



Eurasia Specialized Veterinary Publication

Entomological Research Letters

ISSN: 3108-639X

2026, Volume 6, Issue 2, Page No: 44-56

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Available online at: www.esvpub.com/

Developmental Memory, Compensation, and Fitness Consequences Across Cross-Stage Plasticity in Insects Under Global Change

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ABSTRACT

Environmental conditions experienced during immature insect stages can alter later phenotypes, yet the meaning of those effects is often obscured by treating developmental plasticity, persistent damage, developmental memory, recovery, compensation, and adult fitness as interchangeable outcomes. This umbrella review evaluates cross-stage responses to thermal change, nutritional limitation and drought, and pollution or chemical exposure, with emphasis on how effects are retained, redirected, attenuated, or expressed after developmental transitions. Across the evidence base, global-change responses were strongly endpoint-, stage-, taxon-, and context-dependent. Thermal exposure altered developmental rates and physiological limits but did not consistently translate into beneficial adult performance. Nutritional and drought effects depended on exposure severity, host-mediated pathways, trophic context, and reproductive physiology, while null and heterogeneous carry-over effects remained important. Pollution evidence showed broad organismal toxicity but substantially stronger inference when developmental exposure was directly linked to later reproductive impairment. We therefore distinguish developmental memory from any persistent phenotype and reserve compensation for cases in which a later response offsets, rather than merely follows, an earlier cost. The emerging synthesis treats cross-stage plasticity as a conditional trajectory whose adult consequences depend on developmental gating, environmental matching, recovery capacity, endpoint choice, and scale of inference. This framework is proposed as an evidence-organizing architecture rather than a validated universal mechanism.

Keywords: Cross-stage plasticity, Developmental carry-over, Developmental memory, Global change, Insect fitness, Phenotypic plasticity.

Received: 10 August 2026

Revised: 23 September 2026

Accepted: 27 September 2026

How to Cite This Article: Williams N, Collins G, Brooks E, Green D. Developmental Memory, Compensation, and Fitness Consequences Across Cross-Stage Plasticity in Insects Under Global Change. *Entomol Appl Sci Lett.* 2026;6(2):44-56.

<https://doi.org/10.51847/HuwQWdwnBO>

Introduction

Research on insects under global change increasingly shows that responses cannot be represented adequately by a single temperature threshold, developmental rate, survival value, or reproductive endpoint. Review-level evidence spans warming, drought, chemical contamination, altered nutrition, and interacting stressors, but its coverage is uneven and its biological directions differ among traits and drivers [1]. A response measured at one stage is also insufficient to establish population relevance because survival, development, maturation, mating, fecundity, and other vital rates can contribute differently to demographic outcomes [2]. The inferential problem

is therefore not simply whether an immature insect responds to environmental change, but whether that response is retained across development and what it subsequently means for adult performance.

Ontogenetic plasticity and cross-stage carry-over must be separated analytically. Thermal tolerance and plasticity can differ among life stages, and the probability that an earlier response remains relevant later depends on developmental strategy, ecological similarity between stages, environmental predictability, injury, repair, and stage-specific exposure [3]. Metamorphosis consequently should not be assumed either to erase previous environmental effects or to preserve them. Likewise, persistence is not equivalent to benefit: a retained phenotype may represent information storage, physiological damage, constraint, or a state whose value changes when the adult environment differs from the developmental environment.

Global-change mechanisms add another layer of ambiguity. Environmental drivers can influence development, reproduction, survival, diapause, host quality, and physiological regulation through multiple direct and indirect pathways, while translation from organismal response to population consequence remains incomplete for many insect systems [4]. This review therefore uses cross-stage trajectory as a proposed organizing concept. A trajectory specifies whether an environmentally induced developmental effect persists, disappears, reverses, is repaired, or is offset by a later response, while keeping the resulting adult-fitness inference separate. This distinction is necessary because developmental memory, compensation, and adult fitness occupy different positions in the evidence chain.

The scope is restricted to evidence relevant to global-change-associated thermal conditions, nutrition and drought, and pollution or chemical exposure, together with mechanistic and methodological literature needed to interpret persistence across stages. The review does not treat plasticity as synonymous with adaptation, molecular responses as proof of organismal benefit, or laboratory carry-over as automatic evidence of field-scale population consequences.

Materials and Methods

Search and evidence organization were conducted on 26 August 2026. Six predefined search domains were used: cross-stage developmental plasticity under global change; thermal exposure; nutrition and drought; pollution and chemical exposure; developmental-memory mechanisms; and umbrella-review methodology. Search logic combined insect or arthropod terms with developmental-stage, carry-over, plasticity, fitness, environmental-driver, and review-method terms. Scholarly indexing was supplemented by publisher and PubMed/PMC-linked record verification. Reporting followed the principle that identification and selection procedures should remain explicit rather than being reconstructed retrospectively [5].

The search record retained the six executed conceptual search domains and the final manuscript evidence corpus of 39 publications. Source-specific database hit totals, deduplication totals, title/abstract exclusion counts, and full-text exclusion counts were not retained in a form permitting reliable reconstruction; those intermediate quantities were therefore not estimated or reverse-engineered. The corpus should consequently not be interpreted as an enumerated count of independently included systematic reviews. Review-level syntheses formed the principal evidence layer, whereas methodological publications and selected primary studies were used to establish boundaries, mechanisms, or direct cross-stage examples where review-level evidence alone could not support the required inference.

Eligible evidence was limited to peer-reviewed journal literature within 2017–2026 with verifiable DOI metadata and direct relevance to a planned claim. Evidence was organized by driver, insect taxon, life stage, endpoint, study setting, biological level, and whether the inference concerned immediate plasticity, cross-stage persistence, recovery, compensation, reproduction, or population consequence. Primary-study overlap among reviews was treated as a potential source of non-independence rather than as additional corroboration merely because the same underlying experiment appeared in several syntheses [6]. Review-by-primary-study matrices provide a defensible way to visualize such overlap when complete study-membership information is available [7].

Evidence certainty was considered qualitatively rather than converted into an unsupported numerical score. Ecological critical-appraisal tools vary in scope and are inconsistently used, making direct importation of a single checklist inappropriate [8]. Domain-based review appraisal remains useful, but healthcare-oriented tools such as AMSTAR 2 require adaptation before they can inform ecological inference [9]. Accordingly, later certainty judgments consider directness, review quality, consistency, primary-study overlap, taxonomic and life-stage

breadth, laboratory-to-field transfer, endpoint relevance, and whether a claimed mechanism was functionally validated.

Results and Discussion

Temperature changes developmental biology at several non-equivalent levels. A meta-analysis of butterflies found that higher developmental temperatures generally accelerated growth and development and shortened developmental duration, whereas effects on body mass were weaker and more heterogeneous [10]. Such developmental acceleration is a trait response, not evidence that adult fitness improved. Across insects more broadly, plasticity in critical thermal limits is widespread but usually modest, with juvenile stages showing stronger plasticity than adults in the compiled evidence [11]. Immediate stage-specific plasticity therefore cannot be treated automatically as a retained cross-stage state.

Thermal regime structure adds further conditionality. Across ectotherm studies dominated by invertebrate systems, fluctuating temperatures often produced plastic responses similar to those under constant conditions, with only limited trait-specific deviations [12]. Warming responses in forest insect pests were likewise non-uniform across development, survival, and related performance endpoints [13]. These findings argue against a single direction of “thermal plasticity” and instead indicate that exposure pattern, endpoint, taxon, and developmental stage jointly determine the observable response.

Direct cross-stage evidence illustrates why persistence and consequence must remain separate. In *Enchenopa binotata*, elevated developmental temperature reduced juvenile survival while increasing an adult reproductive component, producing partial buffering of modeled mortality-driven population decline rather than uniform benefit across stages [14]. Extreme sporadic heat in *Culex pipiens pallens* also produced stage- and sex-dependent adult consequences following developmental exposure [15]. Such outcomes support the proposed distinction between persistent cost, redirected effect, and partial compensation, but neither experiment establishes an evolutionarily adaptive response across insects.

Recovery adds another analytical boundary. Heat exposure can create cellular or physiological injury followed by repair, so a later phenotype may reflect residual damage, successful repair, altered allocation, or a mixture of these processes rather than a simple retained acclimation state [16]. Reproductive endpoints are particularly important because heat-associated reproductive impairment may occur below lethal limits, meaning that survival alone can underestimate vulnerability [17]. Adult consequences of developmental heat therefore require explicit measurement rather than inference from juvenile survival or thermal tolerance.

Nutrition and drought

Nutritional stress is inseparable from environmental context because temperature, host physiology, water availability, and resource composition can interact before an insect phenotype is measured. Thermal and dietary stress can produce non-additive responses whose direction depends on genotype, physiological state, and developmental context [18]. Elevated atmospheric CO₂ can additionally reduce plant nutritional quality and thereby alter herbivore development, body size, reproduction, or abundance through an indirect bottom-up pathway [19]. Direct food limitation and host-mediated nutrient dilution must therefore remain distinct exposure classes.

Drought provides a similar example of pathway dependence. Meta-analytic evidence in aphids indicates generally negative consequences for aphid fitness and host-plant vigor, but individual endpoints and studies remain heterogeneous [20]. Broader synthesis of insect herbivores shows that drought and other global-change drivers can be modified by trophic pathway: bottom-up effects mediated through plants need not match top-down effects mediated through enemies, and warming can even generate opposing directions across those pathways [21]. An apparent contradiction among studies may therefore reflect different portions of the trophic system rather than irreproducibility.

Severity also matters. In *Tenebrio molitor*, moderate water deficit produced comparatively limited performance changes, whereas severe water restriction reduced growth or biomass and altered stress- and immune-related gene expression [22]. Molecular response can thus precede, accompany, or exceed detectable organismal performance change; transcriptional alteration alone does not establish beneficial acclimation. Nutritional effects can also propagate into adult reproduction through morphological, behavioral, and endocrine routes in Lepidoptera [23],

although such pathways do not demonstrate that every developmental nutritional perturbation persists through metamorphosis.

Null evidence constrains the strength of any general carry-over claim. A meta-analysis of parental dietary protein in insects and other oviparous invertebrates did not detect a consistent overall maternal or paternal effect on offspring viability and reported substantial heterogeneity across species and designs [24]. Although intergenerational transmission is biologically distinct from within-individual cross-stage plasticity, this result demonstrates that nutritional history does not automatically produce a detectable later-life consequence.

The major nutrition–drought patterns are difficult to compare in prose because their exposure routes and inferential endpoints differ. **Table 1** therefore separates direct nutritional stress, host-mediated resource change, drought severity, trophic mediation, reproductive coupling, and null carry-over evidence before any adult-fitness interpretation is made.

Table 1. Nutritional and hydric pathways that separate developmental response from later consequence

Combined thermal–dietary stress			
Taxonomic or methodological scope Multi-species insect literature Measured construct Developmental and physiological plasticity	Responses may be non-additive and depend on developmental context	Combined-stressor field validation remains limited	Single-driver effects cannot be simply added to predict later phenotype
Host nutrient dilution under elevated CO₂			
Taxonomic or methodological scope Primarily plant-feeding insects Measured construct Host quality, development, body size, reproduction	Resource quality can mediate insect response indirectly	Strongest applicability is to host-dependent consumers	Developmental history may originate in host-mediated nutrition rather than direct climatic exposure
Drought through host plants			
Taxonomic or methodological scope Aphid–plant systems Measured construct Aphid fitness and plant vigor	Predominantly negative but heterogeneous responses	Feeding guild and host physiology limit transferability	Drought effect must be assigned to the trophic pathway actually measured
Top-down versus bottom-up drought/global-change effects			
Taxonomic or methodological scope Herbivore food webs Measured construct Herbivore performance under trophic mediation	Direction can change with the trophic route	Multiple-stressor and multi-trophic experiments remain comparatively sparse	Apparent contradiction may reflect pathway mismatch rather than biological inconsistency
Severity-dependent water stress			
Taxonomic or methodological scope Larval <i>Tenebrio molitor</i> Measured construct Growth, biomass, stress/immune expression	Mild response can precede stronger performance loss under severe restriction	Laboratory rearing context	Molecular response must remain separate from developmental damage and later fitness
Diet-linked reproductive physiology			
Taxonomic or methodological scope Lepidoptera Measured construct Reproductive morphology, behavior, endocrine regulation	Adult reproduction responds to nutritional state through several routes	Evidence is taxonomically concentrated	Adult reproductive change does not by itself establish developmental memory
Weak or heterogeneous nutritional carry-over			
Taxonomic or methodological scope Intergenerational insect/invertebrate experiments Measured construct Offspring viability after parental protein manipulation	No consistent overall effect	Designs and species differ; intergenerational evidence is not within-individual carry-over	absence of consistent persistence is itself evidence against universal nutritional memory

Pollution and chemical exposure

Pollutants create a particularly important distinction between acute toxicity and genuine cross-stage persistence. A systematic review and meta-analysis of terrestrial insect–microplastic interactions found evidence of uptake and transfer together with behavioral and oxidative-stress effects, but also emphasized the relative scarcity of field evidence and problems of exposure realism [25]. A broader insect meta-analysis similarly reported adverse effects

of plastics on survival, growth, development, fecundity, and behavior, with magnitude dependent on plastic type, concentration, duration, and endpoint [26]. These syntheses support toxicity, but not the claim that every developmental plastic exposure becomes a persistent adult phenotype.

Heavy metals add trophic and toxicological complexity. Metals can move through food chains and affect growth, development, reproduction, or biological-control performance of insect natural enemies, while responses at low exposure can differ from those at higher exposure [27]. In pest insects, prior heavy-metal exposure can also alter detoxification systems and later insecticide responses, generating context-dependent cross-tolerance [28]. Cross-tolerance is not equivalent to developmental memory: it may reflect altered detoxification capacity, physiological stress, or exposure history without demonstrating retained environmental information or improved fitness.

Pesticide evidence spans several levels of inference. A global synthesis of non-target organisms indicates broad impairment of growth, reproduction, behavior, and related biological outcomes under pesticide exposure [29]. This establishes a general hazard pattern but is neither insect-exclusive nor specifically cross-stage. Direct evidence is stronger when the exposure and later endpoint are linked within the same life history. In *Osmia cornifrons*, larval deltamethrin exposure reduced subsequent emergence or body condition and impaired adult male sperm quantity and quality, with additional adult exposure compounding reproductive deficits [30]. In *Osmia lignaria*, previous imidacloprid exposure was associated with reduced later reproduction and lower modeled population growth, while repeated exposure added further impairment [31]. These solitary-bee systems demonstrate persistent cross-stage costs, but their magnitude and mechanisms should not be generalized across pesticides, sexes, species, or environmental settings without direct evidence.

The pollution literature therefore supports a graded inference: broad toxicity establishes that environmental chemicals can alter insect performance; stage-linked experiments establish persistence more directly; adult reproductive or demographic measurements strengthen the consequence claim. None of these steps, by itself, proves adaptive developmental memory.

Developmental memory

“Developmental memory” is most useful when defined more narrowly than persistence. Here, the term is proposed to mean a retained biological state or information-bearing developmental modification that influences phenotype after the inducing environment or developmental stage has changed. Persistent injury qualifies as carry-over but not necessarily as memory, whereas a molecular mark qualifies as a candidate mechanism only when its relationship to the later phenotype is demonstrated. Epigenetic processes provide plausible routes through which environmental stress can influence persistent phenotypes, but causal and functional validation remains uneven across insect systems [32].

Aphid wing polyphenism provides a clearer example of environmentally cued developmental state change: sensory and maternal information can be translated through neural, hormonal, and epigenetic regulation into alternative wing phenotypes [33]. This does not establish a universal mechanism for global-change carry-over, but it demonstrates that insect development can retain and transform environmental information through regulated developmental decisions rather than passive damage alone.

Nutritional regulation further illustrates developmental gating. In *Blattodea*, nutritional state interacts with insulin-related signaling, TORC1, AMPK, juvenile hormone, and ecdysteroids to regulate developmental outcomes [34]. More generally, environmentally responsive developmental systems differ in robustness and plasticity according to regulatory architecture and the developmental window during which environmental inputs can modify phenotype [35]. The relevant question is therefore not simply whether an environmental signal occurred, but whether it arrived while the developing organism remained capable of incorporating that signal into a later state. Persistence can also change meaning when the adult environment changes. In *Drosophila melanogaster*, developmental temperature altered later heat tolerance in assay-dependent directions; warm- versus cold-development advantages depended on the subsequent thermal challenge, while cell-size mediation remained suggestive rather than conclusively established [36]. A developmental effect can consequently persist without having a fixed sign. This supports a proposed distinction between state retention and contextual expression: the former concerns whether developmental history remains biologically detectable, whereas the latter concerns whether that retained state becomes advantageous, neutral, or costly under a particular adult challenge.

Because these categories are frequently conflated, **Table 2** reconciles evidence that can look superficially contradictory by separating information retention, regulated developmental state change, physiological gating, residual injury, cross-tolerance, and challenge-dependent expression.

Table 2. Distinguishing developmental memory from persistence, injury, and context-dependent carry-over

State and biological setting	Observable basis	Cross-stage meaning and status	Critical boundary	Discriminating test
Evidence-supported memory substrates and gates				
Developmental-memory state or inference scenario Epigenetic persistence signal Biological context Multiple insect stress contexts	Persistent epigenetic or regulatory change associated with later phenotype	Interpretation for cross-stage plasticity Candidate substrate for retained environmental effects Status Evidence-supported candidate mechanism	Association alone does not establish causal memory	Perturb the candidate mark or pathway and test the later phenotype
Developmental-memory state or inference scenario Environmentally cued developmental state Biological context Aphid wing polyphenism	Cue-dependent endocrine/epigenetic regulation linked to alternative morphology	Interpretation for cross-stage plasticity Demonstrates regulated conversion of environmental information into developmental state Status Evidence-supported exemplar	Aphid-specific architecture limits generalization	Test whether comparable cue-state coupling exists in other taxa/drivers
Developmental-memory state or inference scenario Endocrine-metabolic gating Biological context Blattodea and related developmental systems	Nutritional inputs interact with IIS/TORC1/AMPK and developmental hormones	Interpretation for cross-stage plasticity Environmental information can be incorporated only through responsive developmental circuitry Status Evidence-supported mechanism class	Pathway contribution varies among orders and traits	Manipulate timing and pathway activity across developmental windows
Developmental-memory state or inference scenario Developmental robustness versus responsiveness Biological context Broad insect developmental literature	Same exposure produces strong plasticity in some windows/traits and little change in others	Interpretation for cross-stage plasticity Absence of carry-over may reflect developmental gating rather than absence of exposure Status Evidence-supported boundary	Regulatory architecture is taxon- and trait-dependent	Map sensitive windows rather than averaging across development
Developmental-memory state or inference scenario Challenge-dependent expression Biological context <i>Drosophila melanogaster</i> developmental-temperature experiments	Adult thermal performance changes direction with the subsequent heat assay	Interpretation for cross-stage plasticity Retained developmental state does not have a fixed fitness sign Status Evidence-supported pattern	Laboratory assay and species specificity	Cross developmental and adult environments factorially
Reconciliation categories requiring stronger causal separation				
Developmental-memory state or inference scenario Chemical cross-tolerance Biological context Insect pests under prior metal exposure	Earlier chemical exposure modifies later insecticide response	Interpretation for cross-stage plasticity Prior state can alter later response without demonstrating information-like developmental memory Status Proposed reconciliation category	Detoxification, toxicity, and memory are competing explanations	Separate detoxification persistence from regulated developmental state
Developmental-memory state or inference scenario Persistent developmental injury Biological context	Developmental exposure followed by impaired adult reproductive traits	Interpretation for cross-stage plasticity Strong evidence for carry-over, but persistence may be damage rather than memory Status	Pesticide, sex, and species specificity	Test repair, reversibility, and pathway dependence before labeling memory

Solitary-bee pesticide exposure	Proposed reconciliation category
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CoA review-selection figure is included here because the evidential interpretation above depends not only on biological distinctions but also on how the literature was identified and routed into review-level synthesis versus contextual validation. The flow therefore reports the search information actually retained and explicitly marks intermediate numerical stages that were not recoverable rather than manufacturing arithmetic.

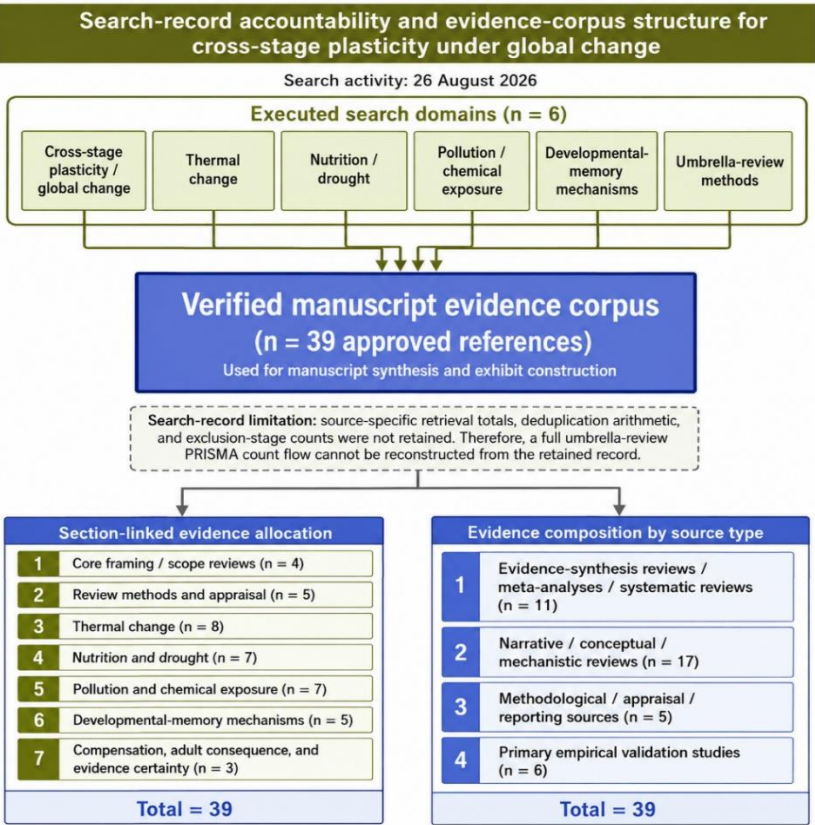


Figure 1. Search-domain routing and recoverability boundaries in the cross-stage plasticity evidence corpus

Compensation and recovery

Recovery after developmental stress should not be equated with restoration of the pre-stress phenotype. Insects can alter metabolite pools, transcriptional activity, tissue organization, and neuroendocrine regulation across multiple timescales, producing metabolic flexibility without necessarily recovering every affected performance trait [37]. We therefore propose four analytically separate post-stress outcomes: restoration, in which an affected trait approaches its earlier state; reallocation, in which resources are redistributed among functions; replacement, in which later-stage processes reduce dependence on an earlier impaired function; and partial buffering, in which a later response offsets only part of an earlier cost. None of these categories implies adaptive evolution or improved fitness unless the relevant adult endpoint is measured directly.

Adult fitness consequences

Adult fitness consequences require evidence beyond molecular persistence or altered juvenile performance. Heat-tolerance research in social insects illustrates this boundary: molecular correlates and candidate pathways are frequently associated with survival-related phenotypes, but functional validation and integration across caste, individual, and colony levels remain incomplete [38]. A retained molecular signature should therefore be interpreted as mechanistic evidence only when experimental manipulation links it to the later organismal phenotype.

Direct cross-stage studies illustrate stronger inference. Elevated developmental temperature in *Enchenopa binotata* produced opposing juvenile-survival and adult-reproductive effects [14]. Reproductive sensitivity to heat can also

appear below lethal limits, showing why adult reproduction cannot be inferred from survival alone [17]. Larval deltamethrin exposure in *Osmia cornifrons* persisted into impaired adult male reproductive traits [30], while earlier imidacloprid exposure in *Osmia lignaria* was associated with reduced later reproduction and lower modeled population growth [31]. These examples support a hierarchy from retained state to adult performance to demographic consequence, but each remains bounded by species, sex, exposure regime, and study setting. The distinctions are compressed in **Table 3** because identical developmental responses can imply different adult outcomes depending on what persists, which adult trait is measured, and whether demographic translation is demonstrated.

Table 3. Stage-linked evidence needed to distinguish adult impairment, compensation, and demographic consequence

Cross-stage state and setting	Observed adult evidence	Fitness meaning and evidential state	Boundary and stronger test
Cross-stage outcome state Retained molecular state Developmental-to-adult context Molecular signal persists after developmental exposure	Expression, metabolite, epigenetic, or pathway change	Adult-fitness interpretation Mechanistic candidate only Status Evidence boundary	Boundary condition Functional effect may be absent Stronger test required Manipulate pathway and measure adult performance
Cross-stage outcome state Persistent physiological damage Developmental-to-adult context Developmental stress followed by impaired adult function	Reduced reproductive or physiological performance	Adult-fitness interpretation Direct adult cost Status Evidence-supported state	Boundary condition Species, sex, exposure and assay specific Stronger test required Replication across environments and taxa
Cross-stage outcome state Reproductive compensation Developmental-to-adult context Earlier survival/developmental cost accompanied by improved later reproductive component	Opposing stage-specific vital-rate changes	Adult-fitness interpretation Partial buffering, not necessarily net benefit Status Proposed synthesis category	Boundary condition Other reproductive or survival costs may remain Stronger test required Full life-cycle demographic integration
Cross-stage outcome state Challenge-dependent reversal Developmental-to-adult context Developmental history changes adult performance differently under alternative challenges	Sign reversal among adult environments or assays	Adult-fitness interpretation Fitness sign is conditional Status Evidence-supported pattern	Boundary condition Laboratory challenge may poorly represent field exposure Stronger test required Factorial developmental × adult environment design
Cross-stage outcome state Delayed reproductive impairment Developmental-to-adult context Early chemical exposure followed by later reproductive deficit	Adult sperm, fecundity, mating or offspring-related endpoint	Adult-fitness interpretation Strong cross-stage fitness cost Status Evidence-supported state	Boundary condition Chemical- and taxon-specific Stronger test required Field-realistic exposure and multisex replication
Population-level translation			
Cross-stage outcome state Demographic translation Developmental-to-adult context Adult carry-over incorporated into population projection	Multiple vital rates or population-growth model	Adult-fitness interpretation Strongest population-level inference in this evidence chain Status Evidence-supported but bounded	Boundary condition Projection remains system-specific Stronger test required Independent field demographic validation

The adult consequences can be recovered more efficiently as a cross-scale architecture than as a linear list because developmental history, retained state, recovery, reproduction, and demographic inference are not equivalent levels.

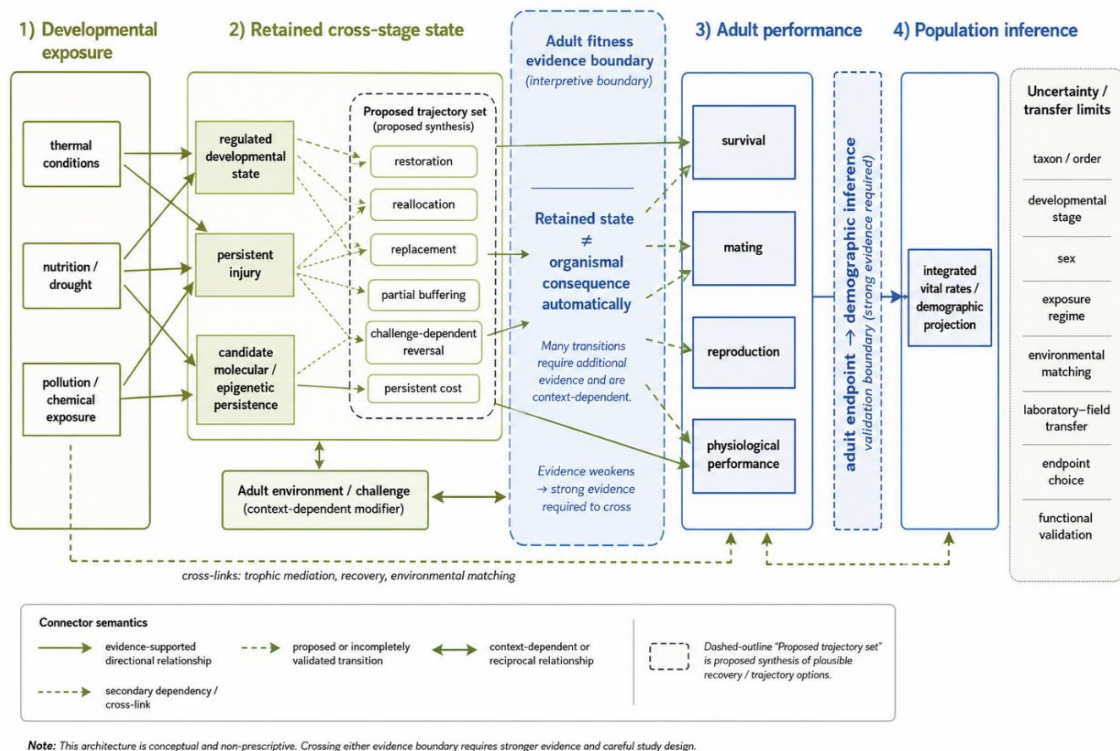


Figure 2. Where cross-stage persistence becomes adult fitness evidence—and where inference must stop

Concordance and contradiction across reviews

Apparent agreement across reviews is meaningful only when driver, life stage, endpoint, trophic pathway, and environmental comparison are sufficiently aligned. The evidence instead shows that broad global-change direction, thermal-regime effects, and trophically mediated herbivore responses can differ substantially with context [1, 12, 21]. A null nutritional carry-over result [24] is therefore not automatically incompatible with evidence of persistent thermal or chemical effects; the studies may ask biologically different questions.

We propose four sources of apparent contradiction: biological effect modification, when taxa, stages, sexes, or environments genuinely alter response; endpoint discordance, when survival, development, reproduction, and molecular measures respond differently; scale discordance, when laboratory, organismal, trophic, or demographic levels are compared as though equivalent; and evidence dependence, when reviews reuse overlapping primary studies. Overlap must be recognized explicitly [6], and graphical or tabular mapping can reveal non-independent evidence structures [7].

Table 4 converts these distinctions into testable reconciliation rules rather than an unsupported consensus score.

Table 4. Prediction-and-test ledger for resolving apparent disagreement among cross-stage plasticity reviews

Concordant direction with aligned endpoint		
Biological or analytical context Same driver, stage and comparable trait Observable basis Similar response direction	Interpretation Strongest form of review-level concordance Status Evidence rule	Principal uncertainty Shared primary studies may inflate agreement Discriminating test Check overlap and independent replication
Different directions across life stages		
Biological or analytical context Same driver, different developmental stages Observable basis Juvenile and adult endpoints diverge	Interpretation Stage-specific biology rather than contradiction Status Evidence rule	Principal uncertainty Endpoint comparability Discriminating test Stage-matched factorial design
Different directions across trophic pathways		
Biological or analytical context Host-mediated versus enemy-mediated exposure	Interpretation Trophic pathway modifies apparent driver effect	Principal uncertainty Co-exposure of trophic levels

Observable basis Bottom-up and top-down effects oppose	Status Evidence rule	Discriminating test Manipulate pathways independently
Molecular response without organismal change		
Biological or analytical context Molecular and performance endpoints disagree	Interpretation Level-of-analysis discordance	Principal uncertainty Timing and assay sensitivity
Observable basis Signal changes while growth/survival remains stable	Status Proposed reconciliation rule	Discriminating test Joint temporal molecular–performance measurement
Carry-over present only under particular adult challenge		
Biological or analytical context Developmental history × adult environment interaction	Interpretation Contextual expression of retained state	Principal uncertainty Environmental realism
Observable basis Sign or magnitude changes across adult environments	Status Proposed reconciliation rule	Discriminating test Crossed developmental/adult environments
Review agreement driven by shared primary studies		
Biological or analytical context Multiple reviews reuse the same experiments	Interpretation Non-independent corroboration	Principal uncertainty Incomplete overlap reporting
Observable basis High evidence overlap	Status Methodological rule	Discriminating test Primary-study citation matrix

Evidence certainty

Certainty was interpreted as multidimensional rather than numerical. Ecological appraisal should match the evidence design and cannot be reduced safely to a generic checklist [8]. Review-level domain appraisal remains informative [9], but confidence in a cross-stage claim also depends on direct exposure-to-adult linkage, consistency across endpoints, independence among reviews, field transfer, taxonomic breadth, and functional validation.

Generalizability is additionally constrained by evidence distribution. Research on insect plasticity and its genetic architecture remains concentrated in a limited subset of orders and traits, and plasticity itself must remain distinct from evolutionary adaptation [39]. High certainty for a specific bee pesticide carry-over effect, for example, does not constitute high certainty that the same trajectory occurs across Diptera, Lepidoptera, Coleoptera, or other Hymenoptera.

The central synthesis is that cross-stage plasticity is better interpreted as a conditional trajectory than as a persistent trait with a fixed fitness sign. Evidence spanning life-cycle thermal plasticity, experimentally demonstrated thermal compensation, and pesticide-induced adult reproductive damage shows that persistence, conditionality, and partial buffering are biologically distinct possibilities [3, 14, 30]. The proposed trajectory architecture therefore asks three sequential questions: what remains after the developmental transition, how that retained state is expressed in the adult environment, and whether the resulting adult phenotype changes a fitness-relevant endpoint.

This formulation also prevents compensation from being mistaken for adaptation. A later response can offset an earlier cost without restoring all traits, increasing total lifetime fitness, or reflecting evolutionary optimization. Likewise, apparent developmental memory can arise from regulated state retention, residual injury, endocrine or metabolic reconfiguration, or challenge-dependent expression. Distinguishing these possibilities requires experimental manipulation rather than terminology alone.

Population inference demands an additional boundary. Life-history integration, delayed pesticide effects on reproduction and modeled population growth, and the limited functional validation of many molecular heat-response correlates collectively show why population consequences require validated links across biological scales [2, 31, 38]. Adult reproduction is stronger evidence than a molecular marker, but even reproduction does not alone establish long-term demographic decline when survival, mating, density dependence, environmental variation, or compensatory vital rates remain unmeasured.

The largest evidence gaps are therefore structural rather than merely numerical. Future studies should cross developmental and adult environments factorially, retain sex-specific endpoints, quantify both damage and recovery, distinguish direct from host- or trophically mediated exposure, and connect molecular candidates to experimentally manipulated organismal outcomes. Review-level synthesis should also document primary-study overlap so that repeated evidence is not mistaken for independent replication.

Conclusion

Cross-stage plasticity under global change cannot be reduced to a single continuum from exposure to adaptation. Developmental effects may disappear, persist as damage, remain as regulated biological states, reverse under a new adult environment, or be partially offset by later responses. Adult fitness consequences become defensible only when later survival, mating, reproduction, or integrated vital rates are measured rather than inferred from developmental or molecular responses. The proposed conditional-trajectory framework therefore separates developmental memory, recovery, compensation, adult performance, and demographic inference while retaining taxonomic, life-stage, sex, environmental, methodological, and laboratory-to-field boundaries. Its principal value is interpretive: it provides a disciplined way to distinguish genuinely concordant cross-stage evidence from superficially similar responses that occupy different biological levels.

Acknowledgments: None

Conflict of Interest: None

Financial Support: None

Ethics Statement: None

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