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Flexible Fuel Use and Redox Regulation Coordinate Insect Survival Under Nutritional and Environmental Stress

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ABSTRACT

Insects survive starvation, heat, and infection by reallocating substrates while maintaining cellular protection and repair. These processes are usually treated separately, obscuring that the value of a metabolic response depends on tissue demand, timing, physiological state, and damage-control capacity. This article develops a non-empirical physiological framework in which survival depends on coordination rather than maximal fuel release or indiscriminate suppression of reactive oxygen species (ROS). Evidence from *Drosophila* and selected lepidopteran and hemipteran systems shows rapid nutritional-state remodeling, regulated carbohydrate and lipid mobilization, amino-acid recycling and acquisition responses, and context-dependent redox signaling. The same literature identifies failure modes: dysregulated mobilization can accompany poorer starvation survival, sustained immune-driven release can deplete reserves, and redox effects vary by tissue, life stage, age, sex, and stress protocol. The proposed architecture therefore separates fuel availability, mobilization control, tissue demand, redox signaling, oxidative damage, and repair capacity. It is intended as a testable organizing framework, not a validated universal mechanism.

Keywords: Adipokinetic hormone, Metabolic flexibility, Nutrient stress, Reactive oxygen species, Redox homeostasis, Insect immunometabolism

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Introduction

Stress survival is often reduced to an energetic problem: stored fuels are mobilized and protective pathways consume them. Yet insect immune activation can remodel cellular metabolism while reallocating systemic resources, and prolonged demand can turn useful allocation into depletion [1]. The relevant problem is therefore not fuel abundance alone, but whether mobilization, tissue demand, redox signaling, and damage control remain matched through time.

Adult male *Drosophila melanogaster* rapidly reorganizes metabolism between fasting and refeeding, with diet altering the separation between some hunger and satiety states [2]. Trehalose metabolism can also buffer glucose homeostasis and developmental stability in *Drosophila* [3]. These findings make a static-reserve interpretation inadequate: physiological state, diet, tissue, and timing alter what a measured substrate pool means.

This article proposes a coordination framework that separates six components—fuel availability, mobilization control, tissue-specific demand, redox signaling, oxidative damage, and repair capacity. The selected literature

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establishes individual relationships among these components but not the combined architecture. The framework is therefore an evidence-bounded synthesis designed to explain why the same directional response may be useful in one context and costly in another, while retaining taxonomic, developmental, laboratory-to-field, and scale-transfer limits.

Fuel selection and metabolic flexibility

Fuel selection begins with regulated access to stored and circulating substrates. In the *Drosophila* fat body, nutrient stress engages a p53–Upd2 route that alters systemic insulin signaling and autophagy [4]. In adult corpora cardiaca, protein kinase G contributes to nutrient-stress responsiveness through adipokinetic hormone (AKH) [5]. Together, these studies support state-dependent endocrine and tissue control rather than a single automatic starvation switch.

Temporal interpretation also requires genetic caution. Loss of the clock gene *period* is associated with altered intermediary lipid metabolism and greater starvation susceptibility, but several metabolic differences disappear after genetic-background control [6]. Thus, a metabolite profile or mutant phenotype cannot be treated automatically as a conserved causal fuel-selection mechanism.

More importantly, mobilization is not monotonically beneficial. Fat-body Ire1 loss increases lipid mobilization while reducing starvation survival, implicating the Xbp1s–FoxO axis in regulated lipid homeostasis [7]. *Drosophila* STING deficiency likewise alters energy-store metabolism and increases sensitivity to starvation and oxidative challenge [8]. Neither study identifies an optimal flux or proves a universal survival pathway. They instead show that disturbed regulation of reserves can coexist with poor stress tolerance.

Metabolic flexibility is therefore defined here, as a proposed construct, as state-appropriate adjustment of substrate access, storage, and use while preserving future physiological options. It is distinct from large reserves, rapid mobilization, or one measured metabolite pool. The appropriate unit of interpretation is the relationship among reserves, endocrine control, tissue demand, and stress duration.

Carbohydrate–lipid switching

Starvation can change substrate contribution over time without following a universal carbohydrate-to-lipid schedule. In *Helicoverpa armigera* larvae, starvation depresses respiratory metabolism, alters respiratory quotient, and depletes energy stores, a pattern compatible with changing carbohydrate and lipid use [9]. Respiratory quotient and pool depletion, however, do not uniquely identify molecular flux, and a larval lepidopteran trajectory cannot define switching in other stages or orders.

Endocrine evidence is similarly non-monotonic. In *Drosophila*, AKH-abundant and AKH-deficient states differ in reserves and starvation resistance, while females are more resistant than males in the tested context [10]. Stronger mobilization signaling therefore need not yield longer survival. Ion transport peptide additionally regulates intake, expenditure, carbohydrate and lipid stores, and AKH signaling [11], indicating distributed endocrine control rather than a single glucagon-like switch.

Within the proposed framework, carbohydrate–lipid switching is consequently treated as context-dependent reweighting of available fuels. Sex, genotype, baseline reserves, tissue requirements, and duration may alter its value. Claims about actual pathway flux should therefore require direct or time-resolved flux evidence rather than inference from reserve pools, respiratory proxies, or endocrine phenotype alone.

Amino-acid mobilization

Amino acids are simultaneously fuels, structural substrates, and signals. In *Bombyx mori* larvae, nutritional state regulates storage-protein expression through insulin-like/FoxO signaling [12]. This demonstrates regulated protein-reserve handling, but storage-protein expression is not a direct measurement of whole-body amino-acid flux or survival benefit.

Fasting can also recruit intracellular amino-acid pools. In *Drosophila* tissues, lysosomal cystine mobilization influences cysteine-related metabolism, TORC1 reactivation, autophagy, and tissue growth [13]. The study establishes an organelle-level recycling mechanism but not a universal route for all amino acids or insect stages. Protein restriction can additionally be sensed through tyrosine-sensitive ATF4/mTORC1 signaling that alters compensatory feeding [14]. Essential-amino-acid deficit can recruit a microbiome–gut–brain response in which enterocyte CNMa signaling promotes appetite and microbial amino-acid supply modifies the response [15].

These studies reveal three non-equivalent responses to protein shortage: regulation of stored protein, intracellular recycling, and compensatory acquisition. The proposed framework keeps them separate because releasing endogenous substrates is physiologically different from increasing external intake. Which route predominates should be expected to depend on developmental stage, tissue, diet composition, microbiome state, and food access; the current evidence does not establish a universal hierarchy among them.

Redox homeostasis

Reactive oxygen species can be damaging oxidants, physiological signals, or both. During acute heat stress in adult *Drosophila*, complex-I reverse electron transport generates mitochondrial ROS, and suppressing that signal reduces heat-stress survival [16]. In this setting, lower ROS is not synonymous with improved homeostasis; provenance and physiological role matter.

Drosophila intestinal stem cells also show context-dependent responses to intracellular ROS [17]. Because these are cellular endpoints, they cannot be translated directly into whole-animal survival, but they establish an important scale boundary. A specialized life-history example comes from *H. armigera* pupal diapause, where physiological ROS participates in insulin-linked lifespan regulation [18]. Diapause therefore demonstrates a signaling role outside *Drosophila* while remaining inappropriate as a general model of acute adult stress.

Protective capacity changes interpretation further. In *Drosophila*, oxidative preconditioning can induce CncC/Nrf2-linked and 20S-proteasome responses, but inducibility and subsequent resistance differ with age and sex [19]. No universal hormetic dose follows from these data; rather, the organism's ability to respond is itself state-dependent.

The synthesis proposed here is a conditional redox state space defined by three dimensions: ROS provenance and role, biological context, and inducible protective capacity. Signaling utility and oxidative liability can therefore coexist. The critical question is not whether ROS is simply high or low, but whether a particular signal remains interpretable and containable within the current tissue and repair state.

Because prose cannot display these cross-scale boundaries compactly, **Table 1** distinguishes evidence-supported redox states from the article's proposed synthesis without converting them into a score.

Table 1. Separating signal-bearing ROS from oxidative liability across scale and physiological state

Signal-bearing states	
Complex-I/RET signal-bearing ROS	*Evidence-supported*
Biological context	Adult Drosophila, acute heat
Evidence establishing the redox distinction	Mitochondrial ROS manipulation with survival outcome
Meaning for redox regulation	**Where inference stops**
Some mitochondrial ROS can support heat-stress signaling	Source- and stress-specific; total ROS is not equivalent
Experiment that separates the alternatives	Manipulate ROS source while measuring damage and survival
Context-dependent intracellular ROS	*Evidence-supported*
Biological context	Adult Drosophila intestinal stem cells
Evidence establishing the redox distinction	Cell-specific ROS perturbation and cell-state responses
Meaning for redox regulation	**Where inference stops**
ROS effects depend on intracellular and tissue context	Cellular response is not whole-animal survival
Experiment that separates the alternatives	Preserve tissue identity and endpoint in comparisons
Life-history-embedded ROS signaling	*Evidence-supported*
Biological context	H. armigera pupal diapause
Evidence establishing the redox distinction	ROS/insulin-pathway manipulation with diapause-longevity outcome
Meaning for redox regulation	**Where inference stops**
ROS can participate in a long-duration endocrine state	Specialized diapause cannot represent acute adult stress
Experiment that separates the alternatives	Compare equivalent stages before inferring conservation
Protection, containment, and net balance	
Inducible defense capacity	*Evidence-supported*
Biological context	Adult Drosophila differing in age and sex
Evidence establishing the redox distinction	Preconditioning, CncC/Nrf2 and proteasome responses
Meaning for redox regulation	**Where inference stops**
Tolerance depends partly on protective-system state	Age, sex and protocol modify inducibility

Experiment that separates the alternatives	Measure response capacity, not exposure alone
Signal–damage balance	*Proposed synthesis*
Biological context	Cross-context synthesis
Evidence establishing the redox distinction	Joint interpretation of source, context and defense capacity
Meaning for redox regulation	**Where inference stops**
Useful signaling requires damage to remain containable	No universal threshold or optimum has been validated
Experiment that separates the alternatives	Test provenance × damage × repair interactions

Figure 1 shows how those dimensions interact without implying a numerical ROS optimum.

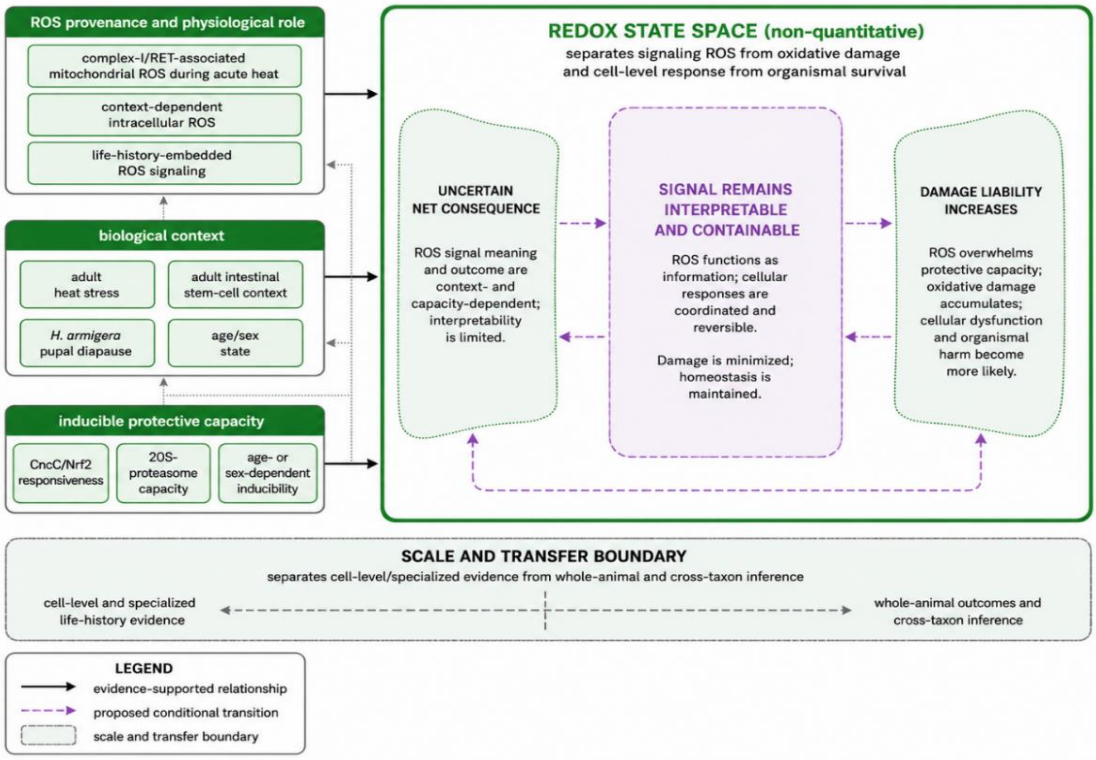


Figure 1. Redox-state trade-offs across ROS provenance, physiological context, and inducible defense capacity

Oxidative damage and repair

Redox regulation becomes consequential when damage accumulates faster than protective systems can respond. The *Drosophila* preconditioning evidence links CncC/Nrf2 signaling and 20S-proteasome capacity to inducible oxidative-stress resistance, while showing that this capacity differs with age and sex [19]. ROS exposure is therefore not interchangeable with persistent molecular damage.

Repair capacity itself is dynamic. During *Drosophila* aging, inducibility of CncC/Nrf2-dependent stress responses declines with changes in chromatin accessibility, whereas preserving stress-responsive transcription can improve aging-related phenotypes [20]. This does not establish a universal anti-aging intervention or show that Nrf2 repairs every oxidative lesion. It demonstrates that transcriptional responsiveness can become limiting.

For the proposed framework, oxidative damage and repair are separated into damage generation, damage burden, and response capacity. Existing evidence directly supports only parts of this partition, especially proteasome- and CncC/Nrf2-linked responses in *Drosophila*; the three-part structure is a proposed analytical device. A stress state may therefore fail because fuel delivery is inadequate, because a redox signal becomes excessive or misplaced, because lesions accumulate, or because repair cannot be induced. Conversely, suppressing ROS may be unhelpful when it also removes an informative signal. Repair capacity consequently acts as a constraint on the value of both fuel mobilization and redox signaling, preparing the later comparison of starvation, heat, and infection without assuming that these stresses share one molecular mechanism.

Metabolic responses to starvation, heat, and infection

Starvation, heat, and infection impose different physiological demands, so their metabolic signatures should not be collapsed into a common “stress response.” Starvation primarily constrains substrate availability and duration

of reserve use. Heat can simultaneously perturb macromolecules, membranes, respiration, and redox signaling. Infection adds a further complication because host tissues and immune cells can compete for, or redirect, circulating substrates.

In the green peach aphid *Myzus persicae*, short heat exposure changes hydrogen-peroxide and lipid-peroxidation markers, antioxidant-enzyme activities, and heat-shock-protein expression [21]. These measurements establish a biochemical and transcriptional response to heat, but they do not demonstrate that greater marker induction improves survival, represents evolutionary adaptation, or predicts field performance. Heat therefore requires separation of molecular response from organismal consequence.

Infection demonstrates an explicit allocation trade-off. In *Drosophila*, extracellular adenosine can promote systemic glucose mobilization during antibacterial defense; acute mobilization can support defense, whereas prolonged mobilization can deplete glycogen and alter host–pathogen outcome [22]. At the cellular level, macrophages increase aerobic glycolysis during antibacterial defense, and disruption of HIF/LDH-linked glycolytic reprogramming impairs effective defense [23]. Cellular demand, however, is not equivalent to whole-body fuel preference. The relevant physiological question is whether systemic supply remains sufficient for immune demand without exhausting reserves required elsewhere.

Environmental threat information may also alter metabolism before direct challenge. Parasitoid odor in *Drosophila* larvae modifies GABA metabolism and HIF-linked hematopoietic programming, priming cellular immune competence [24]. This anticipatory state differs from infection-induced metabolism and should remain analytically separate. Thermal history likewise matters: developmental temperature alters later heat tolerance, but the direction differs between acute intense and chronic mild heat challenges, while proposed mediation by cell size remains unresolved [25]. Thus, even within one stress class, protocol and developmental history can reverse interpretation.

A second distinction concerns the location of the limiting process. During starvation, failure may emerge because accessible reserves fall below continuing maintenance requirements even if redox defenses remain intact. During heat, reserves can remain available while signaling, membrane stability, proteostasis, or repair becomes limiting. During infection, adequate total reserves can coexist with a local allocation problem if immune-cell demand rises faster than substrates are delivered. These are proposed interpretations, not demonstrated universal sequences. Their value is to prevent a shared endpoint—death or reduced performance—from being mistaken for a shared physiological cause.

The comparative lesson is not that starvation, heat, and infection share one mechanism. It is that each stress changes the relationship among substrate availability, tissue demand, redox state, and repair capacity. The proposed framework therefore treats stress identity and duration as boundary conditions on coordination rather than as interchangeable inputs.

Integrated survival architecture

The central proposal is a coordination architecture in which survival depends on whether resource supply, tissue demand, redox signaling, and protective capacity remain mutually compatible over the relevant time interval. Across nutrient stress and infection, regulation rather than maximal mobilization is supported by evidence that fat-body signaling controls systemic state, dysregulated lipid mobilization can accompany poor starvation survival, and prolonged immune-driven glucose mobilization can deplete reserves [4, 7, 22]. This does not establish an optimum; it establishes that directional increases in mobilization cannot be assumed to be beneficial.

The proposed architecture contains four interacting physiological layers. First, a state-appraisal layer integrates current nutrient availability, endocrine status, and stress information. Fat tissue in *Drosophila* can integrate oxygen and nutritional information and remotely regulate insulin secretion and growth [26]. That study concerns developmental growth under oxygen limitation rather than starvation, heat, or infection survival, so it supports only the plausibility of multi-input tissue sensing. Second, a fuel-access layer controls carbohydrate, lipid, and amino-acid availability through endocrine mobilization, storage, recycling, or acquisition. Third, a demand-and-redox layer represents tissue-specific consumption and signaling requirements. Fourth, a damage-and-repair layer constrains whether the preceding responses remain sustainable.

The layers are not intended as a serial pathway. Fuel access can alter redox state through substrate delivery and mitochondrial use; redox signaling can alter endocrine or transcriptional responses; repair processes consume resources and can therefore feed back on reserve availability. Conversely, depletion or accumulated damage changes the physiological state on which subsequent decisions operate. These reciprocal relations are proposed

cross-level connections. Their inclusion makes the architecture explicitly dynamic: a response that is appropriate early in stress can become maladaptive later if reserves fall, tissue demand persists, or repair capacity declines. The same nominal stressor can therefore move an individual between coordination states without requiring a change in external exposure.

Time is not an external decoration on this architecture. Developmental diet can alter later *Drosophila* metabolic and lifespan outcomes in an adult-environment-dependent manner through a study-specific lipid-autotoxin mechanism [27]. The broader inference permitted here is history dependence, not deterministic developmental programming. Current measured state may therefore misrepresent physiological capacity if developmental history, age, sex, previous stress, or environmental carry-over is ignored.

The architecture predicts several distinct failure modes rather than one generic energetic failure. A fuel-limited state occurs when accessible substrate cannot meet essential demand. A depletion-prone state occurs when mobilization outpaces sustainable use and reserve preservation. A redox-mismatch state occurs when signaling requirements and ROS control become incompatible. A repair-limited state occurs when damage accumulates faster than inducible protection or clearance. An allocation-mismatch state occurs when one tissue, such as activated immune cells, draws resources in a way that compromises other functions. These categories are proposed analytical states; they are not empirically validated classes or ranked severities.

Because these failure modes can produce similar survival outcomes through different physiological routes, **Table 2** separates their evidential signatures and the observations needed to distinguish them.

Table 2. Distinguishing coordination failure modes within the proposed insect survival architecture

Fuel-access failures			
Fuel-limited coordination			*Proposed synthesis*
Stress setting		**Observable signature**	
Nutritional stress or sustained demand		Declining accessible reserves with unmet physiological demand	
Location of coordination failure			
Supply may become insufficient for essential functions			
Uncertainty	Pool depletion alone does not identify flux or causal limitation	**Discriminator**	Combine time-resolved substrate flux with tissue demand
Depletion-prone mobilization			*Proposed synthesis*
Stress setting		**Observable signature**	
Starvation or prolonged immune allocation		Strong or sustained mobilization accompanied by reserve exhaustion or poorer survival	
Location of coordination failure			
Mobilization can exceed sustainable benefit			
Uncertainty	Genetic or infection-specific pleiotropy may contribute	**Discriminator**	Manipulate mobilization while tracking reserve preservation and survival
Redox-containment states			
Signal-containable redox state			*Proposed synthesis*
Stress setting		**Observable signature**	
Defined heat or physiological signaling context		Source-specific ROS signal with preserved protective capacity	
Location of coordination failure			
ROS can carry useful information without implying absence of damage			
Uncertainty	Source, tissue, stage, and stress regime constrain inference	**Discriminator**	Measure ROS provenance, damage, protection, and survival together
Redox-mismatch state			*Proposed synthesis*
Stress setting		**Observable signature**	
Heat or oxidative challenge		ROS response no longer matched by protective capacity	
Location of coordination failure			
Signaling and oxidative liability may become physiologically incompatible			
Uncertainty	No universal threshold has been established	**Discriminator**	Perturb ROS source and repair capacity independently

Repair and allocation failures			
Repair-limited coordination			*Proposed synthesis*
Stress setting		**Observable signature**	
Aging or repeated oxidative challenge		Reduced inducibility of protective transcription or proteostasis	
Location of coordination failure			
Damage tolerance can become limited by response capacity			
Uncertainty	Evidence is strongest in Drosophila aging paradigms	**Discriminator**	Measure lesion burden and inducible repair separately
Allocation mismatch			*Proposed synthesis*
Stress setting		**Observable signature**	
Infection or competing tissue demands		Elevated immune-cell demand plus systemic substrate reallocation	
Location of coordination failure			
Local defense can impose systemic opportunity costs			
Uncertainty	Pathogen, tissue, and duration may change direction of benefit	**Discriminator**	Resolve tissue-specific uptake alongside whole-body reserves
Historical misclassification			
History-misclassified state			*Proposed synthesis*
Stress setting		**Observable signature**	
Developmental or prior-stress carry-over		Similar current conditions but divergent later performance	
Location of coordination failure			
Present state may be insufficient without physiological history			
Uncertainty	Mechanisms may be species- and environment-specific	**Discriminator**	Cross current stress with controlled developmental history

Figure 2 compresses these layers and feedbacks while preserving the distinction between directly supported component relationships and the article's proposed cross-level coordination logic.

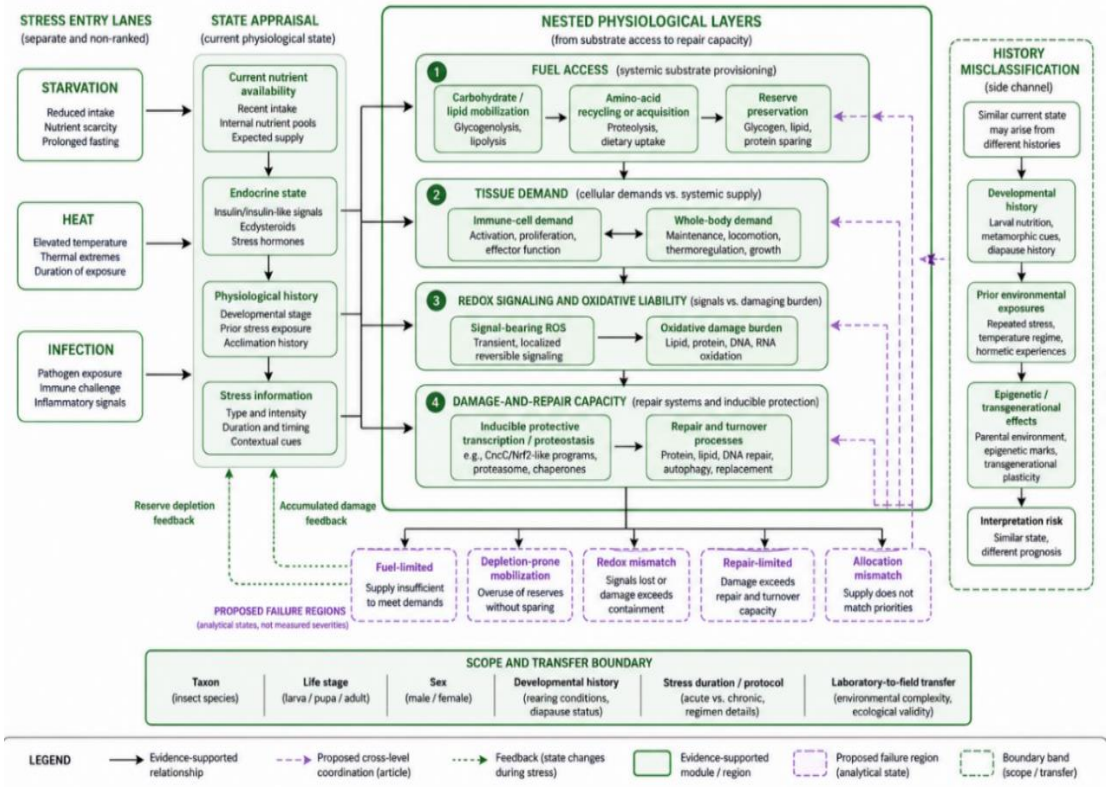


Figure 2. Coordination and failure boundaries linking fuel access, tissue demand, redox signaling, and repair

Predictions

The framework yields testable predictions, but none should be read as prospectively validated. First, survival under nutrient shortage or sustained immune demand should be explained better by context-matched mobilization plus reserve preservation than by maximal mobilization alone. The Ire1 experiment shows that enhanced lipid mobilization can coexist with poorer starvation survival [7]. AKH manipulation also demonstrates that mobilization signaling and starvation resistance need not vary monotonically [10]. During infection, prolonged glucose mobilization can become depleting [22]. A decisive test would manipulate mobilization independently of initial reserve size while measuring tissue uptake, reserve depletion, and survival through time.

Second, the relationship between ROS and survival should depend on ROS provenance, tissue, stress regime, and repair capacity. Complex-I-derived ROS can contribute to heat survival under defined conditions [16]. Cellular ROS responses vary with intracellular context [17]. Oxidative adaptive homeostasis also changes with age and sex [19]. The proposed prediction is therefore conditional, not a universal U-shaped curve: identical bulk ROS measurements could correspond to different physiological states if source and protective capacity differ.

Third, modifiers should alter which coordination point becomes limiting. Trehalose buffering illustrates developmental nutritional-state dependence [3], oxidative-defense inducibility varies with age and sex [19], acute and chronic heat tolerance respond differently to developmental temperature [25], and developmental diet can carry effects into later environmental contexts [27]. Future tests should therefore cross stress identity with life stage, sex, prior nutritional history, and stress duration rather than treating these factors as nuisance variation. No hierarchy among modifiers is predicted a priori.

A useful falsification criterion follows. If direct manipulation shows that survival is consistently predicted by one component alone—such as mobilization magnitude or bulk ROS concentration—after tissue demand, repair capacity, history, and stress duration are controlled, the coordination framework would require simplification. Conversely, support would increase if similar survival outcomes are repeatedly produced by different measured failure points and if interventions that restore matching among components improve performance without necessarily maximizing any single response.

Results and Discussion

The evidence supports a shift in emphasis from isolated stress pathways toward coordination among metabolic supply, tissue demand, redox signaling, and damage control. This article's contribution is the proposed architecture that organizes those relationships while preserving their separate evidential status. It does not claim that insects possess one conserved stress-survival program, nor that the architecture has been empirically validated as a predictive model.

Two one-direction interpretations are particularly difficult to sustain. First, mobilization cannot be equated with benefit: endocrine and fat-body manipulations show that greater or prolonged substrate release may coexist with poorer survival or reserve depletion. Second, ROS cannot be equated simply with damage. Redox effects are conditional on source, tissue, life-history state, age, and sex [16, 17, 19]. The useful distinction is therefore between an interpretable, containable signal and a redox burden that exceeds protective capacity, while acknowledging that current experiments seldom measure every component simultaneously.

Transferability is the major limitation. Genetic-background control changes interpretation of some *Drosophila* lipid phenotypes [6]. Whole-animal starvation evidence from *H. armigera* larvae relies partly on respiratory and reserve proxies rather than direct pathway flux [9]. Storage-protein regulation in *B. mori* is stage-specific and does not directly quantify amino-acid mobilization [12]. Heat-induced molecular markers in *M. persicae* cannot be converted automatically into fitness consequences [21]. Even within *Drosophila*, acute and chronic heat assays respond differently to developmental history [25]. These boundaries make broad cross-order or field generalization premature.

A further limitation is level-of-analysis mismatch. Cell-specific glycolysis, tissue redox signaling, circulating substrates, whole-body reserve depletion, and survival are related but non-equivalent outcomes. A molecular response can support one mechanism without establishing organismal benefit; an organismal survival difference can occur without identifying the causal tissue or flux. The architecture is useful only if these levels remain visible rather than being compressed into a single "stress tolerance" variable.

The framework also has a deliberate non-evolutionary scope. Context-sensitive regulation may be adaptive, neutral, constrained, or historically contingent; the physiological experiments assembled here do not resolve those

alternatives. Demonstrating evolutionary adaptation would require comparative or selection evidence linking the proposed coordination states to heritable fitness consequences. Likewise, laboratory survival is not equivalent to ecological persistence. Field validation would need to preserve natural thermal variability, food intermittency, pathogen exposure, behavior, and reproductive state rather than merely transplant laboratory thresholds into field conditions.

Validation should therefore be prospective and multi-level. Time-resolved metabolomics demonstrates that nutritional state can change rapidly [2], but future work needs direct flux measures where feasible. Mitochondrial redox interventions show the importance of ROS source [16], whereas infection studies demonstrate temporally changing allocation costs [22]. Thermal experiments show that protocol choice can reverse apparent tolerance patterns [25]. Strong tests of the framework would combine substrate flux, tissue-specific uptake, redox provenance, molecular damage, repair inducibility, and organismal survival under matched temporal designs. Replication across orders, stages, sexes, diets, and ecologically realistic environments would be required before claiming broad predictive or evolutionary significance.

Accordingly, validation should compare alternative causal explanations rather than seek confirmation of a single diagram. A useful study would distinguish inadequate supply, excessive mobilization, redox dysregulation, and repair failure under the same survival endpoint.

The framework also suggests that apparently contradictory studies may sometimes measure different coordinates of the same physiological problem. High circulating substrate could reflect effective mobilization, impending depletion, or low tissue uptake. Elevated ROS could reflect signaling, injury, or both. Increased antioxidant expression could indicate successful compensation or severe challenge. These alternatives cannot be separated by endpoint direction alone. Mechanistic interpretation therefore requires measurements that identify where resources move, which tissue demands them, where ROS originates, what damage occurs, and whether repair capacity changes concurrently.

Conclusion

Insect survival under nutritional and environmental stress is better represented as a coordination problem than as the maximization of stored-fuel release or minimization of reactive oxygen species. The proposed architecture separates fuel access, tissue demand, redox signaling, oxidative damage, repair capacity, and physiological history while allowing feedback among them. Its main value is explanatory discipline: the same directional metabolic response can have different consequences depending on timing, tissue, life stage, sex, prior state, and stress regime. Current evidence establishes many component relationships but not the integrated architecture itself, and much of the mechanistic foundation remains concentrated in laboratory *Drosophila*. Testing the framework will require time-resolved, multi-level measurements and broader taxonomic and environmental validation.

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