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Plasticity, Stress Signaling, Cellular Repair, and Physiological Limits Underlying Molecular Mechanisms of Insect Environmental Resilience

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ABSTRACT

Insects respond to environmental stress through molecular plasticity, stress signaling, proteostasis, cellular repair, metabolic reconfiguration, and recovery processes, yet activation of these systems is often interpreted too readily as evidence of resilience. This critical synthesis distinguishes molecular responsiveness from demonstrated maintenance or restoration of organismal function. Evidence from thermal, freezing, chilling, and desiccation studies shows that responses vary with stressor identity, exposure history, life stage, tissue, taxon, and measurement scale. Functional perturbation experiments identify genuine contributions of selected regulatory, proteostatic, repair, and metabolic pathways in focal systems, but the direction of benefit can reverse across stresses or endpoints. Molecular signatures may diverge between laboratory and field settings despite similar phenotypes, while compensatory responses can occur alongside declining performance or irreversible cellular injury. We therefore treat resilience as an evidence-bounded property of linked but separable processes: response activation, functional mediation, cellular maintenance and repair, metabolic reconfiguration, recovery, and persistence of organismal function within physiological limits. This framing does not assume that plasticity is adaptation, that molecular induction is beneficial, or that laboratory mechanisms transfer directly to field populations. The synthesis identifies where stronger inference requires perturbation, temporal resolution, cross-stage replication, orthogonal measurement, and direct linkage to recovery and performance.

Keywords: Thermal plasticity, Stress signaling, Proteostasis, Metabolic reconfiguration, Physiological recovery, Insect stress tolerance.

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Introduction

Environmental resilience in insects is frequently discussed through molecular signatures that rise after stress, particularly heat-shock proteins, oxidative-stress pathways, metabolites, and regulatory transcripts. Yet heat tolerance is increasingly understood as an integrated outcome of exposure, injury, protective processes, repair, and retained performance rather than the presence of one responsive marker [1]. This distinction matters because resilience is an outcome-oriented inference, whereas molecular induction is an observation at one biological level. Cold biology illustrates the same problem from another direction. Freeze-tolerant insects survive through coordinated processes operating before freezing, during extracellular ice formation, and through thawing and

recovery, so no single cryoprotective response is sufficient to represent the phenotype [2]. Chill tolerance likewise reflects interacting ion-homeostatic, membrane, and neuromuscular processes, with functional failure emerging from cross-level physiological disruption rather than a uniquely diagnostic molecular event [3].

Stress history further complicates interpretation. Models linking thermal damage, repair, and recovery show that equivalent thermal extremes can have different consequences when damage accumulates differently or recovery intervals differ [4]. Recent comparative metabolomics in aphids similarly indicates that more cold-tolerant species can show less metabolic disruption and different recovery trajectories, but such associations do not establish any single metabolite as the causal determinant of tolerance [5]. At broader scale, field-comparative evidence across tropical insects indicates that thermal adjustment can itself be bounded and taxonomically structured, cautioning against assumptions that inducible plasticity expands indefinitely with environmental demand [6].

This review therefore asks a narrower question than “which molecules respond to stress?” It examines when molecular plasticity and stress signaling contribute to cellular protection, when repair and metabolic reconfiguration restore function, and where these processes fail to prevent organismal impairment. The central argument is that molecular environmental resilience should be interpreted through explicit transitions between response, mediation, repair, recovery, and retained performance. The integrated architecture developed later is proposed as a synthesis of the selected evidence, not as a universally validated mechanism.

Molecular plasticity

Molecular plasticity is most informative when a regulatory change can be linked to a defined phenotype and when it remains distinguishable from evolutionary change. In *Bactrocera dorsalis*, population differences in thermal plasticity were associated with a miR-276b-centered regulatory network, and reporter and RNA-interference tests supported a functional contribution of this microRNA axis in the focal range-expansion context [7]. This is stronger evidence than transcript association alone, but it does not justify treating miR-276b as a general insect resilience regulator.

The distinction between plasticity and selection is equally important. Experimental evolution of *Drosophila melanogaster* under novel thermal regimes for many generations produced both shared and regime-specific transcriptomic and phenotypic changes [8]. These evolved responses are not interchangeable with short-term acclimation: the causal history, time scale, and heritability assumptions differ even when some downstream genes overlap.

Life stage can alter both the identity and functional importance of responsive genes. Stage-comparative transcriptomic and RNA-interference work in a drosophilid system found substantial differences in cold-responsive transcription and gene function across the life cycle, with some effects also contingent on sex [9]. A molecular mechanism established in one stage therefore cannot be promoted automatically to a species-level mechanism.

Environmental context creates a second transfer boundary. *Drosophila suzukii* exposed to field acclimatization and controlled laboratory acclimation achieved improved cold tolerance through metabolically divergent states, showing that phenotypic convergence does not require identical metabolomic signatures [10]. In the Arctic hemipteran *Nysius groenlandicus*, thermal tolerance and candidate-gene expression also changed rapidly through the season, while field expression did not always mirror expectations derived from laboratory stress assays [11]. Together, these observations support a restrictive interpretation of molecular plasticity: a response is most informative when its taxon, stage, environmental history, timing, and phenotype are specified.

Stress-signaling networks

Stress signaling converts environmental information into regulated cellular and organismal responses, but pathway activation is highly state dependent. In diapause larvae of *Monochamus alternatus*, a cold-activated chemical signal acting through the adipokinetic hormone receptor was functionally linked to mitochondrial remodeling and survival under the tested cold context [12]. The study demonstrates a mechanistic signaling route in a defined developmental state rather than a universal endocrine program for cold resilience.

Canonical stress transcription factors also participate in developmental regulation. In *Helicoverpa armigera*, upstream HIF-1 α and MNK signaling regulates an HSF-1–FoxO pathway involved in diapause induction [13]. This places familiar stress-responsive factors inside a developmentally constrained network and cautions against interpreting their activation only as acute cellular defense.

Even within a canonical heat-shock pathway, functional direction can reverse. Genomic deletion of hsp23 in *D. melanogaster* reduced performance under cold yet could increase fitness-related performance under the opposing thermal extreme, with additional stage dependence in hardening responses [14]. Thus, the presence or abundance of a stress protein cannot be assigned a uniformly positive meaning across environments.

Repeated stress can also recruit neuromodulatory control. Manipulation of dopaminergic signaling in *D. melanogaster* produced different consequences under repeated acute heat and cold exposure, affecting survival and life-history traits in a stress-specific manner [15]. The appropriate inference is that neuromodulation can participate in repeated-stress responses, not that it is inherently adaptive.

Temporal resolution is especially important when transcription is used as a proxy for metabolism. During chilling-induced diapause termination in *Bombyx mori* embryos, insulin-associated metabolic genes changed through time [16]. Because metabolic flux was not directly measured, the evidence supports a temporally organized candidate regulatory program rather than proof that transcript abundance itself represents metabolic activity.

Proteostasis and cellular repair

Protection from stress-related macromolecular damage extends beyond heat-shock induction. In an aphid symbiosis, complementary functions distributed across two endosymbionts assembled an effective DNA mismatch-repair system that supported bacteriocyte integrity and host heat tolerance [17]. The result is mechanistically important because it shows that repair can be biologically distributed rather than entirely host autonomous, but its specialized symbiotic context limits generalization.

Proteostasis also intersects organelle and lipid homeostasis. In *Bombyx mori*, lysosomal acid lipase-1 affected lysosomal acidification, autophagic flux, and lipid degradation through dependence on HSP83 [18]. These experiments establish a cellular maintenance mechanism, yet they do not by themselves demonstrate increased environmental resilience at the whole-organism level.

Thermal injury can push cellular responses across qualitatively different states. In *D. melanogaster* larvae, heat and cold generated distinct signatures of autophagy, apoptosis, and necrosis; matching acclimation improved outcomes, whereas severe injury was associated more strongly with apoptotic or necrotic processes and mortality [19]. Importantly, some autophagy-related signals did not guarantee completion of acidified autophagic flux, demonstrating why pathway markers and pathway function must remain distinct.

Functional manipulation provides an intermediate level of evidence. Rapamycin-mediated alteration of TOR/autophagy-associated processes in *D. melanogaster* increased local gut autophagy and was associated with improved heat tolerance and recovery, but pharmacological pleiotropy prevents assigning the entire phenotype uniquely to autophagy [20]. Conversely, HSP70 was broadly induced after heat but not after survivable freezing in *Gryllus veletis*, despite the cricket's freeze tolerance [21]. A marker can therefore be biologically important under one stress and uninformative under another.

A prediction-and-test ledger is useful here because proteostasis evidence differs not simply in strength but in what would falsify each interpretation. The distinctions below separate observed molecular states from the additional tests required before they can support stronger resilience claims.

Table 1. Proteostasis and repair evidence states that require different tests before resilience can be inferred

Repair state	Biological setting	Observed signal	Permitted meaning and status	Boundary	Decisive functional test
Proteostasis-response distinctions					
Stress-protein induction without functional perturbation	Tissue- and stressor-specific insect response	Increased HSP abundance or transcript	Interpretation permitted Stress-responsive proteostasis is engaged Status Evidence-supported distinction	Induction may be absent under another survivable stress	Test loss- or gain-of-function against a defined performance endpoint
Opposing function of the same stress protein	Developmental stage and thermal extreme	Gene deletion changes hot and cold outcomes in different directions	Interpretation permitted Functional sign is context dependent Status Evidence-supported distinction	Benefit cannot be inferred from expression alone	Cross stressors, stages, and fitness-relevant endpoints

Autophagy-associated signal without confirmed flux	Thermally stressed cells or tissues	Autophagy markers increase	Interpretation permitted Autophagy-related machinery is responsive Status Evidence-supported distinction	Marker accumulation can reflect incomplete flux	Pair markers with flux-sensitive and lysosomal measurements
Manipulated autophagy with improved tolerance	Whole-organism heat challenge with pharmacological intervention	Altered autophagy-associated activity plus improved recovery/tolerance	Interpretation permitted Autophagy-linked processes may contribute functionally Status Evidence-supported distinction	Drug pleiotropy limits pathway-specific causality	Use genetic and tissue-specific perturbation to test mediation
Distributed repair and cellular-homeostasis transfer					
Distributed DNA repair	Aphid–endosymbiont system under heat	Complementary repair functions plus bacteriocyte and host effects	Interpretation permitted Repair can emerge across symbiotic partners Status Evidence-supported distinction	Specialized host–symbiont architecture	Disrupt individual partners/functions and test host-level consequences
Cellular homeostasis without organismal stress phenotype	Insect lysosomal/lipid system	HSP83-dependent autophagic and lipid-homeostasis effects	Interpretation permitted A maintenance mechanism is established at cellular level Status Evidence-supported distinction	Resilience transfer remains untested	Link cellular perturbation to stress survival, recovery, or reproduction

Metabolic reconfiguration

Metabolic reconfiguration is often treated as a compensatory response, but the evidence supports several distinct possibilities: functional adjustment, metabolic flexibility, incomplete compensation, or transition toward failure. In *Spodoptera frugiperda* larvae, integrated transcriptomic and proteomic screening identified heat-responsive candidates, and RNA interference against selected heat-shock and detoxification genes increased mortality [22]. Functional perturbation therefore elevated a subset of omics signals from association toward focal mechanistic support, while leaving the larger response network unresolved.

A second *S. frugiperda* study illustrates why response and resilience must not be equated. High temperature altered chitin-related molecular pathways, and RNA-interference or CRISPR-based perturbation supported functional involvement of selected genes, yet organismal performance still deteriorated under heat [23]. Molecular remodeling can therefore be compensatory without being sufficient to preserve performance.

Metabolism can also cross from adjustment into failure. During honeybee metamorphosis, cold-induced repression of *Cpt1* uncoupled lipid mobilization from fatty-acid oxidation, increased lipid-associated oxidative damage, and contributed to developmental arrest; RNA interference reproduced key features of this state [24]. This provides a mechanistically supported example in which reconfiguration is not protective but part of a physiological limit.

Other systems show flexibility rather than collapse. Comparative tracer and respirometry measurements in *Ceratitis* flies associated greater desiccation resistance with more flexible oxidation of nutrient classes and altered protein use during recovery [25]. Because this is a comparative association, substrate flexibility should be interpreted as one viable strategy rather than a universal causal mechanism.

Taxonomic breadth further weakens the idea of one metabolic signature. Across 43 *Drosophila* species, cold-associated metabolomes contained both recurring patterns and lineage-specific responses; classical cryoprotectant changes were often idiosyncratic [26]. Within a single tropical cockroach species, post-cold metabolism was also strongly tissue- and time-dependent during recovery [27]. Mitochondrial synthesis across insect cold biology likewise identifies several plausible strategies—including maintenance of substrate oxidation, altered coupling, metabolic depression, membrane preservation, and mitophagy—but emphasizes that direct mechanistic evidence remains sparse for many taxa [28].

These observations support a proposed distinction among three metabolic inference states: reconfiguration that is functionally linked to protection, reconfiguration that is compensatory but insufficient to preserve performance, and reconfiguration that accompanies or drives physiological failure. The distinction is not a universal taxonomy of stress metabolism; it is a way to prevent metabolic change from being interpreted automatically as resilience. Because **Figure 1** must preserve both evidence status and the transition from molecular observation to organismal inference, it places metabolic states inside explicit validation boundaries rather than along a single beneficial-response pathway.

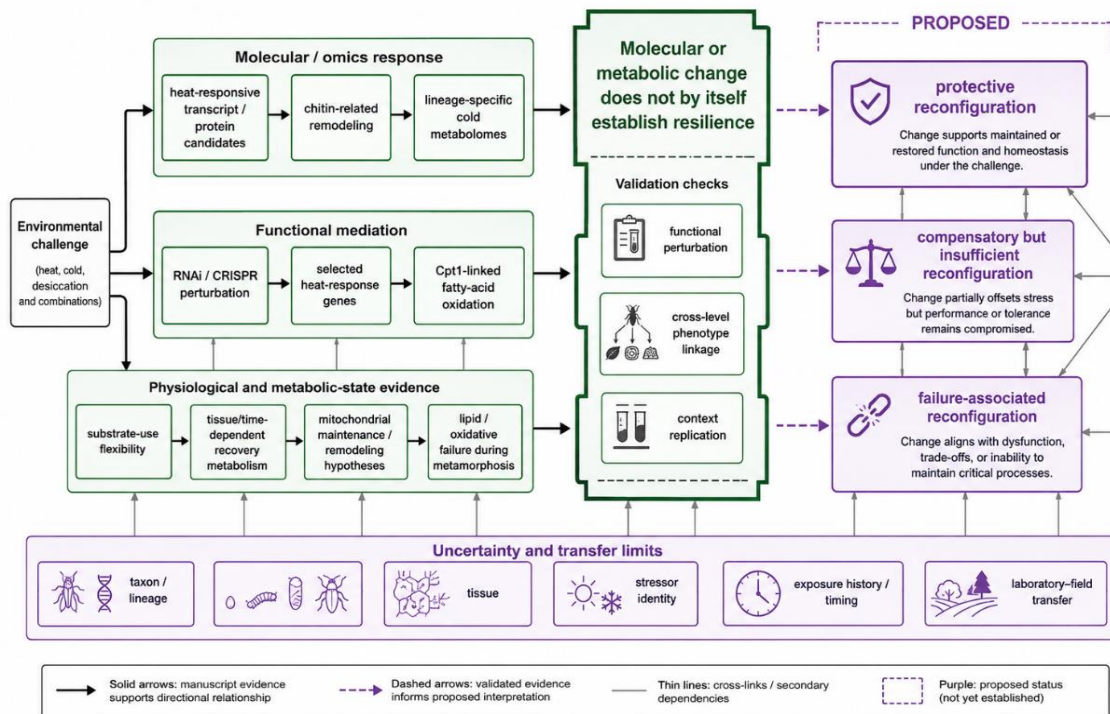


Figure 1. Metabolic reconfiguration can indicate protection, compensation, or physiological failure only after cross-level validation

Recovery and physiological memory

Resilience cannot be judged solely at the moment of stress exposure because recovery may follow a different physiological trajectory from immediate tolerance. In *Cnaphalocrocis medinalis*, prior heat exposure produced a transient transgenerational heat-memory phenotype associated with methylation and oxidoreductase-related changes, but the effect was not indefinitely persistent and could carry costs under particular thermal conditions [29]. Such evidence supports physiological or epigenetic memory without establishing stable evolutionary adaptation.

Developmental history can also separate tolerance from restoration. Developmental and adult acclimation in *Drosophila melanogaster* affected immediate heat tolerance and subsequent recovery differently, showing that these endpoints are partially separable rather than components of one scalar resilience phenotype [30]. At the cellular level, cold acclimation in *Gryllus veletis* increased F-actin in selected tissues while microtubules did not respond similarly; whether this reflected damage prevention or rapid repair remained unresolved [31].

Recovery becomes still more distinct as injury severity increases. In *Chymomyza costata*, supercooling, freezing, and cryopreservation produced different molecular, energetic, and survival trajectories, with the most extreme intervention generating persistent disruption and frequent recovery failure [32]. The ecological relevance of cryopreservation is limited, but the experiment demonstrates that recoverability depends on both injury type and severity.

These findings support a proposed distinction among immediate resistance, restoration after damage, history-dependent physiological memory, and failed recovery. **Table 2** separates these states because identical survival at the exposure endpoint can conceal very different post-stress biology.

Table 2. Recovery states that should remain separate when interpreting insect physiological memory and resilience

Recovery state and setting	Observable evidence	Interpretive consequence and status	Boundary and next test
Immediate resistance			
Recovery or memory state Immediate tolerance without measured recovery Biological context Acute environmental challenge	Survival or thermal-limit endpoint	Interpretation consequence Demonstrates short-term resistance only Status Evidence-supported distinction	Boundary condition Post-stress function unknown Decisive next test Measure restoration of locomotion, metabolism, development or reproduction
History-dependent restoration			
Recovery or memory state Recovery altered by acclimation history Biological context Developmental versus adult thermal experience	Different tolerance and recovery responses	Interpretation consequence Prior history changes restoration independently of immediate tolerance Status Evidence-supported distinction	Boundary condition Model-species and laboratory thermal history Decisive next test Replicate across stages and fluctuating environments
Recovery or memory state Transient physiological or epigenetic memory Biological context Repeated or ancestral heat exposure	Persistence of molecular/phenotypic effects across exposures or generations	Interpretation consequence Prior stress can modify later response Status Evidence-supported distinction	Boundary condition Persistence, cost and inheritance are context dependent Decisive next test Test duration, reversibility, fitness consequence and causal epigenetic mediation
Recovery or memory state Tissue-specific structural fortification Biological context Cold-acclimated freeze-tolerant cricket	Selective F-actin change with component-specific null results	Interpretation consequence Cellular preparation may contribute to later integrity Status Evidence-supported distinction	Boundary condition Prevention versus repair unresolved Decisive next test Resolve temporal sequence before and after injury
Recovery or memory state Recoverable versus persistent injury Biological context Increasingly severe cold treatments	Divergent metabolic, transcriptomic and survival recovery trajectories	Interpretation consequence Recovery capacity declines as injury becomes persistent Status Evidence-supported distinction	Boundary condition Extreme laboratory treatments may lack field analogues Decisive next test Link molecular recovery trajectories to ecologically relevant exposure histories
Cross-study separation rule			
Recovery or memory state Resistance–recovery–memory separation Biological context Cross-study synthesis	Different endpoints respond independently	Interpretation consequence Resilience should not be represented by one exposure-time measurement Status Proposed synthesis	Boundary condition Exact state boundaries require validation Decisive next test Measure exposure, recovery and repeated-challenge performance together

Hard limits to resilience

Plasticity is constrained when protective responses cannot prevent disruption of essential physiological function. Heat-stressed *Locusta migratoria* developed ion imbalance and hyperkalemia, yet ion concentrations recovered before complete functional recovery, indicating that ion disruption contributes to failure without explaining it

completely [33]. Meta-analysis across insect thermal-limit studies likewise found plasticity to be widespread but generally modest, with substantial taxonomic and life-stage heterogeneity [34].

Measurement method can itself define an apparent limit. Rapid cold hardening in locusts improved visual-circuit performance and supported altered in-situ Na⁺/K⁺-ATPase activity, whereas homogenate enzyme assays did not detect the same response [35]. A null bulk assay therefore cannot automatically falsify a mechanism operating at another biological scale. In *Thrips palmi*, heat and cold hardening or acclimation produced asymmetric, null, and sometimes adverse outcomes, demonstrating that prior exposure is not uniformly beneficial [36]. Across broad comparative, meta-analytic, and species-level evidence, thermal plasticity is measurable but bounded and strongly context dependent [6, 34, 36].

Table 3 distinguishes physiological limits from apparent limits created by endpoint choice or measurement scale.

Table 3. Context–mechanism–outcome register for distinguishing bounded plasticity from physiological failure

Ion-homeostatic disruption		
Heat-stressed locust	Hyperkalemia and altered ion balance	Loss of function followed by incomplete temporal correspondence during recovery
Contributing mechanism is not necessarily sufficient cause	Couple ion manipulation to independent functional endpoints	Evidence-supported distinction
Weak thermal-limit plasticity		
Broad multi-species evidence	Small acclimation-related shifts in thermal limits	Limited buffering capacity
Thermal limits do not represent every resilience component	Expand taxonomic and life-stage coverage	Evidence-supported distinction
Assay-dependent apparent null		
Rapid cold hardening	Bulk enzyme assay unchanged while in-situ function improves	Neural performance enhanced
Measurement scale can obscure functional regulation	Use orthogonal biochemical and physiological assays	Evidence-supported distinction
Asymmetric acclimation		
Heat versus cold pretreatment	Positive, null or adverse hardening effects	Stress-specific tolerance changes
Prior exposure is not intrinsically beneficial	Test reciprocal and ecologically realistic stress sequences	Evidence-supported distinction
Molecular compensation with declining performance		
High-temperature or metabolic-failure contexts	Responsive pathways remain active	Development or performance deteriorates
Response activation is not equivalent to resilience	Link pathway perturbation to whole-organism restoration	Evidence-supported distinction
Resilience boundary		
Cross-level synthesis	Protective capacity no longer maintains or restores function	Persistent impairment or failure
Exact thresholds are taxon-, stage- and environment-dependent	Quantify transition conditions without assuming universality	Proposed synthesis

The combined evidence motivates a state-space representation in which resilience is constrained by both biological capacity and evidential uncertainty.

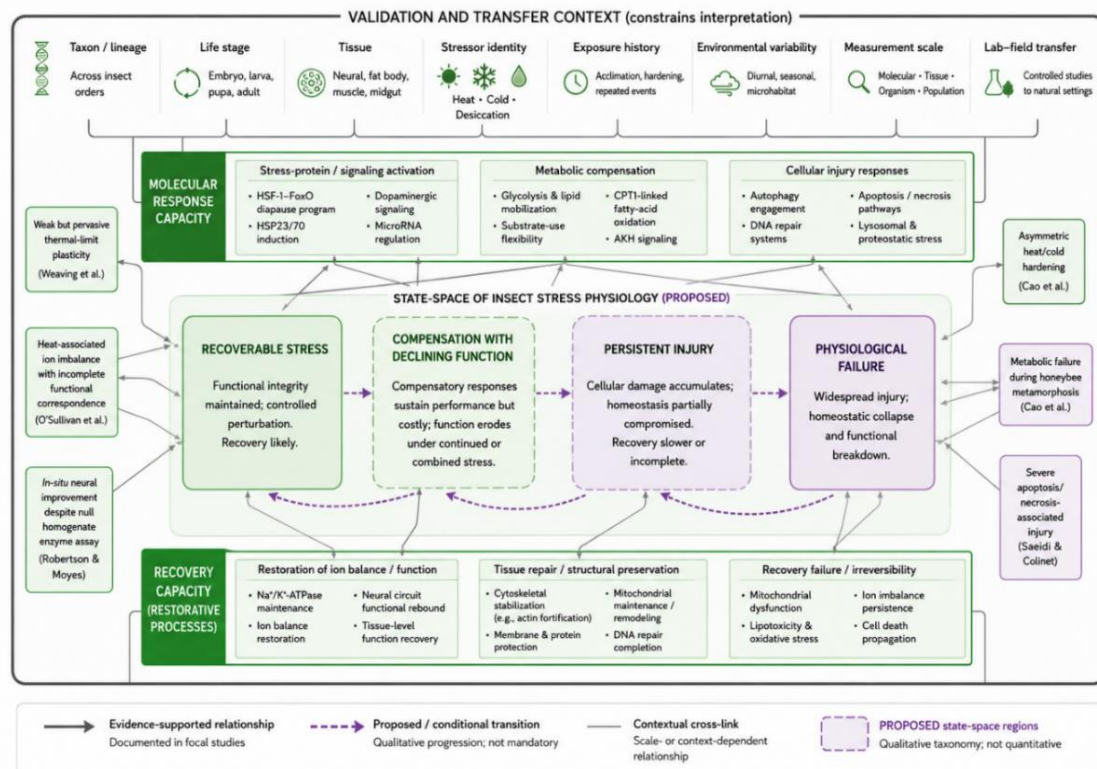


Figure 2. Physiological resilience occupies a bounded state space rather than an unlimited axis of molecular responsiveness

Contradictions across molecular and organismal evidence

The strongest contradiction is that molecular activation and organismal success need not move together. hsp23 function changes sign across thermal extremes [14], while heat-responsive chitin-related remodeling can coexist with deteriorating performance [23]. Cold-induced metabolic reconfiguration can likewise become part of developmental failure rather than protection [24]. These cases make response magnitude unsuitable as a stand-alone resilience index.

Null findings are equally informative. Survivable freezing without HSP70 induction shows that absence of one canonical marker does not imply absence of protective biology [21]. Likewise, disagreement between homogenate and in-situ Na⁺/K⁺-ATPase measurements demonstrates that evidence can change with analytical scale [35]. Transfer across settings is another source of contradiction. Molecular responses differ among life stages [9], field and laboratory metabolomes can diverge despite similar cold-tolerance phenotypes [10], seasonal field expression can depart from laboratory expectations [11], and even broad comparative metabolomics retains strong lineage specificity [26]. Consequently, apparent mechanistic disagreement may sometimes represent real biological plurality rather than experimental error.

Integrated resilience architecture and research priorities

The evidence supports a proposed resilience architecture in which environmental challenge first generates molecular and physiological responses whose significance depends on functional mediation, protection or repair, metabolic consequences, recovery, and ultimately retained organismal performance. Failure can occur at any transition, and successful response at one level does not guarantee success at the next. The architecture therefore treats molecular response as necessary evidence in some systems but never as a sufficient resilience criterion. Mechanistic confidence should increase when associations are supported by perturbation, orthogonal measurement, temporal ordering, and organismal endpoints. Functional miRNA manipulation, endocrine-pathway perturbation, symbiont-repair disruption, candidate-gene RNA interference, and disagreement between biochemical and in-situ physiological assays illustrate why evidence classes should not be treated as equivalent. Future work should particularly connect molecular mechanisms to repeated and ecologically realistic stress sequences, multiple life stages, tissue-specific dynamics, field conditions, and recovery endpoints. Epigenetic

mechanisms offer an important extension because DNA methylation, histone modifications, and noncoding RNAs are increasingly associated with insect stress responses, yet causal, transgenerational, and fitness-linked evidence remains uneven [37]. Stress-associated epigenetic change should therefore remain distinct from demonstrated adaptive evolution.

The architecture does not assign a universal threshold or score. Its purpose is interpretive: to identify which evidential transition has actually been demonstrated, where inference remains conditional, and what validation would be required before extending a focal molecular mechanism across taxa or environments.

Conclusion

Insect environmental resilience is not equivalent to the magnitude of molecular response. Plastic regulation, stress signaling, proteostasis, repair, metabolic reconfiguration, recovery, and physiological memory can each contribute to persistence of function, but their effects depend on stressor identity, developmental state, tissue, taxon, exposure history, and measurement scale. The same response may be protective, neutral, costly, or associated with failure under different conditions.

The proposed architecture therefore places explicit evidential boundaries between molecular activation, functional mediation, cellular maintenance, recovery, and organismal performance. It also makes physiological limits central rather than treating resilience as indefinitely extensible plasticity. Progress will depend less on cataloguing additional stress-responsive molecules than on establishing when particular responses causally preserve or restore function, when they merely compensate for injury, and when they mark the approach to irreversible failure.

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