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From Stress Exposure to Survival: Gene Expression Modules Under Heat–Desiccation Extremes in Insects

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A B S T R A C T

Insects increasingly experience climatic extremes as coupled stressors rather than isolated challenges, with high temperatures frequently co-occurring with low humidity, creating compound heat–desiccation events. These hot–dry episodes threaten survival by simultaneously destabilizing proteostasis, elevating oxidative stress, and accelerating water loss, thereby compressing thermal safety margins and amplifying failure risk during and after exposure. This study synthesizes current evidence that insect stress transcriptomes are organized into coordinated gene-expression modules—rather than scattered, independent responses—and that the timing, magnitude, and integration of these modules can predict resilience, collapse, and recovery capacity. This study emphasizes a conserved "core" program centered on molecular chaperones (including HSP families), redox buffering, and damage-control pathways, which is repeatedly recruited under both heat and severe dehydration. Layered onto this core are stressor- and lineage-specific modules that govern water balance and osmotic stability, including cuticular barrier remodeling, transport and excretory regulation, osmolyte metabolism (notably trehalose pathways in extreme tolerance), and protective proteins. It further examines regulatory architectures that tune these modules—stress-activated signaling networks, transcription factors, and epigenetic or chromatin-linked mechanisms that may shape acclimation and short-term stress "memory," while noting that strong evidence for durable, generalizable epigenetic memory remains uneven across insect taxa. Finally, it highlights emerging priorities for the field: field-realistic thermal–humidity trajectories, life-stage- and tissue-resolved sampling, multi-omics integration, and causal perturbation pipelines that move from signatures to mechanisms. Together, a modular framework clarifies why similar transcriptional activation can yield divergent outcomes across species and populations and provides a practical basis for biomarker discovery, conservation physiology, and climate-aware pest management

Introduction

Insects are ectotherms that rely on ambient conditions to regulate body temperature, which makes them especially sensitive to rapid shifts in temperature and humidity (IPCC, 2021; Halsch et al., 2021; Banfi et al., 2025; Guo et al., 2025). Heat stress and desiccation (drying) stress are therefore among the most pervasive and biologically challenging pressures insects experience across habitats (Filazzola et al., 2021; Benoit, 2023). Episodes of elevated temperature can destabilize proteostasis (e.g., protein denaturation and aggregation), impair enzymatic function, and disrupt membrane integrity; these effects are tightly linked to inducible cellular defenses such as

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the heat-shock response and its molecular chaperones (Harvey et al., 2023; Horváth et al., 2023; Abbasi, 2025; Banfi et al., 2025). In parallel, water deficit perturbs osmotic balance and cellular homeostasis, alters metabolic and neuromuscular performance, and can rapidly lead to lethal dehydration in the absence of effective water conservation and tolerance mechanisms (Benoit, 2023). In natural settings, these stressors frequently co-occur—for instance, hot midday conditions in arid or semi-arid environments typically coincide with low relative humidity, producing compound heat and desiccation exposure extremes that can be more physiologically constraining than either stress alone (Filazzola et al., 2021; IPCC, 2021).

When insects experience extreme heat or dehydration, the immediate challenge is to maintain cellular homeostasis while preventing cascading damage to proteins, membranes, and nucleic acids. Because most protective adjustments cannot be achieved quickly enough by physiological mechanisms alone, rapid and coordinated changes in gene expression become central to stress survival. In practice, insects activate partially overlapping transcriptional programs that include (i) molecular chaperones (e.g., HSP70/HSP90 and small HSPs) that stabilize or refold damaged proteins, (ii) water- and ion-transport regulators such as aquaporins that contribute to dehydration-linked water management, (iii) enzymes that support osmolyte synthesis (including pathways associated with trehalose metabolism in highly desiccation-tolerant systems), and (iv) antioxidant and redox-balancing enzymes that mitigate reactive oxygen species (ROS) generated during stress and recovery (Yoshida et al., 2021; Benoit et al., 2023; Banfi et al., 2025; González-Tokman et al., 2025). An overview of the conceptual framework linking combined heat and desiccation exposure to stress-responsive gene-expression modules and downstream survival outcomes is provided in Figure 1.

Because insects underpin essential ecosystem functions—including pollination, nutrient cycling, and biological control—understanding the mechanistic basis of stress resilience is not only fundamental to insect physiology but also central to forecasting biodiversity and ecosystem stability under warming and drying trends (Harvey et al., 2023; Abbasi, 2025). Critically, integrating physiological assays with transcriptomic evidence shows that stress tolerance is often associated with coordinated gene-expression differences (both basal and inducible), helping to pinpoint molecular pathways that distinguish resilient versus vulnerable phenotypes (Horváth et al., 2023). Furthermore, understanding these highly tuned molecular defense systems in insects provides crucial blueprints for developing stress-resilient biomaterials, enhancing pest management strategies against climate variability, and informing predictive models across broader biological engineering contexts where tolerance to simultaneous thermal and desiccation stress is required.

This article focuses on the gene expression modules and regulatory networks that enable insect survival under combined heat and desiccation challenges. This article synthesizes current knowledge of the molecular pathways activated by these stressors, spanning from immediate cellular defenses to longer-term acclimation and inherited stress memory. It discusses key molecular players – from heat shock proteins and transcription factors to epigenetic marks – and their roles in orchestrating stress responses. Special attention is given to how heat and desiccation stresses intersect: do they activate shared protective genes or unique pathways? It also compares mechanisms across different insect taxa and evolutionary contexts, highlighting conserved strategies and species-specific innovations for extreme stress tolerance. The overarching goal is to provide an integrative understanding of how insects modulate gene expression from the moment of challenge exposure to the achievement of survival, and to identify directions for future research in this era of rapid environmental change.

Mechanisms of Heat and Desiccation Stress in Insects

Physiological Responses to Heat Stress

Exposure to high temperature elicits a multi-layered response aimed at preventing and repairing heat-induced injury. A primary acute consequence is protein unfolding and misfolding, which threatens core cellular functions. Insects counter this by increasing production of heat shock proteins (HSPs), which bind destabilized proteins to prevent aggregation and support refolding

during recovery (Banfi et al., 2025). This transcriptional reprogramming is coordinated by heat shock factors (notably HSF1 in metazoans), whose activation and regulation have been extensively reviewed at the molecular level (Akerfelt et al., 2010; Kmiecik & Mayer, 2022).

Heat stress also promotes oxidative imbalance, in part because elevated temperature can increase metabolic flux and perturb mitochondrial function, thereby elevating ROS production. Across insects, antioxidant defenses (e.g., SOD, catalase, peroxidases, and thioredoxin-linked systems) are repeatedly implicated in buffering heat-associated oxidative damage, and recent synthesis highlights how redox regulation integrates with other stress responses under heat (González-Tokman et al., 2025). In addition, stress-activated signaling can shape survival decisions. In *Drosophila*, the JNK pathway has been experimentally shown to play a role in enabling a full heat shock response, demonstrating that canonical stress kinases can be functionally required—not merely correlated—with heat-shock transcriptional output (Gonda et al., 2012). Overall, acute heat survival depends on rapid deployment of proteostasis safeguards (especially HSPs), coupled redox control, and appropriately tuned signaling networks that determine whether cells recover or transition toward apoptosis when damage is irreparable.

Desiccation Stress and Water Homeostasis

Desiccation challenge arises when water loss exceeds water intake, leading to dehydration, hemolymph concentration, and cellular-level osmotic imbalance. Because many insects have high surface area-to-volume ratios, survival often depends on integrating behavioral buffering (microhabitat selection, reduced activity during peak aridity) with physiological control of water balance, as synthesized in recent cross-arthropod reviews (Benoit et al., 2023). A major first line of defense is minimizing transpiration through the cuticle: cuticular hydrocarbons (CHCs) and other barrier traits can reduce cuticular permeability and thereby lower water-loss rate, with recent experimental evidence in Drosophila continuing to support a causal role for CHC-mediated barrier function in desiccation resistance (Nayal et al., 2024). Water balance is further shaped by respiratory water loss and by excretory regulation via Malpighian tubules and hindgut function, which together determine the extent to which insects can conserve water while maintaining ionic homeostasis (Benoit et al., 2023).

At the cellular level, dehydration elevates intracellular solute concentrations and destabilizes macromolecular structure, creating selective pressure for "compatible solute" and protective-protein strategies. Trehalose is a canonical osmolyte in extreme dehydration tolerance, functioning as a stabilizing solute that can protect proteins and membranes during water loss; in the anhydrobiotic midge Polypedilum vanderplanki, trehalose can reach ~20% of dry body mass during slow desiccation, consistent with its central role in anhydrobiosis induction (Cornette & Kikawada, 2011). In parallel, intrinsically disordered protective proteins—most prominently late embryogenesis abundant (LEA) proteins—are strongly associated with desiccation tolerance in highly resistant systems and are encoded by expanded, dehydration-inducible gene families in P. vanderplanki (Gusev et al., 2011; Hatanaka et al., 2015). Importantly, successful dehydration survival is not solely about enduring the dry state: oxidative stress and molecular damage can peak during rehydration, necessitating antioxidant buffering and repair capacity to restore function after water returns (Benoit et al., 2023). Overall, desiccation tolerance is best viewed as an emergent property of coordinated barrier traits, transport/excretory regulation, osmolyte-based stabilization, and damage-control systems—many of which are transcriptionally regulated and interact with conserved stress-response circuitry discussed in Section 3 (Benoit et al., 2023).

Combined Heat and Desiccation Stress

In natural environments, insects are rarely exposed to heat or desiccation in isolation. High temperatures commonly coincide with low relative humidity—especially in arid/semi-arid habitats and during heatwaves—creating compound hot–dry conditions that can constrain performance more strongly than either stressor alone (IPCC, 2021; Benoit et al., 2023). Mechanistically, warming elevates water-loss risk by increasing the vapor pressure gradient between the insect and the air and by accelerating both cuticular transpiration and respiratory water loss, while dehydration simultaneously challenges osmotic balance and can destabilize metabolism and neuromuscular function (Benoit et al., 2023). This coupling matters because dehydration can become a

proximate failure mode during heat exposure, lowering the "effective" thermal safety margin measured under more humid laboratory conditions (Benoit et al., 2023).

At the molecular level, combined thermal and water stress elicits an integrated transcriptional program that draws from both the canonical heat-shock response and dehydration-associated regulatory pathways. Heat-driven components primarily address proteotoxic stress and redox imbalance by rapidly inducing molecular chaperones (e.g., HSP70/HSP90 and small HSPs) and antioxidant defenses, whereas dehydration-associated modules emphasize water conservation (e.g., cuticle and spiracular control), osmolyte metabolism, and membrane stabilization (Benoit et al., 2023). Importantly, these modules are not independent: they converge on a shared "core" of protective functions—proteostasis buffering, oxidative-stress mitigation, and metabolic reallocation—that can be co-activated under compound stress. **As illustrated in Figure 1,** heat and desiccation thus recruit both stressor-specific pathways and overlapping molecular modules (notably HSP induction and antioxidant activity) that jointly support cellular homeostasis under hot–dry extremes.

Experimental evidence further indicates that responses to combined stress can be non-additive and strongly context-dependent, reflecting pathway cross-talk and competition for limited cellular resources. In *Drosophila simulans*, for example, stress-induced plasticity experiments show that hardening responses and survival outcomes depend on the specific challenge history and combination of stressors rather than following a simple additive model (Bubliy et al., 2012). Reviews focused on multi-stressor biology similarly emphasize that cross-protection (or cross-susceptibility) cannot be assumed a priori and should be explicitly tested, as interactive effects can reverse depending on dose, timing, and ecological context (Rodgers & Gomez Isaza, 2021). From an applied viewpoint, this is also why cross-protection has been highlighted as both an opportunity and a complication for pest management under climate variability, because stress interactions can either strengthen or weaken pest resilience in the field (Bueno et al., 2023).

Finally, real-world examples underscore the biological importance of humidity during heat. Foraging honey bees in hot, dry air face intense water-balance constraints and use behavioral and physiological strategies to conserve water while maintaining function, illustrating how "heat stress" in nature is often inseparable from dehydration risk (Glass et al., 2024). Collectively, available evidence supports a modular view: survival under compound thermal and water stress extremes depends not merely on *whether* stress-responsive genes exist, but on the timing, magnitude, and coordination with which shared protective modules (proteostasis and redox buffering) are integrated with dehydration-specific or heat-specific pathways across the exposure–recovery sequence (Bubliy et al., 2012; Benoit et al., 2023).

Gene Expression Modules Under Heat–Desiccation Stress

Stress-Responsive Genes: Heat Shock Proteins and Related Pathways

Heat shock proteins (HSPs) constitute the best-characterized core module of insect stress transcriptomes under acute heat stress and, in many taxa, under severe dehydration. Functionally, HSPs act as molecular chaperones that prevent irreversible protein aggregation, facilitate refolding during recovery, and support proteostasis triage toward proteasomal or autophagic clearance when damage exceeds repair capacity (Akerfelt et al., 2010; Banfi et al., 2025). Across insects, the dominant families include small HSPs (sHSPs), HSP60, HSP70/HSC70, and HSP90, whose transcriptional induction can occur within minutes to hours following proteotoxic challenge (Akerfelt et al., 2010). Importantly, HSP upregulation should not be interpreted as a heat-exclusive signal; proteotoxicity can emerge under dehydration as well, especially when osmotic imbalance and molecular crowding destabilize protein structure.

Evidence from anhydrobiotic systems illustrates how this "core" proteostasis module can be integrated into broader dehydration survival programs. In *Polypedilum vanderplanki*, dehydration robustly upregulates multiple HSP classes (including HSP70, HSP90, and sHSPs), consistent with a strategy in which proteome stabilization is prioritized during progressive water loss (Gusev et al., 2011). Mechanistically, comparative and functional work indicates that the canonical heat-shock regulatory system can be evolutionarily coopted to activate a wider set of dehydration-tolerance genes in this species, linking HSF-centered control to anhydrobiosis acquisition

rather than limiting it to classical heat-shock targets (Mazin et al., 2018). This co-option provides a useful conceptual template: conserved proteostasis circuitry can serve as a regulatory "spine" onto which lineage-specific protective modules are layered (see Section 2.2 for dehydration-specific effectors such as trehalose and LEA proteins).In many insects, HSP induction co-occurs with oxidative-stress control and repair programs, reflecting the coupled nature of proteotoxicity and redox imbalance under thermal and water-stress extremes. Heat and dehydration (particularly during rehydration) can elevate reactive oxygen species (ROS), and transcriptomic studies commonly report enrichment of antioxidant and damage-control pathways alongside HSPs rather than isolated chaperone activation (Rosete-Enríquez et al., 2025). The HSP module also intersects with immune function: in *Drosophila melanogaster*, viral infection can induce a heat-shock transcriptional program, and perturbation of HSF compromises survival during infection, demonstrating that heat-shock circuitry contributes to host defense as well as environmental stress tolerance (Merkling et al., 2015). Collectively, these observations support a modular interpretation of stress transcriptomes in insects, in which the HSP-centered proteostasis response serves as a broadly deployed core module that integrates with dehydration-specific effectors and redox/repair pathways to shape survival under compound extremes (Banfi et al., 2025; Rosete-Enríquez et al., 2025). A concise overview of the major stress-responsive gene/protein families, their roles under heat versus desiccation, and representative insect examples is provided in Table 1.

Transcription Factors and Signaling Pathways

Stress-induced transcriptome remodeling in insects is governed by hierarchical regulatory control, in which upstream signaling converges on transcription factors (TFs) that rapidly reallocate cellular resources toward protection and repair. Heat Shock Factor 1 (HSF1) is the principal activator of the heat-shock response: under proteotoxic conditions, HSF1 activation drives transcription of multiple HSP families via binding to heat shock elements, and negative-feedback regulation (via chaperone re-binding) contributes to response shutdown during recovery (Akerfelt et al., 2010). Evidence from anhydrobiotic models underscores that HSF control can extend beyond "classic" heat targets. In addition to HSF, insects deploy stress-responsive signaling that integrates osmotic imbalance, ROS, and metabolic state. Although the specific pathway architecture varies by species and tissue, a recurring theme is that stress responses are multiplexed: transcriptomic datasets frequently reveal parallel activation of proteostasis, antioxidant, carbohydrate metabolism, membrane/cuticle biology, and repair programs, consistent with broad TF and signaling involvement rather than a single master switch (Rosete-Enríquez et al., 2025; Yamada et al., 2020). Recent work using high-resolution transcriptomic analyses continues to emphasize that extreme dehydration can elicit genome-wide reprogramming in anhydrobiotic taxa, with a regulatory logic that appears temporally structured—an important point for experimental design, as sampling time strongly shapes inferred "key regulators" (Yamada et al., 2020; Hiki et al., 2026). Thus, for heat–desiccation extremes, a defensible working model is that HSF-centered proteostasis control is necessary but not sufficient; survival phenotypes emerge from coordinated regulation that integrates proteome protection, redox buffering, and resource reallocation across time (Akerfelt et al., 2010; Rosete-Enríquez et al., 2025).

Epigenetic Regulation of Stress Responses

Epigenetic mechanisms—DNA methylation, chromatin remodeling, and small-RNA pathways—can shape both the magnitude and kinetics of stress-induced gene expression, potentially contributing to acclimation, plasticity, and (in some systems) longer-lived stress "memory." In insects, DNA methylation is taxonomically uneven and often lower than in vertebrates, but functional links between methylation machinery and thermal tolerance are increasingly supported in hemipteran pests. In Bemisia tabaci, evidence indicates that DNA methylation state and expression of DNA methyltransferase genes (notably Dnmt3) are associated with thermotolerance differences among lineages and sexes, and experimental manipulation of methyltransferase expression has been used to probe causal relationships with temperature tolerance (Dai et al., 2018; Dai et al., 2025). Importantly, methylation variation correlates with invasive-lineage thermal performance, suggesting that DNA methylation is a candidate mechanism for rapid adjustment to

changing thermal regimes (Dai et al., 2025). These data support the inclusion of DNA methylation as a plausible regulatory layer in thermal and water stress research, while also highlighting that its relevance must be evaluated species-by-species rather than assumed to be universal across Insecta (Dai et al., 2025). Given the breadth of epigenetic layers implicated in stress-responsive transcription, Table 2 summarizes the principal epigenetic mechanisms, their expected regulatory consequences, and representative insect evidence relevant to the biology of combined heat and desiccation exposure.

Chromatin remodeling provides an additional axis of regulation because stress-inducible transcription (including HSP loci) depends on rapid changes in accessibility and polymerase dynamics. In Drosophila, chromatin remodelers can influence heat-shock gene expression; for example, the nucleosome remodeler dMi-2 has been implicated in regulating the heat-shock response and recovery, underscoring that nucleosome architecture contributes to both activation and post-stress reset of transcriptional programs (Mathieu et al., 2012). Small non-coding RNAs further add post-transcriptional and chromatin-linked control. In Drosophila embryos, an early heat shock alters microRNA dynamics and downstream gene expression, demonstrating that small-RNA layers can be stress-sensitive and temporally specific, with potential implications for persistent regulatory effects (Örkenby et al., 2023). In parallel, heat stress can interact with transposable element (TE) expression and TE-derived small RNAs in insects, linking environmental stress to genome regulation and small-RNA pathways, although the magnitude and direction of effects can be population- and sex-specific (Bodelón et al., 2023). Collectively, these studies justify treating epigenetic regulation not as an "optional add-on," but as a mechanistically grounded contributor to variability in stress transcriptomes and survival outcomes—particularly when the goal is to explain why similar stress exposure can yield different transcriptional trajectories or resilience phenotypes across genotypes, populations, or invasive lineages (Bodelón et al., 2023; Örkenby et al., 2023; Dai et al., 2025).

Comparative Gene Expression Across Insect Species

Heat and Desiccation Tolerance in Different Insect Orders

Insect diversity is mirrored by substantial diversity in transcriptional solutions to heat and water limitation. Comparative transcriptomics reveals a recurring dual architecture: a conserved core stress program—encompassing chaperones, antioxidant defenses, and proteostasis—underlies all responses. This core is supplemented by lineage- and ecology-specific modules, including cuticular barrier remodeling, osmolyte metabolism, and water-transport regulation (Wang et al., 2021). Dipterans, especially Drosophila, provide a rich basis for cross-species inference because both natural populations and experimentally evolved lines have been interrogated at genome-wide resolution. In the desert-adapted cactophilic fly Drosophila mojavensis, enhanced desiccation resistance is associated with coordinated transcriptional regulation of metabolic pathways, consistent with physiological reallocation in response to water limitation (Matzkin & Markow, 2009). Moreover, ecologically differentiated populations of D. mojavensis show strong temperature-dependent shifts in transcriptomes alongside changes in cuticular hydrocarbon (CHC) profiles, highlighting how thermal environment can restructure both gene expression and barrier traits that affect water loss (Etges et al., 2017). In D. melanogaster, selection experiments and cross-study syntheses show that desiccation resistance can be accompanied by broad, repeatable changes in basal expression across hundreds of genes, indicating that adaptation often involves both constitutive "readiness" and plastic induction (Telonis-Scott et al., 2016).

Coleopterans include many arid-zone specialists, in which transcriptional investment frequently emphasizes cuticular and metabolic features relevant to water conservation. For example, RNA-seq in the desert tenebrionid beetle Microdera punctipennis revealed extensive transcriptional representation of pathways commonly implicated in stress resilience (including detoxification and protective metabolism), providing molecular entry points into how beetles persist in xeric habitats (Lu et al., 2014). Hymenopterans—especially desert ants—offer an informative contrast in which extreme thermophily is often paired with distinct transcriptomic strategies. Cataglyphis ants exhibit heat-shock protein synthesis linked to high-

temperature performance, and comparative transcriptomics across desert vs temperate ants indicates that some desert taxa display relatively constrained differential expression under heat stress, consistent with either constitutive protection or alternative mechanisms that reduce the need for large inducible responses (Araujo et al., 2023). At a finer phylogenetic scale, Cataglyphis shows molecular signatures consistent with tuning of canonical heat-response pathways (Perez et al., 2021). Importantly, comparative physiology across bee taxa also suggests that ecological niche can reshape the joint distribution of thermal and desiccation traits: nocturnal bees (Megalopta) exhibit low upper thermal limits yet comparatively high desiccation resistance, underscoring that trait combinations do not always align with simple expectations and may reflect niche-specific constraints and selective histories (Gonzalez et al., 2023).

Evolutionary and Genomic Perspectives on Heat–Desiccation Tolerance

This section integrates (i) mechanisms of adaptation that shift stress tolerance phenotypes and reaction norms, and (ii) genomic/phylogenetic divergence patterns (e.g., copy-number change, regulatory architecture, genome organization) that shape how stress modules are encoded and deployed across lineages.

Mechanisms of Adaptation: Polygenicity, Reaction Norms, and Regulatory Rewiring

Evolutionary changes in tolerance to combined heat and desiccation exposure often involve both regulatory rewiring and polygenic shifts in standing variation. In Drosophila, artificial selection and genome-wide association approaches converge on the view that desiccation resistance is a highly polygenic trait, with repeatable genomic and transcriptional signatures across independent datasets (Telonis-Scott et al., 2016). Crucially, adaptive routes can involve changes in baseline expression (constitutive preparedness) as well as plastic response profiles, suggesting that selection may favor altered reaction norms rather than single-gene on/off switches (Wang et al., 2021). Recent work demonstrates that integrating chemical phenotypes with computational methods enhances evolutionary inference.

In diverse Drosophila species, variation in desiccation resistance is largely explained by cuticular hydrocarbon (CHC) composition. Machine learning identified key CHC predictors, and experimental manipulation confirmed causal roles for specific CHC components (Wang et al., 2022). This highlights a practical path for connecting high-dimensional phenotypes to evolutionary mechanisms and prioritizing experimentally testable targets.

Across Hymenoptera, selection in extreme deserts appears to have repeatedly shaped heat tolerance via modifications to heat-response deployment and stress-protective metabolism, yet comparative transcriptomics suggests multiple evolutionary "solutions," including either stronger inducible responses or more constitutive states that reduce the magnitude of differential expression during heat exposure (Araujo et al., 2023; Perez et al., 2021). Thus, similar phenotypic endpoints (high thermotolerance) can emerge from distinct regulatory architectures, cautioning against overgeneralization from single model systems. Meanwhile, ecological niche can impose constraints that favor particular trait combinations; for nocturnal bees, relatively low heat tolerance paired with high desiccation resistance suggests that selection on water balance and selection on thermal maxima can be partially decoupled, producing non-intuitive combinations relevant to climate change risk (Gonzalez et al., 2023).

Finally, extreme dehydration survival in Polypedilum vanderplanki illustrates that the evolution of "super-tolerance" may require both quantitative amplification of conserved pathways and qualitative innovation through gene-family expansions and genome organization that enable coordinated induction (Yoshida et al., 2022). Thus, evolutionary trajectories in insects appear to range from incremental tuning of standing stress networks (common in Drosophila lineages) to major genomic reconfiguration in rare anhydrobiotic specialists.

Genomic and Phylogenetic Divergence: Copy Number, Regulatory Architecture, and Genome Organization

Comparative genomics indicates that divergence in stress tolerance frequently reflects changes in gene copy number, regulatory architecture, and network connectivity, rather than wholesale replacement of core stress genes. In ants, transcriptomic comparisons show that closely related desert and temperate taxa can differ markedly in the scale and composition of heat-induced differential

expression, supporting the view that evolutionary divergence can target regulatory responsiveness (how much the transcriptome "moves") as much as it targets gene identity (Araujo et al., 2023). Focused analyses in Cataglyphis likewise suggest molecular adaptation to heat stress within a canonical framework of cellular protection and proteostasis, consistent with the broader idea that stress networks are conserved while their control parameters—thresholds, timing, and baseline activity—are evolvable (Perez et al., 2021).

The most striking example of lineage-specific augmentation remains P. vanderplanki, where genome-scale analyses reveal anhydrobiosis-related gene "islands" enriched for protective functions and tightly linked to dehydration-inducible expression, implying that genome organization itself can evolve to facilitate coordinated stress-module activation (Yoshida et al., 2022). Collectively, these findings support a modular evolutionary model: core stress genes are broadly conserved, whereas extreme phenotypes arise through expansion, rewiring, and coordinated regulation of accessory modules aligned with the dominant ecological challenge (heat spikes, chronic aridity, or both) (Wang et al., 2021).

Collectively, these findings support a modular evolutionary model: core stress genes are broadly conserved, whereas extreme phenotypes arise through expansion, rewiring, and coordinated regulation of accessory modules aligned with the dominant ecological challenge (heat spikes, chronic aridity, or both) (Wang et al., 2021). From a translational perspective, identifying which regulatory rewiring events are unique to highly specialized taxa (such as P. vanderplanki) versus those conserved across diverse, ecologically relevant groups (such as Drosophila or crop pests) is critical. This comparative approach dictates whether molecular tools (e.g., biomarkers or targeted interventions) developed for agricultural pest control can be readily generalized to non-model systems, or whether sector-specific development is necessary due to regulatory architecture divergence.

Mechanisms of Stress Memory and Long-Term Effects

Heat and Desiccation Stress Memory in Insects

Insects can exhibit short-term "stress memory" (often termed hardening or rapid acclimation), in which a sublethal exposure to heat or dehydration increases tolerance to a subsequent, more severe challenge. Classic work in *Drosophila* demonstrates that brief thermal pretreatments can elevate later survival under ecologically relevant heat stress, and that this benefit covaries with inducible Hsp70 expression among genotypes. More recent comparative work across *Drosophila* species further emphasizes that the magnitude of hardening responses depends on baseline tolerance and the duration/intensity required to induce maximal plasticity (i.e., "threshold shifts"), which is critical for correctly interpreting acclimation capacity under warming and drying climates (Van Heerwaarden et al., 2024).

For desiccation hardening, mechanistic studies in adult *D. melanogaster* show that mild dehydration pretreatments can rapidly increase subsequent desiccation survival primarily by reducing water-loss rate via changes in cuticular permeability, rather than by increasing body water stores (Bazinet et al., 2010). Follow-up work links this rapid desiccation hardening phenotype to acute shifts in cuticular hydrocarbon profiles, consistent with a fast-acting modulation of the transpiration barrier that can be sex-specific in its effectiveness (Stinziano et al., 2015). Together, these findings support a general model in which hardening benefits arise from (i) quick induction of protective proteins (notably HSPs) under heat, and (ii) rapid, reversible changes in water-balance traits (notably cuticular properties) under dehydration—both of which can directly alter survival in acute stress episodes (Sinclair et al., 2022).

At the gene-regulatory level, it is reasonable to expect that hardening involves transient persistence of stress-induced transcripts/proteins and rapid re-engagement of the transcriptional machinery. However, strong claims about long-lived "epigenetic bookmarking" of stress loci in insects should be made cautiously: while heat shock clearly triggers large-scale, rapid transcriptional reprogramming and chromatin-associated changes (e.g., transcriptional repression and altered termination during acute heat shock), direct evidence that specific histone marks persist long enough to function as a durable memory mechanism in insects remains limited and likely context-dependent (Cugusi et al., 2022). A conservative, evidence-aligned interpretation is that hardening primarily reflects short-term physiological carryover (elevated chaperone capacity; altered cuticular/respiratory water loss)

rather than a well-demonstrated, stable chromatin memory system across tissues and timescales in most insects (Sinclair et al., 2022; Van Heerwaarden et al., 2024).

Adaptive Evolution and Cross-Generational Effects

Beyond within-generation plasticity, insect populations can also adjust to recurrent thermal environments through evolutionary change and through cross-generational (parental) effects that do not require immediate DNA-sequence evolution. Experimental evolution provides direct evidence that thermal regimes can drive rapid genomic change: in *Drosophila simulans*, replicated populations exposed to fluctuating temperatures for ~20 generations showed clear evolutionary responses detectable by whole-genome sequencing, while inducible proteomic responses within a generation were largely distinct from the loci showing evolutionary shifts—highlighting that plasticity and adaptation can rely on partly non-overlapping genetic architectures (Sørensen et al., 2020). Moreover, selection on heat tolerance can generate correlated responses in other resistance traits, including cross-tolerance patterns, emphasizing that multi-stressor evolution is not simply additive (Castañeda et al., 2025).

Cross-generational effects can be strongly shaped by the thermal history of parents and even of stored gametes, with major implications for population persistence under heatwaves. In the flour beetle *Tribolium castaneum*, experimentally imposed heatwave conditions reduced male reproductive performance and produced measurable transgenerational damage, including reduced offspring reproductive potential and lifespan when fathers (or their stored sperm) experienced heatwave stress (Sales et al., 2018). Complementary work further supports that the thermal environment experienced by male gametes can influence downstream offspring phenotypes and reproductive outcomes, consistent with a pathway for cross-generational vulnerability or compensation depending on exposure timing and severity (Graziano et al., 2023). Importantly, transgenerational outcomes need not be uniformly beneficial: recent evidence in a moth system demonstrates transgenerational cross-susceptibility, where parental cold hardening increased offspring vulnerability to heat stress, underscoring that "stress memory" across generations can involve trade-offs and antagonistic cross-stressor effects (Mpofu et al., 2024).

Mechanistically, the causal pathways behind cross-generational effects likely include parental provisioning, altered endocrine state, and germline-associated regulatory changes (including small RNAs and chromatin-associated processes), but the strength and persistence of these mechanisms vary widely among insects, and robust causal demonstrations (especially for stable, inherited epigenetic marks) remain uneven across taxa. The most defensible framing is therefore: (i) within-generation hardening is well supported for heat and desiccation, (ii) cross-generational effects are real and can be large, especially for fertility-related endpoints under heatwaves, but (iii) their mechanistic bases and predictability across environmental contexts remain active research frontiers (Sales et al., 2018; Sørensen et al., 2020; Sinclair et al., 2022).

Environmental and Ecological Contexts of Heat–Desiccation Stress

Natural Habitat and Climate Change Impact

An insect's realized exposure to heat and desiccation is set by habitat structure and daily activity patterns, not just regional climate means. In deserts, insects face high operating temperatures and low humidity, which accelerate water loss. Selection favors integrated behavioral avoidance (e.g., activity timing, refuge use) and physiological adaptations enhancing water balance and proteostasis (e.g., cuticular barriers, chaperone capacity). In contrast, humid forests can buffer against dehydration risk but still cause acute thermal stress in sun-exposed strata, meaning that "heat stress" often manifests as short-term peaks in body temperature rather than chronic dehydration. Critically, insect body temperatures are frequently decoupled from macroclimatic air temperatures; fine-scale microclimates can buffer or magnify thermal extremes, and this mismatch can strongly alter predictions of stress exposure and performance (Pincebourde & Woods, 2020; De Frenne et al., 2021). Empirical microclimate work further shows that thermal heterogeneity within habitats (e.g., edges vs. interiors) can intensify during heatwaves, reshaping the magnitude and duration of extreme exposure for small ectotherms (Gols et al., 2021).

These habitat-contingent exposure patterns matter because climate change is increasing the frequency

and intensity of compound extremes (heatwaves and droughts), which can push insects beyond physiological tolerance limits even when seasonal means appear modest. Large-scale evidence indicates that extreme heat events are already linked to population declines in climate-sensitive taxa; for instance, bumble bee occupancy dynamics across continents are consistent with increasing frequencies of temperatures exceeding historically experienced tolerances (Soroye et al., 2020). At broader biodiversity scales, recent syntheses emphasize that climate change is now a major driver of insect risk globally, interacting with habitat alteration and other stressors to elevate extinction probability (Harvey et al., 2023). Mechanistically, these outcomes are expected when heat and low humidity co-occur, because dehydration can become a proximate failure mode that reduces effective thermal limits and compromises post-stress recovery—an interaction that macroclimate-only assessments can miss if they ignore humidity and microrefugia (Pincebourde & Woods, 2020; De Frenne et al., 2021).

From an applied ecological perspective, differential challenge tolerance can reorder communities and weaken ecosystem services. Temperature extremes can produce both "outbreak" and "breakdown" scenarios—amplifying some herbivores while destabilizing trophic interactions (e.g., if predators/parasitoids experience greater mortality or phenological mismatch) (Harvey et al., 2020). In pollinators, colony-level heat exposure has measurable impacts: field data show brood-nest temperatures often exceed optimal ranges during heatwaves, leading to adverse developmental outcomes (Poot-Báez et al., 2020). At the molecular level, heat and humidity stressors elicit coordinated changes in stress- and immune-related gene expression in honey bees (Apis mellifera), supporting the use of transcriptomic markers to diagnose climate-linked physiological strain (Maigoro et al., 2025; Sagastume et al., 2025). Together, these findings argue that predicting insect persistence under climate change requires linking *experienced* microclimates (temperature × humidity) to the gene-regulatory and physiological capacity for short-term survival and long-term demographic resilience.

Role of Microclimates and Habitat-Specific Adaptations

Microclimates—conditions under leaves, within soil/litter, in canopy surfaces, or inside nests—often determine whether insects face lethal combined heat and desiccation exposure stress or remain within a survivable "refuge envelope." Forest understories, for example, can buffer thermal extremes relative to above-canopy air temperatures, but this buffering is spatially patchy and sensitive to vegetation structure and disturbance, making microrefugia a key determinant of persistence as climates warm (De Frenne et al., 2021). Insects exploit these gradients behaviorally (seeking shade, burrowing, timing activity), yet behavior is only part of the story: repeated exposure to predictable microclimatic regimes can select for habitat-specific tuning of stress responses (e.g., altered inducibility thresholds of protective pathways, or higher baseline readiness in consistently hot microhabitats) (Pincebourde & Woods, 2020).

Field and experimental evidence also show that microclimates can differ dramatically across vertical strata. Tropical forest canopies experience high solar loading and rapid heating, selecting for strategies that combine behavioral thermoregulation with physiological tolerance. For instance, twig-nesting canopy ants show distinct behavioral responses to acute heat stress, illustrating how canopy life can impose strong selection on coping strategies even before considering molecular differences (Bujan & Yanoviak, 2022). Likewise, edge habitats can impose higher and more variable temperatures than interiors; microclimatic amplification during heatwaves can therefore produce "thermal traps" where insects are forced into costly trade-offs between dehydration risk and predator avoidance or foraging (Gols et al., 2021).

Habitat-specific adaptation is increasingly visible in human-modified microclimates. Urban heat islands create hotter (and often drier) conditions that can drive rapid evolutionary shifts in thermal tolerance. Common-garden evidence in an acorn-dwelling ant indicates evolved changes in heat (and sometimes cold) tolerance associated with urbanization across multiple cities (Diamond et al., 2018). Similarly, continent-scale sampling in a widespread moth provides evidence consistent with repeated urban evolution of increased heat tolerance (Merckx et al., 2024). These studies underscore an important point for heat–desiccation biology: adaptation may be life-stage specific,

context dependent, and shaped by the joint distribution of microclimate temperature and moisture.

Finally, microclimate integration can improve forecasting and management. Models that incorporate fine-scale temperature data can outperform coarse-scale datasets when predicting insect abundance and phenology in complex landscapes, highlighting the value of mechanistic microclimate mapping for climate-risk assessment (Rebaudo et al., 2016). Practically, this implies that conserving or restoring structural features that generate microrefugia (shaded understories, heterogeneous ground cover, nest-site availability) may reduce lethal exposure, buying time for plastic and evolutionary responses—especially for species with limited dispersal or narrow tolerance margins (De Frenne et al., 2021; Harvey et al., 2023).

Practical Implications and Future Directions

Implications for Insect Conservation and Pest Control

Mechanistic insights into combined heat and desiccation exposure gene-expression modules are increasingly actionable: molecular markers—such as inducible *hsp* transcripts, antioxidant pathways, and immune–stress crosstalk—enable early detection of physiological stress in beneficial insects, supporting proactive conservation. For pest management, these same markers improve resilience forecasting under escalating heat waves and drought. In honey bees, heat stress can reshape transcriptional programs linked to proteostasis, immunity, and pathogen interactions, with measurable colony-level consequences; such evidence supports integrating stress biomarkers into health surveillance and management decisions (McMenamin et al., 2016; McKinstry et al., 2017; González-Tokman et al., 2020). At the practical level, this strengthens the rationale for (i) conserving microclimatic refugia (shade, moisture-retaining vegetation, reduced ground exposure) and (ii) prioritizing locally adapted stocks in breeding and selection programs, while recognizing that "heat tolerance" is not a single trait but an emergent property of coordinated modules (e.g., chaperones + redox control + metabolic stabilization) (González-Tokman et al., 2020; Zhao et al., 2021). Furthermore, the identified molecular pathways—especially those governing proteostasis and desiccation tolerance—offer transferable blueprints for developing stress-resilient materials and bioprocesses in industrial biotechnology sectors.

For pest control, stress-response modules can be exploited as liabilities rather than strengths. A robust body of literature shows that perturbing core proteostasis genes reduces insects' capacity to withstand thermal extremes, suggesting that environmental stress can be leveraged as a synergist when stress defenses are compromised. For example, RNAi targeting *hsp70/hsp90* in an agricultural moth reduced acquired thermotolerance, demonstrating that disrupting chaperone capacity can convert otherwise sublethal heat exposure into a meaningful fitness cost (Bandani & Farahani, 2024). Similarly, functional work in other insects underscores that HSP family members are deeply embedded in survival physiology and can influence systemic performance under resource stress and temperature challenge (Paim et al., 2016). While translation to field-ready interventions requires substantial advances in safety and specificity, these results support a "stress-amplification" concept in IPM: interventions that impair chaperoning, cuticular waterproofing, or antioxidant buffering may increase mortality during naturally occurring hot/dry periods, rather than relying solely on direct lethality.

Genetic control strategies are advancing rapidly, and CRISPR-based approaches (including precision-guided sterile insect technique and gene-drive-related concepts) are being actively reviewed and tested across pest taxa, raising the prospect of targeting stress-related loci or their regulators in tightly controlled programs where ecological and regulatory constraints are met (Komal et al., 2023; Ying et al., 2023).

Gaps in Current Knowledge and Future Research Directions

A central limitation in dual abiotic stress research is the mismatch between laboratory assays and the fluctuating, co-occurring stress regimes insects experience in nature. Many transcriptomic studies still rely on single-factor, acute exposures under constant conditions, whereas real heat waves often couple high temperature with low humidity and altered resource availability. Bridging this gap will require (i) field-realistic thermal–humidity trajectories, (ii) life-stage-aware designs (eggs, diapause, pupae), and (iii) explicit linkage between transcriptional modules and performance outcomes (survival, reproduction, dispersal).

A second gap is the incomplete regulatory mapping of combined-stressor responses. While canonical hubs (HSF–HSP, oxidative stress pathways, metabolic reprogramming) are well recognized, stress-specific enhancers, chromatin accessibility shifts, and combinatorial transcription-factor logic remain under-characterized in most insects. Here, chromatin-level assays can be transformative. For instance, integrative ATAC-seq and RNA-seq in *Bemisia tabaci* under temperature stress has begun to connect chromatin accessibility dynamics to transcriptional remodeling—an approach directly relevant to understanding how "combined stress" can produce expression states not predictable from single stressors alone (Shen et al., 2023). Broader methodological syntheses also highlight both the opportunities and pitfalls (cell heterogeneity, limited tissue amounts, annotation gaps) of bringing ATAC-seq into non-model systems, which is especially pertinent for ecologically important insects (Erdogan et al., 2025).

Third, multi-omics integration remains underused in the biology of insect exposure to combined heat and desiccation. Transcriptomes do not always predict protein abundance, enzyme activity, or metabolite availability, and stress phenotypes often hinge on post-transcriptional control and metabolite-mediated stabilization. Encouragingly, multi-omics studies in major pests are now identifying candidate modules (e.g., chitin/cuticle-associated pathways) that appear to contribute to high-temperature tolerance, providing more mechanistic traction than transcriptomics alone (Yan et al., 2024).

Finally, translation to practice needs stronger causal evidence. Many studies identify differentially expressed genes, yet relatively few confirm necessity/sufficiency via perturbation (RNAi, CRISPR, pharmacology) across ecologically relevant contexts. The field's highest-value next step is a tiered pipeline: field-realistic exposures → multi-omics module discovery → causal tests of a small, well-justified set of hubs → scalable biomarkers for monitoring and prediction. This is the most reliable route to move from "stress signatures" to interventions that work outside the lab (Shen et al., 2023; Yan et al., 2024).

Prospects for Translating Findings to Other Organisms

Many molecular elements emphasized in insects—proteostasis networks, redox buffering, osmolyte-mediated stabilization, and intrinsically disordered protective proteins—are evolutionarily widespread, making insect discoveries valuable beyond entomology. A particularly active translational frontier is biostabilization and induced anhydrobiosis. Work on anhydrobiotic organisms has already shaped strategies for preserving cells and biomolecules in low-water states, with trehalose and protective proteins serving as recurrent design principles (Weng et al., 2021; Olgenblum et al., 2024). Notably, trehalose and tardigrade CAHS proteins can synergistically promote desiccation tolerance in engineered systems, strengthening the mechanistic basis for biomimetic preservation approaches applicable in areas such as pharmaceutical and cell storage (biotech sector) (Nguyen et al., 2022). Recent perspectives further argue that "induced anhydrobiosis" concepts may become increasingly practical for cell and gamete storage, explicitly drawing from stress-tolerant taxa, including insects (Loi et al., 2025).

In agriculture and ecology, insect stress biology improves predictions of pest pressure under climate change: physiological hardening during repeated heat–drought events can increase herbivory risk despite reduced plant nutritional quality. Insects offer tractable models for studying proteostasis and osmotic stress resilience—key challenges in biomedicine and material science. Their modular stress-response strategies—buffering, redundancy, inducible protection, and extreme desiccation tolerance—provide design principles for biotechnology and conservation physiology, not direct solutions, but generalizable blueprints for enhancing resilience across diverse biological and industrial systems (Weng et al., 2021; Olgenblum et al., 2024).

Conclusion

Survival under combined heat and desiccation stress is fundamentally dependent on the effective coordination between the conserved core stress response (focused on proteostasis) and the specialized, dehydration-facing modules (governing water retention and osmotic defense). The modular framework presented here shifts the research focus from identifying which individual genes change to understanding how these modules

are functionally coordinated to preserve essential processes across different timescales of stress exposure and recovery. Key to advancing the field is the adoption of pipelines that match field-realistic temperature × humidity trajectories with causal testing via integrated multi-omics and targeted perturbation studies. Ultimately, understanding this modular coordination provides a robust framework for engineering resilience in diverse systems, bridging fundamental entomological research with applied needs in conservation, biomedicine, and ecological management.

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Table 1- Key stress-responsive genes/gene families involved in heat and desiccation responses in insects.

Gene/protein family	Role in heat stress	Role in desiccation stress	Notable insect examples	References
HSP70 / HSP90 / sHSPs	Proteostasis buffering: prevents aggregation, assists refolding; supports recovery after acute heat shock	Often induced during severe dehydration/anhydrobiosis; supports macromolecule protection when water loss increases proteotoxic risk	*Drosophila* (heat hardening), Pv11 / *Polypedilum vanderplanki* (anhydrobiosis program co-opts heat-shock regulation)	Sørensen et al., 2003; Mazin et al., 2018
Antioxidant enzymes (SOD, catalase, peroxiredoxins, glutathione/thioredoxin systems)	Mitigate ROS generated by heat-accelerated metabolism/mitochondrial perturbation; reduce oxidative damage to proteins/lipids/DNA	ROS management is also critical during dehydration and especially during recovery/rehydration; antioxidant modules commonly co-enriched with proteostasis pathways	Broad across insects; highlighted in dehydration biology syntheses	Benoit et al., 2023; González-Tokman et al., 2025
Aquaporins (AQPs)	Indirect roles via water movement/osmoregulatory physiology (context-dependent; less "heat-specific" than HSPs)	Central for controlled water flux and tissue water management under dehydration; AQP regulation is frequently discussed as a dehydration-relevant module	Antarctic midge (*Belgica antarctica*) and other insects (reviewed comparatively)	Yoshida et al., 2021
Trehalose synthesis pathway (e.g., TPS/TPP; trehalose metabolism genes)	Can support heat stress indirectly via protein/membrane stabilization and energy buffering (species- and context-dependent)	Major compatible solute in extreme desiccation tolerance/anhydrobiosis; trehalose-driven stabilization is a hallmark in anhydrobiotic systems	Pv11 / *P. vanderplanki* (trehalose-dependent entry into anhydrobiosis)	Mazin et al., 2018
Cuticular hydrocarbon (CHC) biosynthesis/cuticle barrier genes	Mainly indirect (heat may alter barrier chemistry and thereby water loss; ecology-dependent)	Core mechanism for reducing cuticular water loss; CHC composition is strongly linked to desiccation resistance and divergence across taxa	*Drosophila* (comparative and evolutionary patterns); broad insect comparative framing	Wang et al., 2021
LEA-like protective proteins / intrinsically disordered protectants	Not typically central to "classic" heat response; relevance increases in extremophile contexts	Key macromolecule protectants in extreme dehydration/anhydrobiosis; gene family expansions and "gene islands" are linked to dehydration-inducible expression	*P. vanderplanki* (anhydrobiosis-related gene islands)	Gusev et al., 2011; Mazin et al., 2018
Master regulators (TFs / signaling hubs): HSF (heat-shock regulator), FOXO (stress–metabolic reallocation), stress kinases (e.g., JNK)	HSF drives rapid HSP transcription; FOXO integrates stress with metabolic downshift and defense programs; JNK can promote FOXO activity in stress contexts	FOXO/stress-kinase logic can support survival under resource limitation and dehydration-associated tradeoffs (often via metabolic reprogramming)	*Drosophila* (mechanistic foundation for JNK→FOXO; broad relevance)	Wang et al., 2003; Akerfelt et al., 2010

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Table 2- Epigenetic mechanisms implicated in insect stress responses (heat and/or desiccation).

Epigenetic mechanism	What changes (examples)	Relevance to heat/desiccation stress	Notable insect examples	References
DNA methylation machinery	DNMT activity; gene-body methylation patterns (taxon-dependent in insects)	Can modulate transcriptional responsiveness and plasticity; evidence in some pests links DNMT function to temperature tolerance	*Bemisia tabaci* (DNMT1 functional importance for temperature tolerance)	Dai et al., 2018
Chromatin accessibility (ATAC-seq)	Stress-dependent opening/closing of regulatory regions; altered accessibility at stress-response loci	Provides a direct map of regulatory logic under temperature stress; helps link "regulation" to transcriptional modules (important for an epigenetics-framed journal)	*Bemisia tabaci* temperature stress (ATAC-seq + RNA-seq integration)	Shen et al., 2023
Chromatin remodeling complexes	Nucleosome positioning/remodeling; regulation of activation and recovery phases	Can affect induction kinetics and post-stress reset (turning stress genes off efficiently)	*Drosophila* (Mi-2 involvement in heat-shock gene regulation/recovery)	Mathieu et al., 2012
Small RNAs (miRNAs)	Stress-altered miRNA profiles: post-transcriptional control and pathway tuning	Can reprogram networks rapidly and fine-tune translation/decay of stress-related transcripts; can contribute to persistence of response signatures	*Drosophila* (heat shock reshapes miRNA dynamics; system-level modeling)	Örkenby et al., 2023
Transposable elements (TEs) & TE-linked regulation	TE expression changes under stress; interaction with genome regulation and small-RNA silencing pathways	Heat stress can engage TE activity and genome defense regulation; useful to frame as "genome stability under stress"	*Drosophila* populations under heat stress (sex- and population-dependent TE responses)	Bodelón et al., 2023

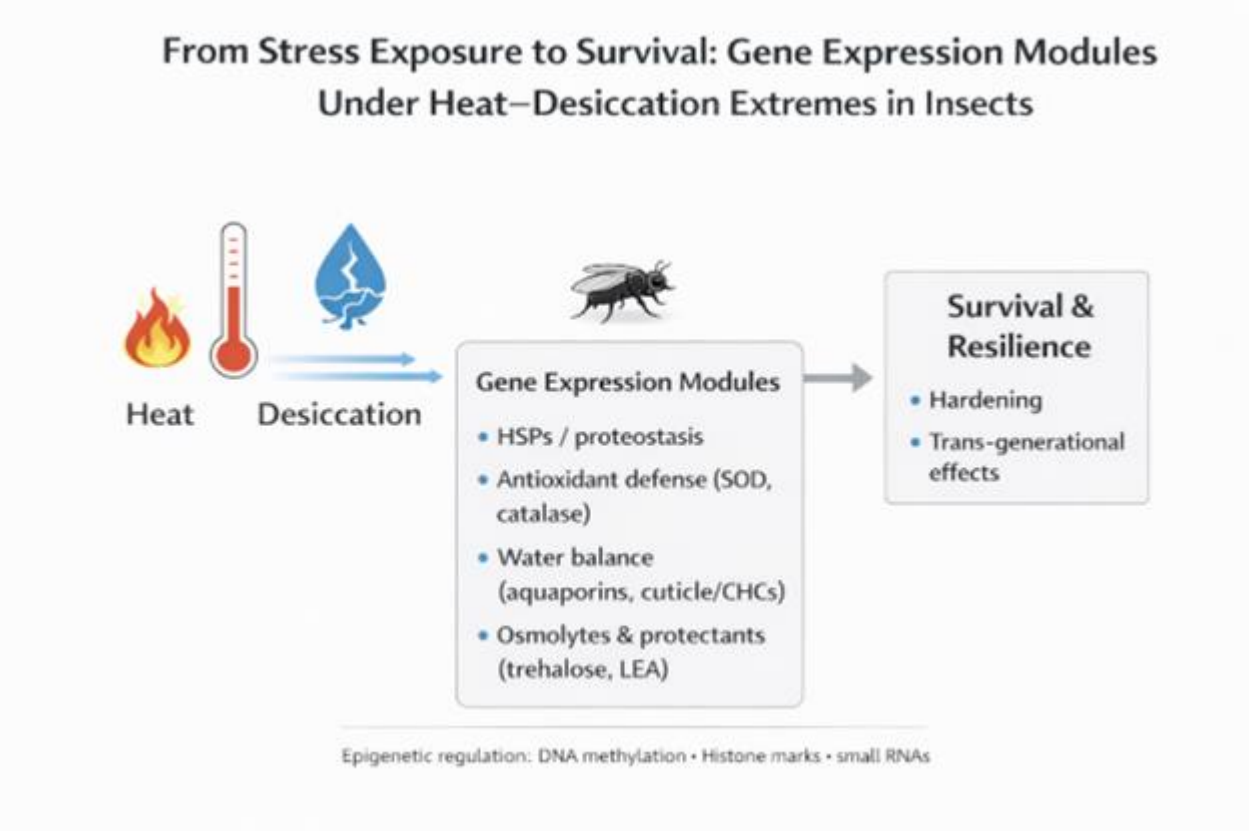

Figure. 1- Overview of gene-expression modules that mediate insect survival under combined heat–desiccation stress Heat stress and desiccation frequently co-occur in nature, particularly during hot–dry episodes, and jointly impose proteotoxic, oxidative, osmotic, and water-balance challenges. The diagram highlights core, partially shared gene-expression modules that are repeatedly implicated across insects: (i) proteostasis maintenance via heat shock proteins (HSPs), (ii) antioxidant defenses that buffer reactive oxygen species (ROS) during stress and recovery, (iii) water-balance regulation involving aquaporins and cuticular barrier traits (including cuticular hydrocarbons, CHCs), and (iv) osmolyte and protectant pathways such as trehalose metabolism and late embryogenesis abundant (LEA)-type proteins in highly desiccation-tolerant systems. These modules are tuned by regulatory layers including transcriptional and epigenetic mechanisms (DNA methylation, histone marks, and small RNAs), culminating in variation in survival and resilience phenotypes, including short-term hardening and cross-generational effects.

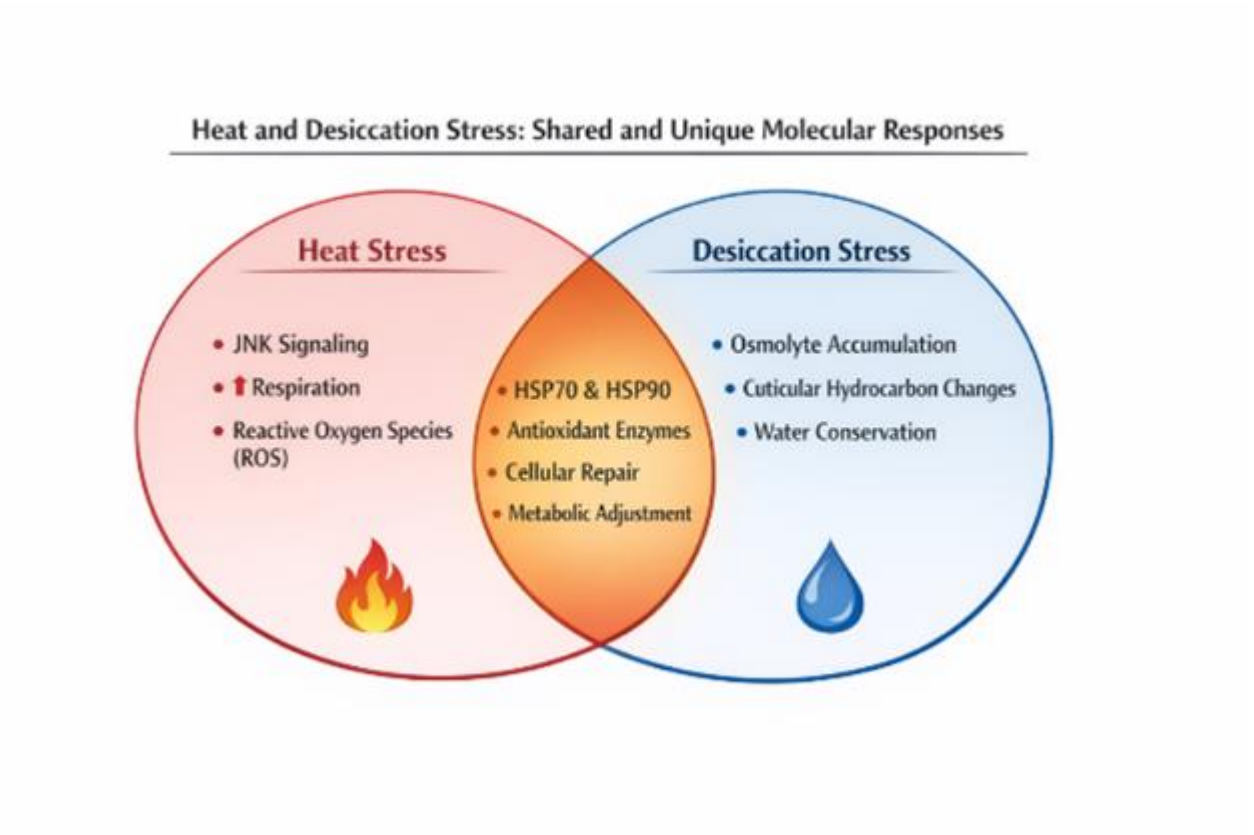

Figure 2. Conceptual framework of shared and stress-specific gene expression modules under heat and desiccation extremes in insects

The Figure illustrates a conceptual overview of the molecular pathways and gene expression modules activated in insects under heat stress, desiccation stress, and their combined occurrence. Heat stress predominantly induces proteotoxic and oxidative stress, activating signaling pathways such as JNK and increasing metabolic flux, thereby elevating reactive oxygen species (ROS) production and necessitating robust proteostasis responses. In contrast, desiccation stress primarily disrupts water balance and osmotic homeostasis, promoting the accumulation of compatible osmolytes (e.g., trehalose), remodeling cuticular hydrocarbons to reduce water loss, and regulating water transport and conservation mechanisms.

The overlapping region highlights shared protective modules induced by both stressors, including the upregulation of heat shock proteins (HSP70 and HSP90), antioxidant enzymes, cellular repair pathways, and metabolic adjustment processes. These shared modules represent a conserved core stress-response program that buffers cellular function under combined heat–desiccation extremes. The diagram emphasizes that insect survival under compound stress depends on the coordinated activation of both stress-specific and shared gene expression modules, rather than on a single tolerance pathway.