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Temperature, Nutrition, Infection, and Reproductive Allocation Interact to Determine Life-History Pace Under Compound Stress in Insects

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ABSTRACT

Insects rarely experience temperature, food availability, infection, and reproductive demand as independent pressures. Yet these drivers are commonly interpreted one at a time, obscuring how their timing, biological level, and internal-state dependence reshape development, survival, defense, and reproduction. This article develops a proposed compound-stressor architecture in which life-history pace is treated as a multidimensional state rather than a single rate of development or reproduction. The framework separates thermal demand, nutritional constraint, infection costs, and reproductive allocation before examining their pairwise and higher-order interactions. Evidence from diverse insect systems shows that interaction direction is not fixed: nutritional history may strengthen, weaken, or fail to modify infection costs; thermal buffering may protect survival without rescuing reproduction; and recovery intervals may reverse expectations based on cumulative exposure. The framework therefore emphasizes stage, sex, sequence, resource state, tissue capacity, and endpoint identity as boundary conditions. It predicts that simultaneous stresses are most likely to expose shared-capacity conflicts, whereas sequential stresses can produce either carry-over damage or recovery depending on the residual state entering the next challenge. The architecture is intended as a testable organizing model, not an empirically validated universal mechanism.

Keywords: Compound stress, Life-history pace, Thermal physiology, Nutritional ecology, Host–pathogen interaction, Reproductive allocation.

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Introduction

Life-history pace is often summarized through correlated traits such as development rate, activity, reproduction, and survival, but those traits need not respond coherently when environmental demand changes. In damselfly larvae, a faster pace-of-life covaried with lower thermal-performance position, showing that life-history expression can be constrained by performance context rather than varying independently of it [1]. For compound stress, this matters because a change in one trait cannot safely be interpreted as a whole-organism acceleration or deceleration.

Nutrition, immunity, and infection provide a second reason to reject single-axis interpretations. Their relationships depend on nutrient identity, host state, pathogen biology, and ecological context rather than following a universal

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resource-deficit rule [2]. Reproduction is similarly temperature-sensitive across multiple behavioral and physiological stages, with substantial variation among taxa, sexes, and reproductive processes [3]. Experimental manipulation of Toll signaling in Tribolium castaneum further shows that increased resistance can coexist with nonlinear costs to fecundity and longevity [4]. A multifactorial honey-bee experiment demonstrates an additional complication: nutritional context can change the magnitude and direction of responses to other co-occurring stressors [5]. These findings do not establish one common four-way mechanism, but they establish that the components relevant to life-history pace are conditional and potentially nonadditive.

The central problem addressed here is therefore inferential. Temperature, nutrition, infection, and reproduction are routinely measured as separate explanatory domains, whereas an insect encountering them together must respond through one physiological state. The proposed contribution is a compound-stressor architecture that keeps those domains analytically separate while asking when they converge on shared limits, when they remain effectively independent, and when prior exposure changes the state in which a subsequent stressor is experienced. Context dependence across nutritional–immune and multistressor evidence reinforces the need for this separation [2, 5]. Stage, sex, taxon, temporal sequence, environment, and measurement level are consequently treated as possible moderators rather than nuisance variation.

Materials and Methods

This framework was constructed by separating four domains—thermal demand, nutritional constraint, infection costs, and reproductive allocation—and then mapping relationships directly supported by the cited literature before proposing cross-domain synthesis. The approach does not pool effect sizes or assume a common stress metric. Evidence is interpreted at the level at which it was measured: molecular responses remain distinct from organismal performance, trait responses remain distinct from population consequences, and laboratory effects are not automatically transferred to field conditions.

Three design principles organize the architecture. First, interaction is not assumed merely because two stressors co-occur. In Aedes aegypti, resource depletion altered the shape of the thermal fitness relationship, demonstrating context dependence [6], whereas larval diet in Drosophila melanogaster altered development and reproduction without modifying the adult survival or reproductive consequences of infection in the tested design [7]. Second, temporal position is treated as part of exposure itself: in Nicrophorus vespilloides, heatwave effects on reproduction depended strongly on whether exposure occurred during mating rather than before or after it [8]. Third, internal capacity is considered a moderator. In D. melanogaster, an energetically compromised mitonuclear genotype exposed immunity–fecundity trade-offs that were not similarly expressed in controls [9].

Accordingly, the framework distinguishes supported single-domain effects, supported interactions, null or context-dependent interactions, and proposed cross-domain predictions. “Life-history pace” is used as a descriptive multidimensional state integrating development, maintenance, defense, survival, and reproductive output; it is not treated as a validated latent variable or an adaptive strategy.

Results and Discussion

Temperature changes the energetic and physiological conditions under which immunity, development, and reproduction operate, but its effects are neither uniformly detrimental nor adequately described by a single optimum. Thermal stress can alter insect immune performance through several physiological routes, with direction and magnitude varying among systems [10]. Ontogeny further changes the relevant thermal window: in Anabrus simplex, thermal performance curves differed among embryonic, hatching, and nymphal stages [11]. A stage-invariant thermal-demand term would therefore erase biologically important variation.

Thermal buffering can also protect traits unequally. In a plant-feeding hemipteran, thermoregulation enhanced survival without an equivalent rescue of reproduction [12]. Long-term high-temperature acclimation in Liriomyza trifolii produced regime-dependent combinations of thermotolerance and reproductive performance rather than a uniform benefit [13]. Developmental heat exposure in Drosophila suzukii reduced viability and later reproductive performance, while maternal thermal history modified some offspring outcomes [14]. These observations support including thermal history without interpreting every persistent response as adaptation.

Exposure sequence is equally important. Repeated cold stress separated by warm recovery periods improved ionoregulatory recovery, survival, and reproductive outcomes compared with continuous cold exposure of

equivalent total duration [15]. Thermal demand in the proposed framework therefore has three separable properties: current thermal conditions, prior thermal history, and opportunity for recovery. Their consequences must be assessed against the particular trait being measured rather than collapsed into a generic thermal-stress score.

Nutritional constraint

Nutritional constraint is not synonymous with low food quantity. Temperature can change whether stored energy is accumulated or consumed, and the same thermal condition can have different consequences during feeding and starvation. In D. melanogaster, starvation resistance depended on temperature and nutritional state, partly through lipid-storage and expenditure dynamics [16]. Nutrient timing can matter independently of mean composition: irregular fluctuations in nutrient balance reduced male reproductive output despite matched average nutrient composition [17].

Nutritional effects on infection are similarly conditional. Adult D. melanogaster showed altered survival and immune expression across macronutrient balances during bacterial challenge [18]. In Bactrocera tryoni, larval diet carried over to adult infection outcomes in a sex-specific manner, with a survival advantage detected in females but not equivalently in males [19]. Nutritional history therefore belongs in the state entering infection, but its direction cannot be generalized across sexes, pathogens, or life stages.

Specific nutrients can also change apparent allocation trade-offs. Dietary sterol availability in D. melanogaster females affected maintenance of viable egg production without producing the simple reproduction–lifespan pattern expected from a generic resource-pool model [20]. Conversely, a high-sugar diet reduced female fertility even when genetically induced obesity alone did not reproduce that effect [21]. The proposed architecture therefore separates energy quantity, macronutrient balance, limiting nutrients, temporal availability, and metabolic state. Collapsing these dimensions into one “nutrition” variable risks conflating distinct constraints and, in some cases, reversing interpretation.

Infection costs

Infection cost comprises at least two analytically distinct demands: burden imposed by the pathogen and host expenditure on defense, repair, and tolerance. High-sugar diets increased susceptibility of D. melanogaster to bacterial infection through host- and pathogen-dependent processes; antimicrobial-peptide transcription could remain induced while protein output was reduced, showing that molecular activation does not guarantee equivalent functional defense [22]. Infection therefore cannot be represented adequately by pathogen load alone or immune-gene expression alone.

A second constraint emerges when immune and reproductive functions use shared tissue machinery. Single-nucleus transcriptomics and physiological perturbation of the D. melanogaster fat body showed that simultaneous reproductive and immune demands can constrain protein translation and antimicrobial output [23]. This is a demonstrated mechanism in one female fly system, not a universal insect rule. Within the proposed architecture it exemplifies a broader candidate class—shared-capacity conflict—that should be invoked only where overlap in tissue, processing capacity, or limiting resources is demonstrated.

Infection can consequently alter life-history pace through different routes: direct pathogen damage, costs of defense, reduced performance of competing functions, or persistent changes in physiological state. Which route dominates remains contingent on pathogen identity, nutritional conditions, reproductive state, thermal history, and the biological scale of measurement.

Reproductive allocation

Reproductive allocation is treated here as a changing physiological state rather than a single output such as egg number. In female D. melanogaster, mating produced a persistent reduction in antibacterial defense that extended beyond the immediate mating period [24]. Germline manipulation in the same species showed an inverse relationship between germline activity and immune-gene expression, although this transcriptional evidence cannot be converted directly into a whole-organism fitness effect [25]. Together with demonstrated limits on fat-body protein production during concurrent reproductive and immune demand, these studies support reproductive–immune conflict at several biological levels while leaving the responsible mechanisms analytically distinct [23-25].

The boundary of that conflict is equally important. In honey-bee drones, viral infection did not measurably reduce fertility in older males under the tested conditions [26]. The null result argues against treating reproduction–immunity trade-offs as universal across sex, age, or social organization. Thermal and nutritional evidence likewise indicates that reproduction depends on the state in which allocation occurs: thermal buffering may preserve survival without restoring reproductive performance, sterol availability can relax one form of reproductive constraint, and high-sugar feeding can impair fertility through diet-specific processes.

The proposed synthesis therefore separates four reproductive-allocation dimensions: reproductive state, provisioning to reproduction, shared tissue or processing demand, and measured reproductive output. These dimensions may covary, but they need not. Declining fertility may reflect direct dietary impairment rather than strategic reallocation; reduced immune output after mating may arise through endocrine or tissue-level processes rather than depletion of a generic resource pool; and an absent fertility cost does not demonstrate absence of infection-related costs elsewhere. This separation is necessary before compound exposure can be interpreted as acceleration, slowing, or selective reorganization of life-history pace.

Because these distinctions are difficult to recover efficiently from prose alone, Table 1 separates reproductive states from their evidential basis, interpretive consequence, and boundary conditions without converting them into a composite score.

Table 1. Reproductive allocation states that change how compound-stressor effects on life-history pace should be interpreted

Biological context · Evidence basis · Biological setting · Evidence	Consequence for interpreting pace	Boundary or uncertainty · Research or test · Limits and uncertainty	Evidential standing
Persistent post-mating immune suppression			
Adult female Drosophila **Reduced antibacterial defense following mating**	Reproductive state can persist beyond the mating event and alter response to a later challenge	Female, species-specific laboratory evidence Separate current mating from persistent post-mating state	Evidence-supported pattern
Germline–immune transcriptional trade-off			
Female Drosophila **Germline manipulation accompanied by altered immune-gene expression**	Reproductive investment can covary inversely with immune transcription	Transcript level does not establish whole-organism fitness cost Pair transcript measurements with pathogen control, survival, and fecundity	Evidence-supported pattern
Shared fat-body translational constraint			
Reproductive female Drosophila under immune challenge **Concurrent demands reduce protein-production capacity**	Simultaneous functions may compete through shared tissue machinery	Demonstrated in one polyfunctional tissue and host system Test analogous shared-capacity limits in other tissues and insect orders	Evidence-supported mechanism
Nutrient-buffered reproductive output			
Adult female Drosophila under sterol limitation **Sterol availability changes maintenance of viable egg production**	Reproductive costs can depend on limiting nutrient identity rather than resource amount alone	Sterol-specific laboratory evidence Manipulate defined limiting nutrients during thermal or infection challenge	Evidence-supported pattern
Diet-specific fertility impairment			
Adult female Drosophila on high-sugar diet **Fertility declines without an equivalent effect from obesity alone**	Reduced reproduction can reflect dietary state rather than generalized energy scarcity	Diet composition and genotype remain relevant Distinguish dietary chemistry from body-condition proxies	Evidence-supported pattern
Social-male null boundary			
Older Apis mellifera drones with viral infection **Fertility not detectably reduced despite infection**	Reproduction–immunity conflict is not obligatory across sex and social context	Drone age, infection spectrum, and colony history constrain transfer Test sex-, age-, and caste-specific allocation responses	Evidence-supported boundary
Conditional reproductive-demand state			

Compound-stress setting Joint consideration of mating state, provisioning, shared capacity, and output	Life-history pace may shift through selective protection or sacrifice of reproductive functions rather than uniform slowing	Not validated as a common insect state variable Factorially manipulate stress timing while measuring multiple reproductive endpoints	Proposed synthesis

The same distinctions can be represented mechanistically only if evidence-supported links are kept separate from proposed cross-domain connections. **Figure 1** therefore maps reproductive demand onto nutritional, thermal, and infection boundaries without implying a universal causal pathway.

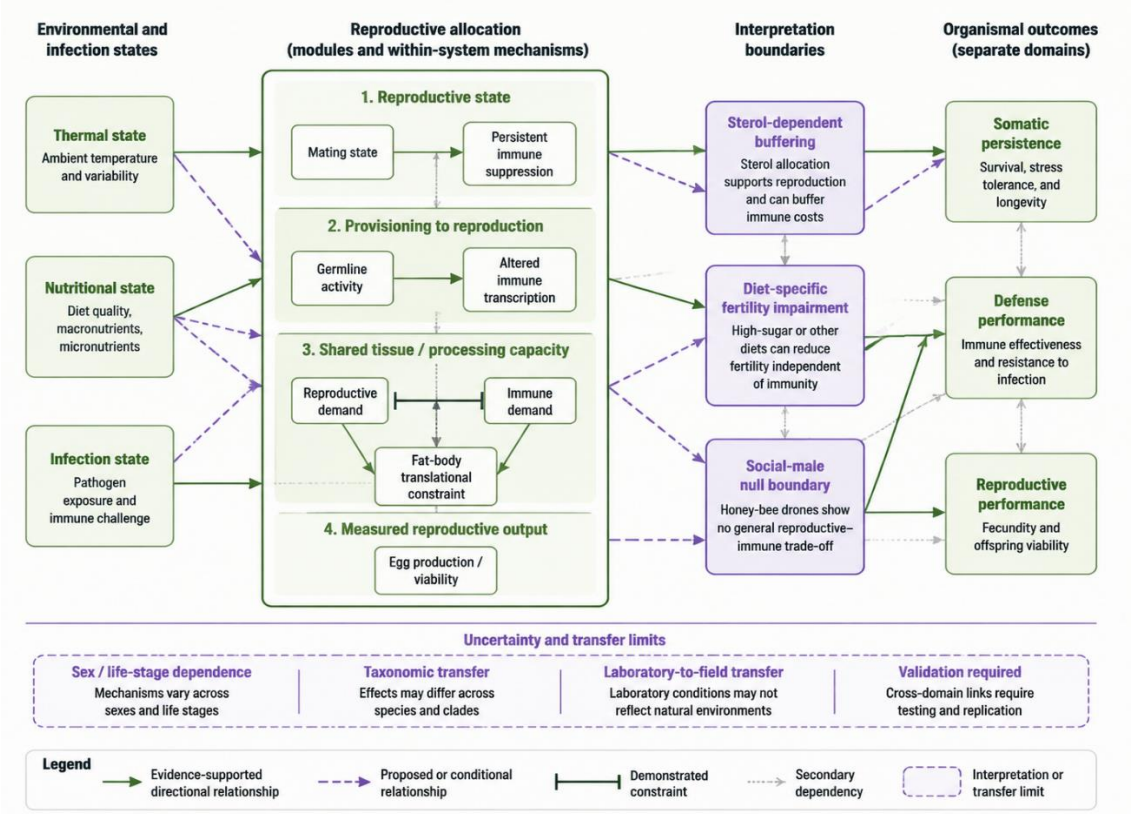


Figure 1. Reproductive demand changes compound-stress interpretation only when state, shared capacity, and endpoint boundaries remain explicit

Pairwise interactions

Pairwise interactions reveal why compound stress cannot be represented by adding independent penalties. Thermal effects are conditional on resource state: resource depletion altered the thermal fitness relationship in Aedes aegypti, while temperature changed starvation resistance through state-dependent lipid expenditure in D. melanogaster [6, 16]. These studies address different outcomes and mechanisms, but together establish that the biological meaning of temperature depends partly on available or stored resources.

Nutrition–infection relationships are still less directionally consistent. Developmental diet failed to modify infection-related survival and reproductive costs in one female D. melanogaster experiment [7], whereas larval nutrition produced sex-specific adult infection benefits in Bactrocera tryoni [19]. High-sugar feeding instead increased bacterial susceptibility through host- and pathogen-dependent processes in adult Drosophila [22]. Thus, nutritional history may buffer, intensify, or fail to alter infection costs.

Temperature–infection coupling provides a further experimentally supported interaction. A simulated natural heatwave reduced survival and antibacterial immune performance and increased susceptibility to Crithidia infection in Bombus impatiens workers [27]. This establishes thermal modification of infection resistance in that system, but not a general population-level fitness effect. Pairwise evidence therefore supports interaction-sensitive modeling while simultaneously rejecting a fixed interaction sign.

Higher-order interactions

Higher-order interactions arise when the effect of one stressor changes the relationship between two others. In honey bees, diet altered responses to combined pesticide and viral exposure, including changes in effect direction across nutritional contexts [5]. This does not directly demonstrate the temperature–nutrition–infection–reproduction combination developed here; instead, it provides evidence that a third condition can modify an otherwise pairwise relationship.

The proposed higher-order architecture therefore treats capacity, timing, life stage, sex, and stressor identity as moderators rather than assuming a universal interaction coefficient. The null developmental-diet interaction in Drosophila [7], the timing dependence of reproductive heatwave effects [8], the energetic-genotype dependence of immunity–fecundity conflict [9], the sex-specific nutritional carry-over in B. tryoni [19], and the absent fertility cost in infected honey-bee drones [26] show why superficially contradictory observations may be biologically compatible.

The evidence base does not establish a four-way insect mechanism. Table 2 therefore reconciles conflicting patterns by identifying which boundary must be resolved before an interaction can be interpreted.

Table 2. Reconciling apparently conflicting compound-stressor outcomes through state, timing, and biological-level boundaries

Higher-order interaction state	Context inversion	Null modification	Timing-gated interaction	Capacity-revealed interaction	Sex-dependent carry-over	Four-domain compound state
Observable pattern · Interpretation for compound stress	Direction or magnitude changes across nutritional contexts A third condition can reshape a pairwise effect	Diet changes life history but not infection cost Co-occurring stressors need not interact detectably	Strong cost during one reproductive window but weak before or after Overlap can matter more than cumulative exposure	Trade-off appears under constrained energetic capacity Latent interaction may emerge only below a capacity boundary	Adult benefit differs between females and males Prior state can alter later stress response asymmetrically	No directly established common response pattern Interaction sign should be treated as conditional, not assumed
Required test	Fully crossed factorial design	Replicate across pathogens and sexes	Shift identical stress across defined life-history states	Manipulate capacity independently of stressor identity	Cross-stage factorial experiment with both sexes	Multi-endpoint factorial validation
Biological setting	Social insect under diet, chemical, and viral challenge	Developmental diet followed by adult infection	Heatwave around reproduction	Energetically compromised genotype	Developmental nutrition followed by infection	Temperature, nutrition, infection, and reproductive demand jointly considered
Evidential standing · Critical uncertainty or boundary	Evidence-supported precedent Chemical identity and social-insect physiology constrain transfer	Evidence-supported boundary Sex, pathogen, diet range, and endpoint choice	Evidence-supported pattern Duration and reproductive stage	Evidence-supported pattern Genotype-specific mechanism	Evidence-supported pattern Sex and developmental stage	Proposed synthesis Taxon, stage, sequence, shared capacity, and endpoint

These reconciliations require visualization across biological scales rather than as a single causal chain. **Figure 2** therefore separates external demands, internal capacity, interaction state, and measured consequences while marking where evidence becomes proposal.

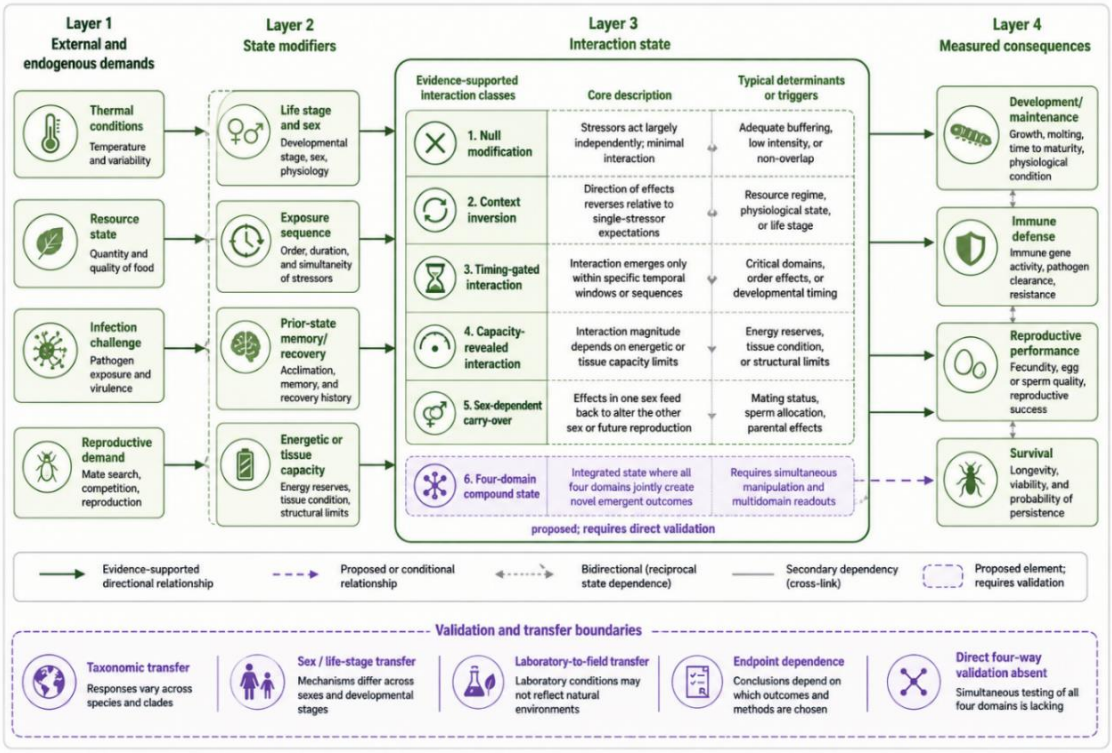


Figure 2. Higher-order stress effects emerge only after scale, sequence, and capacity boundaries are resolved

Integrated life-history pace model

The proposed model treats life-history pace as a vector of coordinated but separable rates and allocations rather than as one scalar continuum. Thermal demand modifies physiological operating conditions; nutritional state changes accessible resources and metabolic constraints; infection adds pathogen burden and defense requirements; reproductive allocation changes endogenous demand. Their joint consequence is expressed through development or maintenance, defense, survival, and reproduction rather than through an assumed common fitness currency.

Three response modes follow. Pace deceleration occurs when several functions are reduced or delayed. Selective reallocation occurs when one function is maintained while another declines, as illustrated by thermal buffering that protects survival more effectively than reproduction [12]. Conditional release occurs when a limiting component is removed or recovery is permitted, as suggested by sterol-dependent reproductive buffering [20] and interrupted-cold recovery [15]. These modes are proposed descriptive states, not adaptive categories.

The model therefore predicts no universally adverse compound-stress direction. Interaction sign depends on which biological capacity becomes limiting, which endpoint is measured, and whether previous exposure leaves damage, acclimatory change, altered resource stores, or recovery.

Predictions for sequential versus simultaneous stress

Simultaneous stress should be most likely to expose conflict where functions share limiting machinery. Concurrent reproductive and immune demands in the Drosophila fat body provide a specific mechanistic example of such overlap [23]. The framework predicts that analogous simultaneity costs will occur only when the relevant stressors converge on a shared capacity; this requires direct testing in each system.

Sequential stresses have two contrasting possibilities. Earlier exposure can leave residual damage or altered physiological state, as developmental heat does for later reproductive performance [14]. Alternatively, spacing may permit recovery, as warm intervals did during repeated cold exposure [15]. Developmental nutrition can likewise carry forward into adult infection response [19]. Sequence should therefore be represented as a state transition, not merely as accumulated dose.

Order effects must also be tested explicitly. The strong timing dependence of heatwave exposure relative to reproduction [8], the consequences of temporal nutrient composition [17], and persistent post-mating immune alteration [24] all indicate that the same nominal stressors can be encountered from different starting states. A decisive validation design would cross stressor identity, sequence, life stage, and sex while retaining multiple endpoints rather than seeking a single composite outcome.

The principal contribution of this framework is to reorganize heterogeneous insect stress physiology around a common inferential question: when does compound exposure alter life-history pace through interaction rather than through independent trait responses? The answer cannot be derived from stressor count alone. Pace-of-life associations, nutritional immunology, pairwise experiments, and higher-order precedents instead indicate that internal state and biological timing determine whether stressors reinforce, offset, or fail to modify one another [1]. Contradictory evidence is therefore informative. A null developmental-diet effect on infection [7], beneficial recovery during interrupted cold exposure [15], female-specific nutritional carry-over [19], and an absent infection–fertility effect in older honey-bee drones [26] do not invalidate compound-stress theory. They identify boundaries that a valid theory must retain.

The framework also requires strict separation of biological levels. Immune transcription, protein production, pathogen burden, survival, fecundity, and modeled population performance are not interchangeable. High-sugar infection experiments demonstrate this directly because transcriptional activation can coexist with reduced antimicrobial protein production and poorer infection outcomes [22]. Molecular responses should therefore not be described as adaptive, beneficial, or fitness-enhancing without organismal evidence.

Generality remains limited. Mechanistic evidence is concentrated heavily in Drosophila, while social insects, beetles, mosquitoes, orthopterans, odonates, and hemipterans contribute fewer but important boundary cases. Much of the evidence is laboratory-based, although heatwave simulation and mesocosm studies improve ecological realism. The proposed four-domain architecture consequently requires validation across taxa, developmental stages, sexes, pathogens, nutritional environments, and naturally fluctuating thermal conditions.

Future studies should manipulate interaction structure rather than merely increase stressor number. Factorial experiments should distinguish simultaneity from order, quantify recovery between exposures, identify shared versus independent capacities, and retain separate developmental, immune, survival, and reproductive endpoints. Only after such validation could the model support stronger claims about prediction or generality.

Conclusion

Temperature, nutrition, infection, and reproductive allocation cannot be interpreted reliably as independent determinants of insect life-history pace. Existing evidence instead supports context dependence, null interactions, outcome-specific trade-offs, carry-over effects, and recovery. The proposed framework organizes these patterns by separating stressor identity from internal state, timing, shared capacity, and measured consequence. Its central claim is therefore conditional: compound stress changes life-history pace only through the particular biological relationships actually engaged in a given insect, stage, sex, environment, and sequence. Testing those relationships directly is the necessary next step.

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