Eurasia Specialized Veterinary Publication

Entomological Research Letters

ISSN: 3108-639X

2024, Volume 4, Issue 2, Page No: 104-118
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Available online at: www.esvpub.com/

Developmental Carry-Over, Compensation, and Hidden Trade-Offs Across Life Stages in Insect Life Histories

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ABSTRACT

Developmental environments can influence insect phenotypes long after the exposure itself has ended, yet persistence across metamorphosis is neither inevitable nor uniform among traits. This systematic review examines how juvenile nutritional, thermal, microbial, toxicological, competitive, and other environmental conditions translate into persistent effects, compensation, reversal, or delayed costs at later life stages. The review distinguished immediate developmental responses from cross-stage outcomes and classified later phenotypes according to whether an earlier effect persisted, was buffered, disappeared, changed direction, or re-emerged in a different fitness-related trait. The systematic search yielded 32 eligible primary studies after deduplication and staged screening. Across the evidence base, adult body size, physiology, immunity, reproductive performance, longevity, and behavior frequently responded differently to the same developmental exposure. Apparent normalization of development time or size therefore did not reliably indicate whole-organism recovery. Conversely, persistent juvenile effects were sometimes restricted to particular sexes, phenologies, adult environments, or outcome classes. We propose a trajectory-based interpretation in which carry-over, compensation, reversal, and hidden cost are treated as analytically separate cross-stage states. This approach avoids equating metamorphic persistence with mechanism, phenotypic compensation with fitness recovery, or adult trait change with adaptive benefit.

Keywords: Developmental carry-over, Metamorphosis, Compensatory responses, Life-history trade-offs, Developmental plasticity, Adult fitness

Received: 06 October 2024
Revised: 24 November 2024
Accepted: 25 November 2024

How to Cite This Article: Novak P, Svoboda J. Developmental Carry-Over, Compensation, and Hidden Trade-Offs Across Life Stages in Insect Life Histories. Entomol Res Lett. 2024;4(2):104-18.
https://doi.org/10.51847/ejlCN3W5TO

Introduction

Insect development occurs as a sequence of biologically distinct environments rather than as a single continuous phenotype. Embryonic, larval or nymphal, pupal, and adult stages differ in feeding opportunity, mobility, tissue organization, energetic demand, and exposure to environmental stress. Developmental processes also differ in robustness: environmentally sensitive growth can coexist with tightly buffered patterning, and consequences of a perturbation therefore depend on both the trait affected and the developmental interval in which exposure occurs [1]. A later phenotype cannot consequently be inferred from the magnitude of an earlier response alone.

This problem is particularly important in holometabolous insects because metamorphosis extensively reorganizes tissues while retaining resources, cell lineages, endocrine histories, microbial effects, and developmental constraints capable of influencing the adult phenotype. Experimental manipulation of larval bacterial environments in *Aedes aegypti*, for example, altered pupation, adult size, antibacterial activity, and dengue-virus-related phenotypes while leaving adult lifespan unchanged [2]. In a seminatural longitudinal experiment with

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Coenagrion puella, larval pesticide exposure reduced female lifetime mating success, but the magnitude of the carry-over effect depended strongly on egg-hatching phenology [3]. These findings illustrate two problems with a binary "carry-over present versus absent" interpretation: different adult traits can disagree, and the same developmental exposure can produce different outcomes under different temporal contexts.

Mechanistic continuity across stages is likewise variable. Larval microbial interactions in *A. aegypti* have been linked to persistent changes in metabolic state, lipid storage, starvation resistance, transcriptomic profiles, and adult survival [4]. Yet larval dietary restriction in the butterfly *Speyeria mormonia* reduced adult size and flight metabolic capacity without shortening adult lifespan, while fecundity declined less than expected from the reduction in body mass [5]. Such responses are better interpreted as allocation changes among adult functions than as uniform deterioration or recovery.

Proxy traits add another inferential difficulty. In *Aedes albopictus*, larval diet and temperature altered development, pupal mass, wing length, immune-gene expression, and adult longevity, but the two body-size measures showed opposing relationships with lifespan [6]. In *Drosophila melanogaster*, larval macronutrient composition affected adult reproduction without detectably changing survival [7]. Thus, emergence size, development time, or pupal mass may be biologically informative without functioning as interchangeable proxies for adult performance.

The strongest conceptual challenge arises when developmental conditions alter relationships among traits rather than simply shifting their means. Developmental diet in *D. melanogaster* changed the fecundity–longevity relationship from positive through neutral to negative patterns even though mean lifetime fertility and longevity changed relatively little [8]. Larval exposure to *Bacillus thuringiensis israelensis* in tolerant *A. albopictus* produced reduced adult emergence, shorter adult lifespan, lower fecundity, altered gut microbiota, and persistent immune responses [9]. Extreme larval heat exposure in *Culex pipiens pallens* likewise affected later reproductive performance, with sex- and temperature-dependent sensitivity [10]. These studies indicate that later-life consequences may emerge as altered covariance, reproductive allocation, physiological performance, or survival rather than as simple persistence of the original juvenile phenotype.

This review therefore addresses a narrower question than whether developmental environments "matter" in adulthood. It asks how an earlier-stage effect changes state as an insect moves through subsequent developmental boundaries. We distinguish persistent carry-over, compensatory response, reversal, and hidden adult cost, while separating directly observed trajectories from mechanistic interpretation. The proposed trajectory classification is an interpretive synthesis of the reviewed evidence rather than an empirically validated universal model.

Materials and Methods

Search strategy, eligibility, cross-stage outcome coding, and quality assessment

The review followed systematic-review reporting principles consistent with PRISMA 2020 [11]. Searches covered literature published from 2017 through 2024 and combined insect terms with developmental-stage terms and cross-stage outcome concepts. Core search strings combined variants of *insect*, *larva*, *nymph*, *pupa*, *development*, *metamorphosis*, *carry-over*, *carryover*, *developmental stress*, *compensation*, *recovery*, *reversal*, *adult*, *fecundity*, *reproduction*, *longevity*, *survival*, *metabolism*, and *fitness*. Exposure-specific searches added diet or nutrition, temperature or heat, density or competition, pesticide or pollutant, microbiota or bacteria, pathogen or immune challenge, and predator-related terms. The search window covered records indexed through 31 December 2024.

Database searches identified 318 records from Scopus, 286 from Web of Science, 174 from PubMed, 141 from Zoological Record, and 29 records identified by citation chaining or supplementary searches, giving 948 records before deduplication. Removal of 312 duplicates left 636 unique records for title-and-abstract screening. Of these, 501 were excluded and 135 reports were sought for full-text assessment. Four reports were not retrieved, leaving 131 full texts assessed for eligibility.

A primary study was eligible when it examined an insect, imposed or quantified an exposure or developmental condition in an earlier life stage, and measured at least one biologically distinct outcome after a subsequent developmental transition. Studies restricted entirely to one stage were excluded from the primary synthesis. Transgenerational studies were retained only when they also contained an interpretable within-individual cross-stage component; purely parental or offspring effects were not treated as developmental carry-over within the focal individual. Reviews and methodological papers were used for conceptual or reporting support but were not counted as primary evidence units.

Ninety-nine full texts were excluded: 35 lacked a later-stage biological endpoint, 16 did not concern insects, 14 did not permit the developmental exposure to be separated from later exposure, 12 were not eligible primary research, nine failed the predefined journal-eligibility criterion, eight fell outside the publication window, and five represented overlapping or companion reports without an independent evidence unit. Thirty-two primary studies entered the qualitative synthesis.

Each study was coded at the level of the exposure–transition–outcome trajectory, rather than receiving one article-wide carry-over label. Persistent carry-over required a measurable later-stage difference associated with the earlier developmental condition. Compensation denoted partial or complete attenuation in a focal phenotype after the developmental transition, without assuming restoration of other traits. Reversal required a change in direction, ranking, or relationship at the later stage. Hidden adult cost was assigned when an earlier-stage phenotype appeared weak, normalized, or inadequately informative but a later physiological, reproductive, behavioral, or survival cost remained detectable. Null later-stage findings were retained explicitly.

Quality assessment emphasized six domains: temporal separation of exposure and outcome; adequacy of the comparator; independence of later-stage exposure; validity of the adult endpoint; handling of sex, developmental timing, and environmental moderators; and the extent to which mechanistic claims exceeded the measurements obtained. Evidence from controlled experiments was not automatically treated as field-generalizable, and associations among developmental traits, adult traits, and fitness proxies were not recoded as causal mechanisms.

Results and Discussion

The evidence landscape was taxonomically and methodologically heterogeneous. Dipteran systems, especially *Drosophila* and mosquitoes, supplied many of the mechanistically detailed nutritional, microbial, immunological, and longevity studies. Odonates contributed unusually valuable longitudinal evidence spanning aquatic larval environments and terrestrial adult performance, including experiments where adult fitness could be observed under seminatural conditions. Lepidopteran studies provided direct tests of energetic allocation following larval food limitation, while other holometabolous taxa broadened the evidence for nutritional, thermal, and reproductive carry-over.

Temperature was rarely interpretable independently of developmental rate, density, nutrition, or the adult environment. In *A. albopictus*, larval competition interacted with temperature, and differences between larval and adult thermal environments altered fecundity and survival [12]. This indicates that an early-life effect can remain conditional on the environment encountered after emergence rather than becoming a fixed adult phenotype.

Nutritional evidence showed similarly incomplete correspondence among traits. Larval diet could alter adult reproduction while leaving survival unchanged [7], and food restriction could depress adult size and flight capacity without shortening lifespan [5]. Earlier insecticide exposure also produced delayed reproductive consequences that could not be reduced to immediate developmental mortality alone [13]. At a molecular level, larval stress in the Antarctic midge *Belgica antarctica* altered adult reproductive biology and reproductive-tissue products associated with progeny performance [14].

More recent dietary experiments strengthened the distinction between developmental and adult control. Developmental macronutrient composition in *D. melanogaster* influenced age-specific reproductive output through effects associated with ovariole formation and vitellogenesis, whereas adult diet exerted a different temporal pattern of influence [15]. In the black soldier fly *Hermetia illucens*, larval substrate altered adult development time, body mass, lifespan, and egg production, demonstrating that larval nutritional quality can remain visible across multiple adult life-history dimensions [16].

The evidence therefore did not support a universal ordering in which juvenile growth effects precede equivalent adult fitness effects. Instead, studies repeatedly separated into discordant trait trajectories: an exposure could persist in morphology but not longevity, persist in reproductive output but not survival, alter immune or metabolic state without equivalent demographic effects, or become detectable only through interactions with later conditions. This heterogeneity motivated the trajectory-specific synthesis below rather than an article-level vote counting of “positive” and “negative” carry-over.

Persistent carry-over

Persistent carry-over was clearest when the later-stage measurement remained biologically linked to an experimentally isolated juvenile exposure. Larval bacterial environments in *A. aegypti* altered adult phenotypes

even though the experimental exposure occurred before metamorphosis [2]. Subsequent work connected larval bacterial interaction to adult metabolic and transcriptional states, strengthening evidence that persistence can involve physiological organization rather than merely adult size [4]. Larval bacterial exposure has also altered adult arbovirus susceptibility and immune-gene expression in the absence of simple persistence of the experimentally supplied bacteria themselves [17]. These results support transstadial biological influence but do not justify treating every persistent molecular signature as the mechanism responsible for an adult performance outcome.

Persistent toxicological effects provide a second pattern. In *C. puella*, the adult reproductive consequence of larval esfenvalerate exposure depended on hatching phenology [3]. This is important because it demonstrates persistence without invariance: the developmental effect survived metamorphosis, but its adult expression was conditional on when development began. Multiple-stressor work in aquatic insects similarly indicates that nutritional stress can amplify pollutant effects and that the combined survival-related cost may become stronger during or after metamorphosis than during the directly exposed larval stage [18]. Persistence can therefore involve amplification across a developmental boundary.

Nutritional persistence was often multidimensional. In *S. mormonia*, larval food restriction produced smaller adults and reduced flight metabolic capacity, while adult lifespan remained unchanged and reproductive allocation was partly buffered [5]. In *A. albopictus*, diet and temperature jointly modified developmental traits, body-size measures, immune expression, and longevity, but no single juvenile metric consistently represented the adult outcome [6]. Persistent carry-over should consequently be attributed to a defined trait trajectory, not to the whole organism.

Thermal exposure also showed stage specificity. Repeated high-temperature exposure in the predatory ladybird *Propylaea japonica* generated immediate developmental effects and negative consequences in subsequent stages, with sensitivity differing among eggs, larvae, pupae, and adults [19]. Extreme larval heat in *C. pipiens pallens* affected later reproductive performance while responses differed by sex and thermal intensity [10]. Such studies caution against describing “thermal carry-over” without specifying exposure stage, intensity, later endpoint, and adult environment.

Because persistent effects arise through several distinct biological routes, a simple study-by-study list obscures the crucial distinction between what actually survives metamorphosis and what is merely correlated with an earlier exposure. The adjudication matrix below therefore separates the observable persistence pattern from the inference that can safely be made.

Table 1. When an Earlier-Life Effect Survives Metamorphosis: Adjudicating Persistent Carry-Over Without Treating Persistence as a Single Biological State

Carry-over route	Developmental context and retained signal	Safe interpretation and caveat	Synthesis consequence
Persistent biological carriers			
Microbial-environment persistence	Larval mosquito–bacteria interaction Adult immune, metabolic, transcriptomic, or vector-related phenotype	Earlier microbial environment can alter adult state Persistent phenotype does not by itself identify the causal molecular route	Treat persistence and mechanism as separate evidence classes
Phenology-conditioned toxicological persistence	Aquatic larval pesticide exposure followed by terrestrial adulthood Adult mating or reproductive performance	Larval exposure can retain later fitness relevance Effect magnitude depends on developmental timing	Persistent does not mean context-invariant
Nutritional energetic persistence	Larval food restriction Adult size, metabolic performance, resource allocation	Developmental nutrition can constrain selected adult functions Other adult traits may remain unchanged or buffered	Code individual trait trajectories rather than whole-organism impairment
Discordant or amplified readouts			
Proxy–outcome discordance	Larval diet and temperature Pupal mass, wing length, immune expression, longevity	Juvenile and emergence traits can carry information into adulthood Alternative size proxies may predict adult outcomes differently	Do not use morphological recovery as automatic evidence

			of functional recovery
Cross-stage stress amplification	Larval pollutant combined with resource stress Survival-related performance after metamorphosis	A combined juvenile stress effect can intensify after exposure has ended Interaction may be system- and dose-dependent	Persistence can strengthen rather than simply decay
Stage- and sex-bounded persistence			
Stage- and sex-bounded thermal persistence	Early thermal stress Later development, survival, reproduction, or physiology	Earlier heat can influence later performance Sensitivity differs among developmental stages, sexes, and heat intensities	Thermal carry-over requires explicit boundary specification

Compensatory responses

Compensation was not equivalent to erasure of developmental history. Instead, the reviewed studies showed that insects can attenuate an earlier deficit in one phenotype while retaining costs in another. The clearest example came from larval dietary restriction in *S. mormonia*. Restricted larvae became smaller adults with lower flight metabolic capacity, but adult lifespan was unchanged and fecundity declined less than expected from reduced body mass [5]. The pattern is therefore inconsistent with both full recovery and uniform impairment. It is better described as trait-selective compensation, in which reproductive allocation is buffered while locomotor or energetic capacity remains constrained.

A similar inferential problem emerged from mosquito morphometrics. In *A. albopictus*, pupal mass and wing length responded differently to combinations of larval temperature and nutrition and showed opposite associations with adult longevity [6]. If compensation were judged from only one size metric, the biological conclusion could change with the proxy selected. This review therefore treats compensation as an outcome-specific state requiring explicit identification of the trait that recovered and the traits that did not.

Dietary effects on *D. melanogaster* reinforce this distinction. Larval macronutrient composition changed adult reproduction but not survival in one experiment [7], whereas developmental diet in another altered the age-dependent architecture of fecundity through developmental effects on reproductive anatomy and physiology [15]. These findings do not support a universal compensatory rule. They indicate that different adult systems retain different degrees of dependence on developmental nutrition.

The adult environment can further mask or expose developmental history. When adult conditions supply resources unavailable during juvenile development, later traits may converge even though developmental costs were previously expressed. Conversely, an adult challenge can reveal latent differences between individuals that appeared similar at emergence. We therefore propose that compensation should be diagnosed only when a defined earlier deficit attenuates at a defined later endpoint. It should not be inferred from survival to adulthood, restoration of body mass alone, or the absence of a statistically detected difference in one adult trait.

Because the review-selection arithmetic determines which studies underpin these distinctions, the search-to-inclusion sequence is shown explicitly before moving from compensation to reversal.

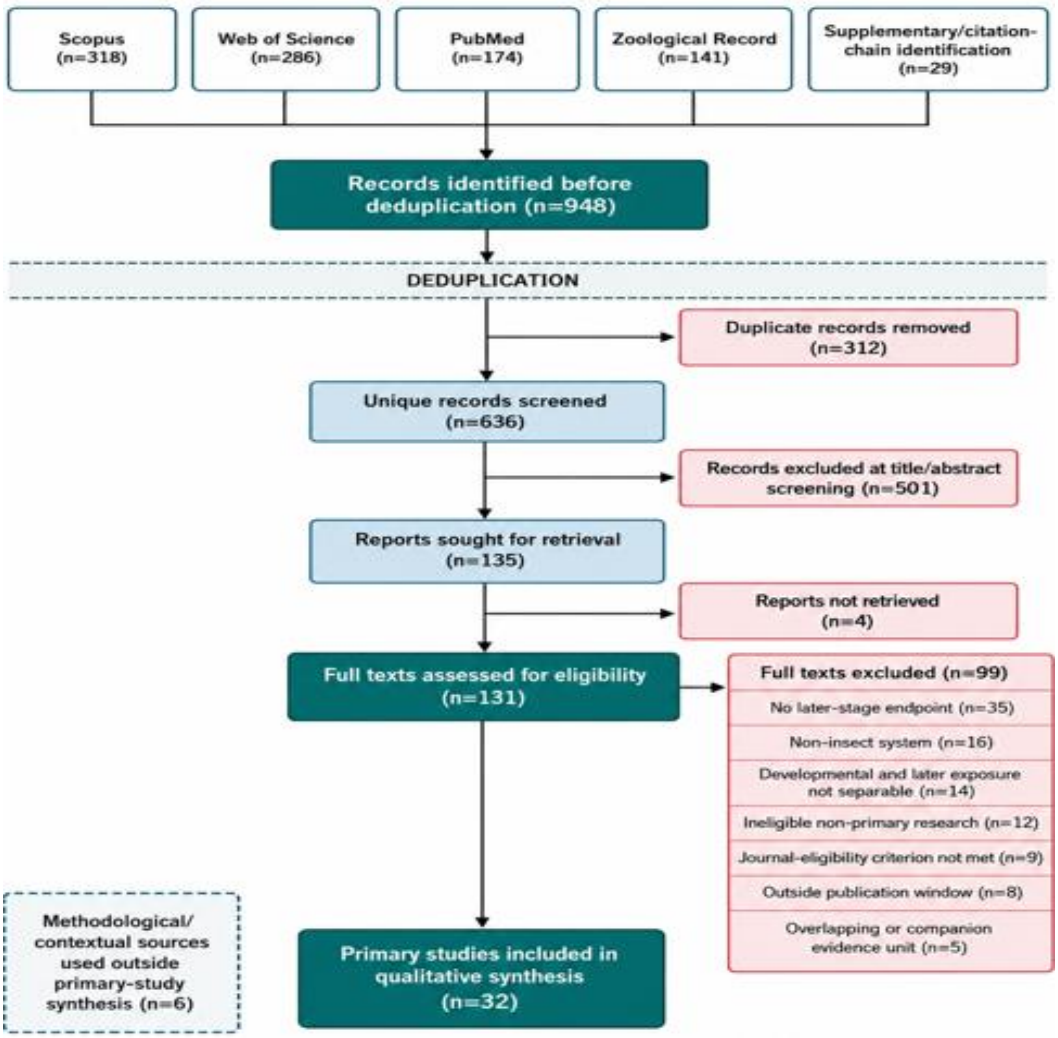


Figure 1. Record Attrition Across the Developmental Carry-Over Review and the Boundary Between Retrieved Literature and Eligible Cross-Stage Evidence

Reversal of effects

Reversal is analytically distinct from compensation because the later phenotype does not simply move toward the control state; the direction or structure of the relationship changes. The strongest example concerns developmental diet and the adult fecundity–longevity relationship in *D. melanogaster*. Different larval diets produced adult relationships ranging from positive to negative even though mean lifetime fertility and longevity changed comparatively little [8]. The developmental exposure therefore reorganized the association between two adult life-history traits rather than producing a simple monotonic deficit.

Reversal can also occur through context-dependent ranking. In *A. albopictus*, the magnitude and direction of adult survival and fecundity differences depended on both larval conditions and the temperature experienced by adults [12]. A developmental phenotype that appears advantageous under one adult environment can consequently lose that advantage, disappear, or become detrimental under another. Such interaction-dependent outcomes should not be described as developmental adaptation without evidence that the resulting phenotype increases fitness in the relevant environment.

A second form of apparent reversal arises when traits move in different directions. Larval restriction in *S. mormonia* reduced adult body size and flight metabolic capacity while reproductive allocation was comparatively buffered and lifespan remained unchanged [5]. This is not a sign reversal within one trait, but it can generate a reversal in the organism-level interpretation: a developmental treatment that looks strongly detrimental from locomotor physiology may appear much less consequential from lifespan or relative reproductive allocation. Likewise, opposite relationships between pupal mass and wing length with adult longevity in *A. albopictus* demonstrate that the direction of inference can depend on the developmental proxy chosen [6].

The review therefore separates true directional reversal, rank-order reversal across environments, and interpretive reversal caused by trait discordance. Only the first requires the same focal relationship to change sign. The other forms remain biologically important because they explain why conclusions based on one stage, one proxy, or one environment may fail when adult performance is measured directly.

Table 2. Reversal Is Not Recovery: Cross-Stage State Changes That Alter the Meaning of an Earlier Developmental Effect

Directional and rank reversals		
Reversal comparison dimension	**Directional relationship reversal**	**Environment-dependent rank reversal**
Context and observable configuration	Developmental diet and adult life-history covariance Adult trait relationship changes sign among developmental treatments	Different larval and adult environments Treatment ranking changes with adult conditions
Correct classification	Development reorganizes later trait covariance	Developmental history remains conditional on later environment
Boundary condition	Mean trait values need not reverse	Requires explicit stage × environment comparison
Research implication and state	Measure covariance and individual trajectories, not means alone Evidence-supported category	Use factorial juvenile–adult designs Evidence-supported category
Trait or proxy-based apparent reversal		
Reversal comparison dimension	**Trait-discordant interpretive reversal**	**Proxy-based reversal**
Context and observable configuration	Morphology, metabolism, reproduction, and survival respond differently One trait indicates impairment while another is buffered or unchanged	Alternative emergence or size measures predict adult performance differently Contrasting associations between developmental proxies and adult endpoint
Correct classification	Whole-organism labels become misleading	Inference depends on proxy selection
Boundary condition	Traits are not interchangeable fitness measures	Proxy validity may be species- and environment-specific
Research implication and state	Report multiple adult endpoints Proposed synthesis	Validate proxies against adult outcomes Proposed synthesis
Latent cost and dissipation		
Reversal comparison dimension	**Apparent recovery with unresolved latent cost**	**Dissipation rather than reversal**
Context and observable configuration	Juvenile deficit attenuates at emergence Focal morphology or development time converges	Earlier effect no longer detectable at later endpoint Later-stage treatment and control converge
Correct classification	Compensation cannot yet be equated with recovery	Effect may have disappeared for that measured trait
Boundary condition	Later physiology or reproduction may remain unmeasured	Statistical null does not establish restoration of all functions
Research implication and state	Extend follow-up beyond emergence Proposed synthesis	Preserve explicit null outcomes and endpoint specificity Proposed synthesis

Note: "Proposed synthesis" identifies distinctions introduced by this review to organize heterogeneous cross-stage evidence. No numerical score or hierarchy is implied.

Hidden adult costs

The most consequential cross-stage effects are not necessarily those that remain obvious at emergence. A juvenile exposure may produce little difference in development time, body size, or immediate adult survival yet alter reproductive ageing, physiological reserves, stress tolerance, or performance under a later challenge. Developmental and adult thermal environments illustrate this distinction. In *Callosobruchus maculatus*, developmental thermal stress reduced female adult reproductive performance, whereas relationships with ageing

depended on sex, trait, and the adult environment [20]. The important signal was therefore not a single persistent developmental deficit but a later redistribution of performance across adult life.

Predation history provides a different route to latent cost. Larval exposure to predators in *Anopheles coluzzii* and *Anopheles gambiae* produced species-dependent changes in adult life-history traits, nutritional reserves, and oxidative state [21]. Because the two species did not respond identically, predator-induced carry-over cannot safely be generalized even between related mosquitoes. A phenotype that successfully survives larval predation risk may thus reach adulthood carrying altered energetic reserves whose consequences are not represented by larval survival itself.

Nutritional restriction can similarly remain concealed until adult demands are imposed. Experiments manipulating larval and adult nutrition in *Aedes aegypti* found that nutritional stress reduced egg production and altered antiviral immunity and dengue-virus susceptibility, while adult nutritional stress exerted additional effects on survival [22]. This interaction is informative because an early nutritional deficit may change the adult's response capacity without determining the adult outcome independently of later conditions. Hidden cost is therefore best understood as conditional debt, not as an irreversible deficit carried unchanged through metamorphosis.

Not all hypothesized carry-over mechanisms withstand direct testing. Adult *A. aegypti* lacking a gut microbiota did not maintain normal reproductive and metabolic function through presumed larval nutrient carry-over alone; B-vitamin provision or recolonization with suitable bacteria restored important functions [23]. This result establishes an important negative boundary: the persistence of developmental resources should not be invoked when continuing adult microbial provisioning better explains the phenotype.

Developmental and adult diets can also contribute unequally to adult fitness components. In *D. melanogaster*, sex-specific analyses showed that larval and adult diets interacted differently with lifespan and reproductive success [24]. Similarly, antibiotic exposure during larval development in *Culex pipiens* altered subsequent adult survival, but the direction depended on sex, adult antibiotic exposure, and the duration of larval feeding before emergence [25]. These studies caution against labeling a treatment as beneficial or costly without specifying the adult context in which the effect is expressed.

The latent-cost concept used here is therefore narrower than “any delayed effect.” It requires a mismatch between what an earlier endpoint would lead an investigator to conclude and what a later biologically consequential endpoint reveals. **Table 3** distinguishes the principal ways such mismatches arise.

Table 3. Where Apparent Developmental Recovery Conceals Adult Debt: Mechanisms, Detection Boundaries, and Tests of Hidden Cost

Hidden-cost configuration	Developmental condition	Adult endpoint revealing debt	Reason the earlier endpoint misleads	Alternative explanation	Resolving test
Reproductive and metabolic debt					
Reproductive debt after successful emergence	Thermal, nutritional, toxicological, or microbial challenge	Fecundity, mating success, fertility, reproductive ageing	Emergence and adult morphology can normalize before reproductive demand peaks	Adult diet, mating environment, sex, and age can modify expression	Follow cohorts through age-specific reproduction
Metabolic debt after developmental compensation	Resource limitation or predation stress	Energy stores, metabolic performance, oxidative state, starvation response	Extended development can restore emergence while altering reserve acquisition	Longer development itself may change adult phenotype	Measure both developmental duration and adult physiological reserves
Challenge- and microbiome-revealed debt					
Challenge-revealed debt	Juvenile nutritional, microbial, or chemical exposure	Adult infection response, thermal tolerance, toxicant sensitivity	Adults may appear equivalent under benign conditions	Later challenge may generate rather than merely expose differences	Cross juvenile treatment with standardized adult challenge
Microbiome-dependent latent effect	Larval microbial environment or antibiotic exposure	Adult survival, immunity, metabolism, vector competence	Microbial composition can be reorganized across metamorphosis	Adult reacquisition or dietary provisioning can dominate	Track microbial function and composition across the transition

Proxy and sex boundaries					
Proxy–fitness mismatch	Developmental size, mass, or development-time response	Reproduction or longevity	Morphological convergence is not functional equivalence	Proxy validity differs across taxa and environments	Validate each proxy against the adult endpoint of interest
Sex-bounded hidden cost	Any stage-specific stressor with sex-dependent allocation	Male mating/reproductive traits versus female fecundity or survival	Sexes may be pooled at emergence despite divergent adult demands	Sexual dimorphism and sex-specific resource allocation	Analyze adult outcomes separately by sex

Note: "Hidden cost" and the configurations above constitute the proposed synthesis of this review. They describe inferential states rather than universal mechanisms or quantitative severity classes.

Reproductive and longevity consequences

Reproduction and longevity are especially informative endpoints because they occur after developmental exposures have passed and integrate multiple physiological processes. Yet they should not be collapsed into a single measure of adult fitness. Larval diet, microbial exposure, thermal stress, density, and toxicants can affect egg production, mating performance, survival, and ageing in different directions, making their joint measurement central to identifying hidden trade-offs.

Food quality can influence adult survival as well as pathogen-related phenotypes. In *Anopheles arabiensis*, larval pollen diets altered larval performance, adult size, longevity, and susceptibility to *Plasmodium*, with outcomes depending on pollen source [26]. The implication is not that one larval diet is generally advantageous; improved adult survival may coexist with altered parasite susceptibility, creating a biological trade-off whose ecological meaning depends on the endpoint considered.

Larval density provides another route by which juvenile ecology reaches the adult stage. Crowding in *A. aegypti* altered later dengue-virus loads, demonstrating that larval competition can influence an adult phenotype relevant to pathogen transmission even after the crowded environment has disappeared [27]. Temperature further modifies density-dependent effects. Experiments with *A. aegypti* showed that competition and resource depletion reshape the thermal response of population fitness, making extrapolation from individually reared or resource-replete larvae particularly uncertain [28].

Some of the strongest evidence for developmental regulation of longevity comes from experimental systems in which adult conditions were explicitly controlled. In *D. melanogaster*, developmental yeast restriction altered adult lifespan through later production of lipid-derived autotoxins, and the adult environment modified the magnitude of that effect [29]. This provides direct evidence that a developmental exposure can influence a later physiological process rather than simply preserving the original larval phenotype.

Comparable complexity occurs in mosquitoes. Nutrient-deprived *A. albopictus* larvae could prolong development sufficiently to compensate for resource limitation at one level, yet emerging females retained altered nutrient metabolism and longevity [30]. Developmental compensation in time therefore coexisted with persistent adult metabolic reorganization. Likewise, microbial composition in larval water changed *A. aegypti* developmental performance and later Zika-virus competence even though differences in adult microbial diversity did not mirror the larval-water contrast straightforwardly [31]. Persistence of function can consequently occur without persistence of the presumed biological carrier.

Larval environments also alter responses to adult toxicological challenges. Temperature, density, and nutrition during larval development changed adult insecticide susceptibility in *Anopheles gambiae* and *Anopheles stephensi*, with nutrition contributing strongly to body size and mortality differences [32]. In laboratory *Anopheles funestus*, larval density affected development and adult production, whereas provision of additional anchoring surface modified survival and adult size under crowded conditions [33]. Such methodological sensitivity matters because laboratory rearing conditions can themselves create developmental histories that change the adult phenotype subsequently measured.

Finally, genotype can bound nutritional responses. Extensive genetic variation in macronutrient tolerance has been demonstrated in *D. melanogaster*, including variation in developmental and metabolic responses to dietary composition [34]. Consequently, developmental diet effects obtained from one laboratory genotype cannot be assumed to define the reaction norm of a species.

These findings support a conditional map rather than a linear early-stress-to-adult-fitness pathway. **Figure 2** separates the developmental exposure, what crosses the metamorphic boundary, the adult state in which it becomes expressed, and the reproductive or longevity endpoint used to infer consequence.

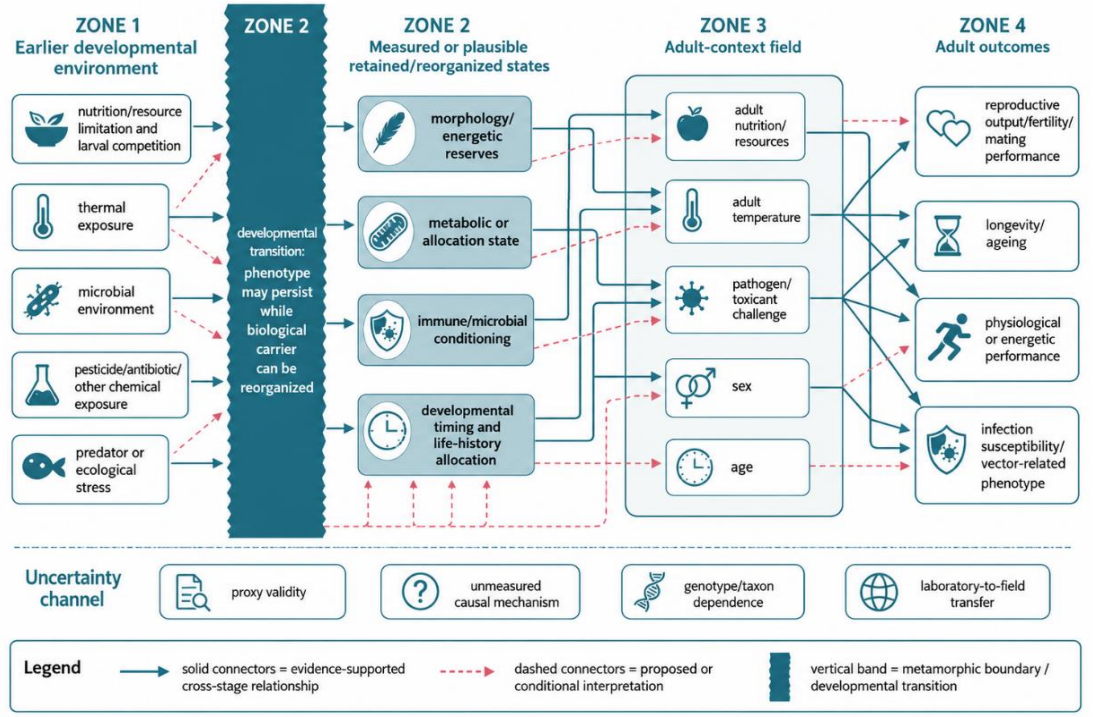


Figure 2. Adult Reproduction and Longevity Emerge from Cross-Stage Retention, Reallocation, and Later-Life Context

The reviewed evidence rejects two symmetrical simplifications. Metamorphosis is neither a complete developmental reset nor a guarantee that juvenile effects persist into adulthood. Instead, environmental history is selectively retained, attenuated, reorganized, or exposed by later conditions. Microbiome research reinforces this interpretation: mosquito-associated microorganisms influence development, reproduction, immunity, and pathogen interactions, but microbial communities are dynamic across environments and developmental transitions [35]. An adult phenotype associated with larval microbial exposure therefore need not be explained by literal transstadial persistence of the same microorganisms.

Thermal evidence imposes a similar boundary. Insect heat responses involve metabolic, neural, hormonal, behavioral, and cellular processes whose importance varies among taxa and stages [36]. Consequently, an adult consequence of larval heat exposure cannot automatically be labeled acclimation or adaptation. Demonstrating adaptation requires evidence about fitness and evolutionary context beyond demonstration of developmental plasticity or stress-response gene expression.

The same caution applies to developmental plasticity more broadly. Comparative work shows that environmentally induced insect phenotypes arise through diverse developmental and neuroendocrine routes, while mechanistic knowledge remains concentrated in relatively few model systems [37]. The taxonomic concentration evident in this review therefore matters: *Drosophila*, *Aedes*, *Anopheles*, and several experimentally tractable insects provide unusually detailed evidence, but their developmental architectures and ecological transitions cannot stand for all insect orders.

The synthesis proposed here consequently treats developmental history as a trajectory problem. Persistent carry-over describes continuity in a defined later trait; compensation describes attenuation in that trait without presuming organism-wide recovery; reversal describes a change in direction, ranking, or trait relationship; and hidden cost describes a later consequence that would be missed by an earlier proxy. This classification complements evolutionary distinctions between direct, latent, and indirect carry-over effects, which emphasize developmental linkage and the evolutionary conditions under which stages may become more strongly coupled

or decoupled [38]. It does not replace those distinctions or claim that the categories correspond to unique molecular mechanisms.

A central implication is that metamorphosis should be treated as an inference boundary. A developmental exposure and an adult phenotype can be connected experimentally without establishing what crossed that boundary. Stored nutrients, persistent cells, endocrine programming, altered development time, tissue scaling, microbial products, immune conditioning, and subsequent resource allocation are biologically different explanations. Where only the exposure and adult outcome are measured, describing a "mechanism" exceeds the evidence.

A second implication concerns fitness proxies. Adult size, wing length, development time, fecundity, mating success, longevity, metabolic capacity, immunity, and pathogen susceptibility answer different questions. Their discordance is not statistical inconvenience but information about allocation across the life history. Recovery in one trait can coexist with a cost in another; conversely, a persistent morphological difference can occur without an observed longevity penalty. The appropriate unit of synthesis is therefore the exposure–stage transition–outcome trajectory.

A third limitation is environmental transfer. Most mechanistically rich studies use laboratory colonies, standardized diets, controlled temperatures, or experimentally manipulated microbes. Seminatural longitudinal studies demonstrate that developmental timing and ecological context can substantially alter later consequences [3]. Extrapolation to field populations requires adult resource availability, predation, mating opportunity, climate variability, microbial acquisition, and density to be treated as potential moderators rather than as background noise.

The cross-scale problem created by these observations is summarized below. The grid specifies where an observation remains descriptive and where additional evidence is required before moving toward mechanism, fitness consequence, or generalization.

Table 4. Cross-Stage Evidence Changes Meaning Across Biological Scale: Boundaries Between Developmental Observation, Adult Consequence, and Life-History Inference

Exposure and metamorphic transition		
Cross stage comparison dimension	**Developmental exposure**	**Metamorphic transition**
Directly observed quantity	Diet, temperature, density, microbes, chemical or predator treatment	Development time, emergence, mass, morphology, measured transstadial state
Legitimate inference	Individuals experienced defined juvenile conditions	Earlier conditions remain associated with transition phenotype
Additional bridge required	That the exposure created an adaptive developmental response	That the same mechanism or biological carrier persists into adulthood
Boundary and reporting priority	Dose, timing, duration and developmental stage Report exposure chronology and stage precisely	Tissue remodeling and biological reorganization Distinguish phenotype persistence from mechanism persistence
Adult physiology and life history		
Cross stage comparison dimension	**Adult physiology**	**Adult life history**
Directly observed quantity	Metabolism, immunity, reserves, microbiota, stress response	Fecundity, mating success, survival, longevity, ageing
Legitimate inference	Developmental history is associated with a later physiological state	Earlier conditions have later life-history consequences
Additional bridge required	That physiological difference improves or reduces fitness	That one endpoint represents total adult fitness
Boundary and reporting priority	Adult environment and challenge conditions Measure adult context concurrently	Sex, age, reproductive opportunity and trait covariance Use multiple age- and sex-resolved endpoints
Population and cross-taxon transfer		

Cross stage comparison dimension	**Population or transmission consequence**	**Cross-taxon generalization**
Directly observed quantity	Population growth or pathogen-related phenotype	Replicated responses across species or populations
Legitimate inference	Individual carry-over may affect a higher-level process	A pattern may extend beyond one experimental system
Additional bridge required	Population persistence, disease transmission, or ecological impact	Universal insect response or conserved mechanism
Boundary and reporting priority	Density dependence, feedback and field environment Validate organismal findings at the relevant ecological scale	Phylogeny, life cycle, ecology and genotype Prioritize comparative, multispecies designs

Cross-stage research agenda

Progress requires designs that follow individuals or cohorts far enough to discriminate developmental success from adult success. Studies should predefine the developmental exposure, the transition at which compensation is expected, and the adult endpoint capable of revealing residual cost. Emergence alone is insufficient when the biological hypothesis concerns reproduction, ageing, immunity, locomotor performance, or stress tolerance.

Factorial designs crossing juvenile and adult environments are particularly important. They distinguish persistence from environment-dependent expression and can reveal rank reversals that remain invisible when every adult is maintained under one benign condition. Sex and age should also be incorporated prospectively because developmental effects may emerge only during reproduction or late life.

Mechanistic studies should measure candidate mediators on both sides of metamorphosis. When microbiota are implicated, composition should be separated from microbial function and adult reacquisition. When energetic carry-over is proposed, reserve quantity, metabolic use, adult feeding, and allocation should be measured rather than inferred from body size. When endocrine or transcriptional pathways are implicated, a persistent molecular signature should not be equated with causal mediation unless perturbation or other appropriate tests connect it to the adult phenotype.

A broader taxonomic program is equally necessary. Current detailed evidence is disproportionately derived from flies and mosquitoes. Comparative work across hemimetabolous and holometabolous insects, across weak and extreme ecological niche shifts, and across species with contrasting reproductive schedules would test whether metamorphic reorganization predicts the probability that developmental effects persist, dissipate, or reappear in different traits.

Finally, future syntheses should preserve null results and trait discordance. A study in which juvenile stress alters fecundity but not lifespan is more informative for cross-stage allocation than a binary label of “carry-over detected.” Reporting exposure timing, developmental rate, multiple adult traits, sex, age, adult environment, and mechanistic evidence status would make future evidence quantitatively comparable without forcing biologically different outcomes into one effect category.

Conclusion

Developmental history remains biologically relevant beyond the stage in which an environmental exposure occurs, but its later expression is neither uniform nor inevitable. Across insect life histories, persistent carry-over, compensation, reversal, and hidden adult cost represent different trajectories that can coexist among traits within the same organism.

The central distinction is therefore between survival of a developmental effect and recovery of the organism. Normal emergence, restored size, or unchanged longevity cannot establish complete compensation when reproductive, metabolic, immune, behavioral, or stress-response consequences remain. Conversely, an adult difference does not establish persistence of the original developmental mechanism through metamorphosis.

A trajectory-based approach preserves these distinctions. It treats metamorphosis as a biological and inferential boundary, adult environment as a modifier rather than an afterthought, and trait discordance as evidence about allocation rather than noise. This framework is proposed as an organizing synthesis rather than a universally validated model. Its value will depend on longitudinal, factorial, mechanistically explicit, and taxonomically

broader tests that connect developmental conditions to multiple adult outcomes without confusing phenotypic persistence with causation or compensation with absence of cost.

Acknowledgments: None

Conflict of Interest: None

Financial Support: None

Ethics Statement: None

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