damaging heat. PIF4-FT, FLM-SVP, phyB, ELF3, and related photoperiod-circadian regulators influence the timing of reproductive transition, whereas HSF/HSP systems, ER protein quality control, calcium/ROS signaling, autophagy, and DNA-repair pathways become increasingly important when temperature becomes damaging [12,19,22,23,25,29,31,33]. It remains unclear which molecular nodes directly connect these two temperature-response regimes and whether such connections are conserved across crop species. Determining whether thermosensory flowering genes influence intrinsic reproductive thermotolerance, rather than only modifying heat exposure through flowering time, should therefore be a priority.

A second major question concerns the identity of the earliest heat-sensitive cell types and developmental processes within male and female reproductive organs. Much of the literature has emphasized pollen and anther responses, although reciprocal-cross

experiments and physiological analyses demonstrate that pistils, ovules, and other female tissues can independently constrain fertility under heat stress [38,39,61,62]. Future studies should therefore combine precise developmental staging with reciprocal crosses and cell-type-resolved approaches. Single-cell transcriptomics, spatial transcriptomics, proteomics, and metabolomics could help determine whether heat failure begins in meiocytes, tapetal cells, microspores, stigma tissues, ovules, or other reproductive cell populations before it becomes detectable at the whole-organ level.

A third challenge is to identify reproductive biomarkers that reliably predict final yield. Pollen viability and related laboratory measurements are useful screening traits, but their relationships with fruit or seed yield can vary among genotypes [32,60]. Future studies should determine which combinations of cytological, physiological, molecular, and reproductive traits provide robust prediction of fertility and yield under heat. This will require standardized heat treatments defined by developmental stage, temperature intensity, duration, time of day, and recovery period, followed by validation across genotypes and environments.

The genetic causality and agronomic value of candidate heat-resilience loci also require stronger validation. Genes such as ONAC023, OsMS1, and BrDMC1 provide mechanistically informative examples of stress regulation, temperature-dependent reproductive development, and meiotic stability [34,58,85]. Other natural heat-adaptive loci, including TT2 and SLG1, represent potentially useful breeding resources [82,83], but their contributions specifically to reproductive-stage thermotolerance remain to be established. Functional validation using near-isogenic lines, allele replacement, cis-regulatory editing, or tissue-specific manipulation will be important for distinguishing direct reproductive protection from broader effects on plant development or vegetative thermotolerance.

Greater attention is also needed for the environmental realism of reproductive heat experiments. Acute daytime heat, elevated night temperature, repeated heatwaves, and chronic warming can impose biologically different stresses, yet they are often collectively described as “heat stress.” Their effects may also interact with vapor-pressure deficit, water availability, radiation, and developmental history. Controlled-environment experiments should therefore be complemented by multi-environment field studies in which flowering time and reproductive developmental stage are recorded explicitly. Such designs will help distinguish true intrinsic thermotolerance from phenological heat escape and genotype-by-environment effects.

Post-fertilization resilience remains comparatively understudied despite its direct importance for yield stability. Successful pollination and fertilization do not guarantee final yield when heat subsequently disrupts embryo development, endosperm metabolism, seed filling, fruit growth, or assimilate allocation [41,66,87,89–91]. Future genetic and physiological studies should therefore extend reproductive phenotyping beyond fertilization and quantify the capacity to convert successful fertilization into viable seeds, fruits, and harvestable yield under continued heat exposure.

Atmospheric humidity can further modify reproductive heat injury through changes in vapor-pressure deficit, transpiration, floral-tissue water status, pollen hydration, and stigma–pollen interactions. Likewise, photoperiod and light environment can alter the timing and magnitude of thermosensory flowering responses through their interaction with circadian and flowering-time pathways, meaning that identical thermal episodes may produce different reproductive outcomes across latitudes and cropping environments.

Ultimately, reproductive heat resilience should not be treated as a single trait or reduced to a universal definition of “heat tolerance.” Breeding strategies will need to integrate temperature-appropriate phenology, protection of male and female reproductive functions, and post-fertilization stability while accounting for their genotype- and environment-

dependent contributions. Developing validated multi-trait selection indices, integrating stage-specific phenotyping with genomic prediction, and testing candidate alleles across realistic thermal environments represent important next steps toward crop varieties capable of maintaining reproductive performance and yield under increasingly variable climates.

8. Conclusions

Finally, flowering is a developmental stage at which temperature adaptation is directly reflected in plant fitness and crop production. Reproduction is surrounded by a thermal environment defined by thermosensory networks involving phyB, ELF3, PIF4, FT, FLM, SVP, CRY2 and other photoperiod–circadian components [19,20,22,23,29]. Examples of mechanisms that regulate fertility when damaging heat occurs together with reproduction include HSF/HSP pathways, ER proteostasis, calcium and ROS signaling, antioxidant systems and hormone balance, along with either autophagy or developmental regulators, and DNA-repair systems [12,31,33,43,44,90]. These mechanisms can be combined genetically via natural alleles, QTLs, genomic prediction and comparative omics [30,34,35,57,66]. Making mechanically meaningful breeding targets, it is, therefore, not simply a heat-tolerant plant, but a reproductive system that flowers at a suitable time, maintains male and female function at inevitable periods of heat, and achieves a stable yield from successful fertilization.

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