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Metamorphosis Selectively Filters Early-Life Effects so That Some Persist, Others Disappear, and Others Reverse in Adult Insects

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ABSTRACT

Complete metamorphosis is often treated either as a developmental reset that disconnects juvenile and adult phenotypes or as a stage boundary across which early-life effects simply "carry over." Both interpretations are too coarse. Evidence from holometabolous insects shows that larval temperature, nutrition, crowding, predator exposure, microbial conditions, toxicants, and social information can influence later phenotypes, yet the affected adult traits, directions, and conditions vary markedly. This perspective argues that metamorphosis should instead be viewed as a selective filter acting on biological states with different capacities to survive, be reconstructed, be compensated, or become conditionally expressed. Persistence is most plausible when a relevant substrate, state, or downstream consequence bridges metamorphic remodeling; disappearance may reflect tissue replacement, state reinitialization, or selective loss of affected individuals; compensation requires a later adjustment rather than mere absence of an adult difference; and phenotypic reversal should be reserved for sign changes in the same homologous response dimension. A further class comprises latent effects that become detectable only under adult challenge. The proposed selective-filter model therefore separates observed adult outcomes from their possible mechanisms and from fitness or adaptive interpretation. It yields comparative predictions based on tissue fate, exposure timing, sex, taxon, developmental environment, and adult context while emphasizing that most existing studies measure cohort-level phenotypes rather than continuous individual trajectories. The framework is intended to organize, not replace, mechanistic tests.

Keywords: Carry-over effects, Complete metamorphosis, Developmental plasticity, Tissue remodeling, Compensatory response, Life-stage interactions

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Introduction

Complete metamorphosis creates one of the sharpest developmental transitions in animal life histories. Larval organs, tissues, physiology, and behavior can be extensively reorganized during pupation, while adult structures and functions emerge under endocrine control [1]. This transformation can increase ecological and functional separation between juvenile and adult stages, but it does not imply that every larval substrate or state is destroyed and rebuilt independently [2]. The central problem is therefore not whether early environments can matter later, but which components of an early-life effect remain biologically available to influence the adult phenotype after metamorphic remodeling.

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Carry-over theory already recognizes that conditions in one life stage may alter performance in another and that such effects need not have a single fitness meaning [3]. Empirical work likewise shows persistence without uniformity. Larval heat stress in *Drosophila melanogaster* altered adult feeding, locomotion, metabolites, and insulin-signaling-related transcription, with different adult traits changing in different directions [4]. Treating this entire outcome as one carry-over effect obscures the more informative question: what was preserved, what was reconstructed, and what changed only because later development responded to the earlier perturbation?

This perspective proposes that metamorphosis acts as a selective filter rather than as either a universal reset or a passive conduit. The argument separates five postmetamorphic outcome classes: persistence, erasure, compensation, phenotypic reversal, and latent expression under adult challenge. These are outcome categories, not assumed mechanisms. A persistent adult phenotype need not contain the original larval molecular state; an adult null effect need not prove biological erasure; and an apparent opposite effect need not constitute reversal if different traits are being compared. The analytical task is therefore to match adult outcomes to developmental substrates, timing, tissue fate, and present environmental context without extending evidence beyond the taxa, traits, or experimental scales actually studied.

Classes of early-life effects

The exposures capable of generating later-stage effects are biologically heterogeneous. Predator experience can alter behavior across developmental transitions, but detectability may depend on the current environment: in *Aedes aegypti*, larval predator experience affected pupal antipredator behavior under some resource conditions but not others [5]. Chronic predator-associated stress also produced adult physiological and life-history consequences in two *Anopheles* species, with responses differing between species and sexes [6]. These cases show why "early-life stress" is not a mechanistic category; similar ecological labels can act through different sensory, energetic, endocrine, or allocation pathways.

Nutritional and thermal histories similarly produce multi-trait rather than unitary outcomes. In *Aedes albopictus*, larval diet and temperature altered development, body size, immune-related measures, and relationships with adult longevity, but the measured proxies did not respond as interchangeable indicators of a single latent condition [7]. Conversely, sublethal photosensitive insecticide exposure altered juvenile development in *Anopheles gambiae* while the adult life-history traits tested were largely unaffected [8]. Combined exposures can also be amplified across the transition: in a damselfly, food stress and pollutant exposure produced survival-related effects that strengthened during and after metamorphosis [9]. Thus, persistence, attenuation, and amplification can coexist across different traits and exposure combinations.

Exposure magnitude and timing add further structure. Larval crowding in *D. melanogaster* produced density-dependent transcriptomic and developmental responses that were not reducible to a simple linear stress gradient [10]. In the moth *Spodoptera littoralis*, adult host preference depended on when larval host experience occurred, identifying a sensitive developmental period rather than uniform retention of all experience [11]. Under seminatural conditions, pesticide carry-over to female damselfly fitness depended strongly on egg-hatching phenology [12]. These studies support a first principle of the proposed framework: an early-life effect should be classified by the trajectory of a defined trait or state across development, not by the mere fact that juvenile exposure occurred.

Persistence mechanisms

Persistence across metamorphosis requires some route by which developmental history continues to influence later phenotype. Endocrine coordination provides the temporal architecture for such continuity and transformation. Steroid-hormone action in *Drosophila* governs developmental timing and tissue-specific responses, with competence depending on developmental state rather than hormone exposure alone [13]. More broadly, juvenile hormone and ecdysteroid systems coordinate metamorphic transitions across insects, making tissue fate and developmental timing central to any mechanistic account of what can persist [14]. These systems establish plausibility for gating and remodeling, but they do not by themselves demonstrate that a particular early-life perturbation is "remembered" endocrinologically.

The nervous system provides direct evidence that metamorphosis can combine continuity with reconstruction. *Drosophila* postembryonic neural development includes programmed cell death, pruning, remodeling, and adult-specific neurogenesis, so adult circuitry contains both retained and newly generated components [15–17]. Identified larval neurons can survive metamorphosis and be repurposed for adult sexual behavior, while other

homologous cells are eliminated in a sex-specific manner. Such findings establish selective cellular continuity, not automatic continuity of larval behavior or learned information. A circuit can preserve constituent cells while substantially changing connectivity and function.

Host-associated microbes provide a second, mechanistically different route. Experimental manipulation of larval bacterial conditions in mosquitoes can generate metabolic changes with later adult fitness-related consequences [18]. Direct work in *Drosophila* shows that some microbial strains can persist transstadially whereas others do not, with host species, strain identity, and sex influencing transmission [19]. Comparative evidence likewise indicates that metamorphosis can disrupt, protect, or require reacquisition of different host–symbiont associations [20]. The appropriate inference is selective microbial continuity rather than intact inheritance of a larval microbiome.

Equally important, adult phenotype can depend on the current postmetamorphic microbiota. *Aedes aegypti* deprived of a normal adult gut community showed altered fecundity, longevity, metabolism, and vector competence, with phenotypic rescue under appropriate microbial restoration [21]. Developmental history and adult state must therefore be distinguished even when both involve the same biological subsystem. Persistence may arise through survival and repurposing of biological substrates, enduring downstream consequences of an early perturbation, or reconstruction of adult state under constraints generated earlier in development. Similar adult phenotypes can consequently emerge from different histories, and persistence of phenotype is weaker evidence than persistence of mechanism.

Erasure through tissue replacement

Erasure is the most tempting inference from complete metamorphosis, but it requires stricter evidence than visible anatomical transformation. Tissue replacement can remove cells, local molecular states, stored resources, or microbial communities that carried an early-life effect, yet metamorphic remodeling is mosaic rather than total. Tissue turnover therefore creates an opportunity for erasure rather than proving that erasure occurred.

The strongest empirical support comes from cases in which a juvenile response is detected but later-stage measurements converge. Larval insecticide exposure can alter development without producing detectable differences in the adult traits examined [8]. Likewise, juvenile immune challenges in an insect produced measurable early responses that were not carried into subsequent stages [22]. These findings justify "no detectable later-stage effect" for the measured endpoints, but not organism-wide resetting. Unmeasured molecular states, altered developmental timing, or differential survival could still distinguish the adults that reach measurement.

Microbiomes expose the same interpretive problem at another level. Transstadial tracking demonstrates selective microbial continuity rather than uniform persistence [19], while host–symbiont associations can also be disrupted or reacquired during metamorphosis [20]. Field evidence from aquatic insects further shows substantial shifts in microbial diversity and composition between larval and adult stages under chronic metal exposure [23]. Such restructuring is consistent with strong filtering, but observational stage differences cannot by themselves identify tissue replacement as the causal mechanism.

An erasure claim must therefore specify what disappeared, at which biological level, relative to which comparator, and with which alternatives excluded. The distinction is especially important when adult means converge after juvenile mortality, because disappearance of vulnerable individuals can mimic biological recovery. The following decision matrix separates patterns that are often compressed into the single idea of a metamorphic "reset."

Table 1. Distinguishing developmental erasure from apparent postmetamorphic convergence

Reset candidate	Biological context and cross-stage evidence	Permitted claim and critical mimic	Minimum test and state
Remodeling, removal, and selective continuity			
Juvenile difference absent in adult assay	Same or closely corresponding trait across stages	Later expression is attenuated or undetected	Track comparable endpoints across the transition
	Detectable juvenile difference followed by adult convergence	Assay sensitivity and selective survival can mimic erasure	Evidence-supported pattern
Local substrate removed during remodeling	Tissue or cell class known to be eliminated	Direct continuity through that substrate becomes unlikely	Couple lineage fate with later phenotype
	Developmental lineage or tissue-fate evidence	Another substrate may transmit an indirect consequence	Proposed inference rule

Selective cellular continuity	Tissues containing both surviving and replaced elements Retained and eliminated cell classes coexist	Erasure must be component-specific Surviving cells may be repurposed	Map cell fate and adult function together Evidence-supported pattern
Microbial restructuring			
Microbial restructuring	Gut or other host-associated community Loss, turnover, persistence, or reacquisition across stages	Metamorphosis filters microbial continuity Similar adult taxa can be environmentally reacquired	Use strain-level transstadial tracking Evidence-supported pattern
Differential-survival artifact			
Adult convergence after differential juvenile mortality	Cohort-level adult comparison Survivors become phenotypically similar despite earlier divergence	Erasure remains unproven Cohort composition changed before adult measurement	Condition adult inference on survival history Proposed caution

Compensation

Compensation differs from erasure because it implies that an early perturbation continues to matter developmentally even though a later trait is restored, buffered, or offset. A null adult difference is therefore insufficient evidence of compensation. In *Ischnura elegans*, predator-cue exposure at egg or larval stages affected some developmental and survival traits while mass at emergence was unchanged [24]. This demonstrates trait-specific carry-over and convergence, but compensation can be claimed only when evidence supports a restorative or offsetting process rather than merely an unchanged endpoint.

Nutritional experiments sharpen this distinction. In *Speyeria mormonia*, larval food restriction carried into adult energetics and life-history traits, yet adult outcomes did not shift uniformly: some traits retained costs whereas others were buffered [25]. Metamorphosis can therefore redistribute developmental consequences across traits without returning the organism to an undisturbed biological state. At a higher level, developmental warming in *Enchenopa binotata* reduced juvenile survival while increasing adult fertility within the tested range, partly offsetting demographic consequences in a population model [26]. This is cross-component compensation, not reversal of a single trait.

The proposed framework accordingly distinguishes same-trait restoration, buffering of a focal adult output while another state remains displaced, and cross-component offsetting among distinct fitness-related traits. These states are analytically separate because an apparently normal adult endpoint may coexist with retained energetic, physiological, or demographic costs. **Figure 1** preserves those parallel trajectories and the principal alternatives that can imitate compensation.

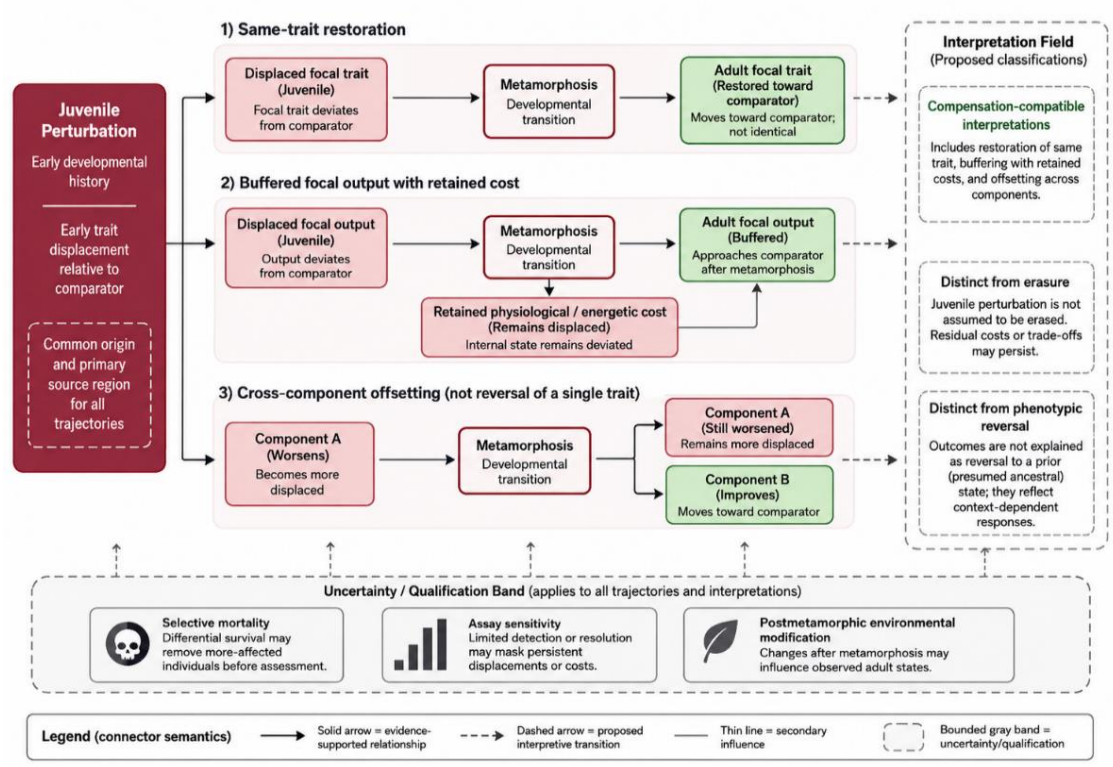


Figure 1. Compensation can conceal retained developmental costs without restoring the original biological

Phenotypic reversal

Phenotypic reversal is the narrowest and most easily overused outcome class. Here, reversal means that the direction of a response in the same homologous trait changes across developmental context relative to an appropriate comparator. Opposing effects on different traits do not qualify. Developmental warming in *E. binotata*, for example, decreased juvenile survival while increasing adult fertility [26]. Those responses may offset one another at a higher demographic level, but they are different fitness components and therefore constitute compensation rather than phenotypic reversal.

Stage-specific thermal experiments show why the distinction matters. In *Ostrinia nubilalis*, reproductive consequences depended on whether elevated temperature occurred during larval, pupal, or adult stages [27]. Such timing dependence can produce adult responses that vary in magnitude or direction among treatment histories, but it does not itself demonstrate that metamorphosis inverted an earlier phenotype. A convincing reversal requires a comparable response dimension on both sides of the developmental contrast or another design that makes the sign change unambiguous.

The current environment adds a second source of apparent reversal. In *Manduca sexta*, gene-expression responses depended jointly on previous and current thermal conditions [28]. A developmental-history × current-environment interaction can therefore generate opposite adult responses in different current contexts without making metamorphosis itself the causal inverter. Similar ambiguity can arise from acclimation, altered developmental timing, selective survival, or comparison of nonhomologous endpoints.

The proposed framework consequently assigns reversal a deliberately high evidential threshold. This conservatism prevents a biologically interesting but common pattern—different traits responding in opposite directions—from being mislabeled as reversal. The register below separates genuine candidate reversal from the principal patterns that can imitate it.

Table 2. Discriminating phenotypic reversal from cross-trait compensation and context-dependent expression

Same-trait reversal versus cross-trait compensation		
Phenotypic reversal comparison dimension	**Same-trait sign change**	**Opposing effects on distinct fitness components**

Context and sign pattern	Homologous response dimension across developmental contexts Response relative to comparator changes sign	Survival versus fertility or another nonhomologous trait pair One component decreases while another increases
Correct interpretation	Candidate phenotypic reversal	Cross-component compensation
Principal mimic	Measurement scales may differ between stages	Aggregate consequence depends on weighting and environment
Discriminating test and state	Use biologically comparable endpoints before and after the transition Proposed diagnostic class	Retain component-specific trajectories Evidence-supported distinction
	Exposure timing and adult-environment dependence	
Phenotypic reversal comparison dimension	**Exposure-stage dependence**	**Developmental-history × adult-environment interaction**
Context and sign pattern	Larval, pupal, and adult exposures differ Adult response varies with exposure timing	Past and current environments jointly shape phenotype Adult response direction changes among current contexts
Correct interpretation	Stage-specific sensitivity	Context-dependent expression
Principal mimic	Effective dose and developmental physiology can differ	Current conditions may determine the apparent sign
Discriminating test and state	Match exposure factorially across developmental windows Evidence-supported pattern	Cross developmental and adult environments factorially Evidence-supported pattern
	Selective-survival convergence	
Phenotypic reversal comparison dimension	**Adult convergence or sign change after selective survival**	
Context and sign pattern	Mortality occurs before adult measurement Survivor mean normalizes or becomes opposite	
Correct interpretation	Reversal not established	
Principal mimic	Cohort composition changed	
Discriminating test and state	Track survival-conditioned individual or family trajectories Proposed caution	

Latent effects revealed by adult challenge

Developmental history may remain biologically consequential even when adults appear similar under benign baseline conditions. The strongest evidence for such latency comes from designs in which an adult challenge reveals a difference among developmental histories. Larval competition between mosquitoes, for example, altered subsequent arboviral infection measured after adult challenge [29]. Likewise, larval exposure to *Bacillus thuringiensis* in tolerant *Aedes albopictus* was associated with later differences spanning life-history, immune, and microbiome-related traits [30]. These outcomes demonstrate developmental-history dependence, but they do not identify a single persisting causal substrate.

Molecular persistence can provide another candidate latent state. Adult monarch butterflies retained brain-transcriptomic differences associated with diapause history despite subsequent adult conditions being standardized [31]. Such molecular signatures are important because adult phenotypic equivalence at one observational level does not require equivalence at all underlying levels. Nevertheless, a persistent transcriptomic state should not be equated automatically with altered performance, fitness, or adaptive preparation. Under the proposed terminology, latency is best reserved for cases in which developmental histories produce little or no detectable difference under one adult condition but diverge when the adult environment, infection state, resource regime, or another challenge changes.

Selective-filter model of metamorphosis

The evidence considered here supports neither universal developmental continuity nor universal resetting. The proposed Selective-Filter Model of Metamorphosis instead treats the transition to adulthood as a transformation in which early-life effects encounter several biologically distinct filters. The first is substrate continuity: cells, circuits, symbionts, metabolites, or other states differ in whether they survive metamorphic remodeling. Selective neuronal persistence [15] and strain-specific microbial continuity [19] illustrate this principle without implying that the original juvenile function necessarily persists unchanged.

The second filter is developmental reconstruction. Even when the original substrate disappears, earlier conditions may alter timing, allocation, tissue construction, or adult state indirectly. Conversely, substantial juvenile disruption can be followed by little detectable adult difference [8]. The third is postmetamorphic compensation, in which later allocation or physiology buffers one endpoint while another remains displaced, as demonstrated by trait-specific nutritional carry-over [25]. The fourth is adult-context gating, whereby the expression of developmental history depends on present conditions. Thermal-history interactions [28] and challenge-dependent infection phenotypes [29] exemplify this dependence.

The model therefore classifies outcomes only after specifying the focal trait, developmental comparator, biological level, and adult context. Persistence means continued developmental-history dependence in the focal outcome; erasure means that a previously demonstrated difference is no longer detectable at the defined level after alternatives such as selective mortality are considered; compensation requires evidence of offsetting or restoration rather than simple convergence; reversal requires a sign change in a homologous response dimension; and latency requires context-dependent revelation of a developmental-history effect. These categories are proposed analytical states rather than mutually exclusive organism-wide labels.

Because evidence is uneven across these filters, a coverage-and-gap map is needed to distinguish well-supported biological continuity from the more provisional interpretive rules built upon it.

Table 3. Evidence coverage and unresolved tests within the selective-filter interpretation of metamorphosis

Substrate continuity and reconstruction			
Selective filter comparison dimension	**Substrate continuity**	**Developmental reconstruction**	
Present coverage	Strong examples from identified neurons and selected microbial strains	Juvenile perturbations can alter later phenotype despite extensive remodeling	
Biological level	Cell, circuit, microbiome	Tissue, physiology, organism	
Interpretation enabled	Some juvenile-origin substrates survive metamorphosis selectively	Adult effects need not require survival of the original juvenile state	
Gap, priority test, and state	Limited matched evidence across insect orders and tissues Longitudinal lineage or strain tracking linked to adult function Evidence-supported dimension	Mediating developmental steps are often unmeasured Repeated mechanistic measurements before, during, and after metamorphosis Evidence-supported dimension	
Erasure, compensatory filtering, and adult gating			
Selective filter comparison dimension	**Apparent erasure**	**Compensatory filtering**	**Adult-context gating**
Present coverage	Later-stage convergence occurs for selected measured traits	Adult traits can be buffered while other costs persist	Developmental history interacts with later thermal, ecological, or infection context
Biological level	Physiology, immunity, life history	Energetics, reproduction, fitness components	Molecular, behavioral, infection phenotype
Interpretation enabled	Some effects become undetectable after transition	Recovery of one endpoint need not equal restoration of whole-organism state	Early effects can remain conditional or latent

Gap, priority test, and state	Selective mortality and assay insensitivity can imitate erasure Survival-conditioned longitudinal measurement Evidence-supported pattern; causal rule proposed	Active compensation rarely separated from passive convergence Measure compensatory process and retained cost simultaneously Proposed classification	Limited factorial designs across multiple adult contexts Cross developmental-history and adult-environment treatments Evidence-supported dimension
	True reversal		
Selective filter comparison dimension	**True phenotypic reversal**		
Present coverage	Direct demonstrations remain comparatively sparse		
Biological level	Trait-specific		
Interpretation enabled	Reversal should require homologous-trait sign change		
Gap, priority test, and state	Different traits are often compared as though they reversed Same-trait longitudinal or stage-matched factorial test Proposed high-threshold class		

The model's multilevel logic is summarized visually only after these evidence-supported and proposed components are separated.

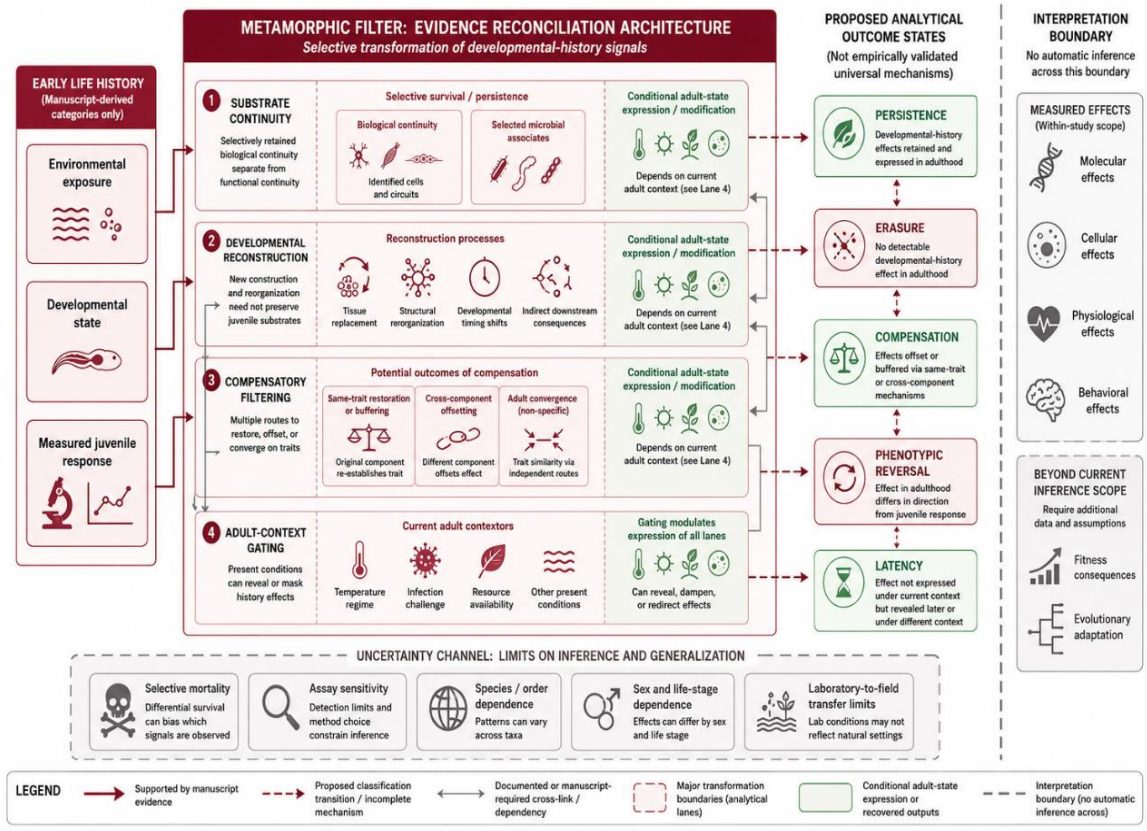


Figure 2. Metamorphic filtering reconciles substrate continuity, developmental reconstruction, and adult-context dependence

Comparative predictions

The proposed model generates predictions that can be falsified rather than merely accommodated retrospectively. First, continuity should depend on when exposure occurs. Late larval social information altered adult sperm

allocation in *Ephestia kuehniella*, indicating a developmental sensitive window [32]. The model therefore predicts that equally intense exposures imposed at different developmental windows will differ in adult persistence when they intersect differently with tissue commitment, endocrine timing, or substrate survival.

Second, developmental and adult environments should be crossed factorially rather than studied separately. Adult oviposition behavior in *Pararge aegeria* depended on larval experience together with ecotype and adult environmental experience [33]. Such interactions predict that an early-life effect classified as absent under one adult environment may become detectable under another. This is especially important for distinguishing erasure from latency.

Third, adult molecular states should be compared with organismal outcomes rather than treated as interchangeable evidence. Adult *Drosophila simulans* retained transcriptomic signatures of larval crowding [34], yet a molecular signature alone does not establish persistent impairment or fitness consequence. Comparative studies should therefore pair substrate-level continuity with homologous adult traits and, where possible, survival-conditioned longitudinal designs.

Finally, true reversal should remain the hardest category to assign. Stage-dependent thermal effects [27] show that exposure timing can change reproductive responses, but demonstrating reversal requires following the same biologically comparable dimension or an explicitly homologous response across contexts. The model predicts that many published "opposite" carry-over effects will resolve instead into cross-trait compensation, context dependence, or cohort selection when analyzed with these stricter criteria.

Results and Discussion

The principal value of the selective-filter interpretation is that it prevents metamorphosis from being treated as a binary boundary. Evidence for persistent neural substrates, microbial continuity, adult molecular signatures, and long-lasting physiological effects coexists with explicit later-stage null effects and major tissue restructuring. Neither "developmental memory" nor "complete reset" therefore functions well as a general explanation.

Mechanistic inference remains the largest weakness in the evidence base. Adult phenotypes are often measured after juvenile exposure without tracking the intervening substrate. An altered adult trait may arise from surviving cells, resource depletion, altered timing, microbial change, developmental reconstruction, or selective mortality. Conversely, adult convergence may reflect genuine attenuation, compensation, insensitive measurement, or survival filtering. These alternatives cannot be resolved from endpoint comparisons alone.

Taxonomic generalization also requires restraint. Much of the direct mechanistic evidence comes from *Drosophila* and mosquitoes, while Lepidoptera, Odonata, Hemiptera, and other groups provide important but less densely sampled comparisons. Tissue remodeling, ecological separation between stages, diapause, sex-specific development, and microbial dependence differ among lineages. The proposed outcome categories may therefore be broadly useful while the relative frequency of each filtering route remains taxon-specific and empirically unresolved.

The same caution applies to evolutionary interpretation. Persistence can be costly, neutral, beneficial, or simply unavoidable; compensation need not restore total fitness; and history-dependent responses are not automatically anticipatory or adaptive. Future tests should combine matched developmental and adult environments, repeated measurements across metamorphosis, explicit mortality accounting, tissue or lineage tracking where feasible, and adult challenges selected before outcomes are known. Such designs would convert the model from an organizing hypothesis into a set of discriminating mechanistic tests.

Conclusion

Metamorphosis neither guarantees the disappearance of early-life effects nor allows them to pass unchanged into adulthood. Its biological importance lies in selective transformation. Some developmental substrates persist, others are replaced, some early perturbations are buffered, and some consequences become visible only under particular adult conditions. Distinguishing persistence, erasure, compensation, reversal, and latency therefore requires trait-specific, level-specific, and context-specific evidence. The proposed selective-filter model provides a disciplined way to organize these alternatives while keeping phenotype, mechanism, fitness consequence, and evolutionary interpretation separate. Its main empirical challenge is now to follow developmental histories

through metamorphosis closely enough to identify which biological information actually survives, which is reconstructed, and which only appears to have vanished.

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References

1. Konopová B. Evolution of insect metamorphosis—an update. Curr Opin Insect Sci. 2025;67:101289. doi:10.1016/j.cois.2024.101289
2. Rolff J, Johnston PR, Reynolds S. Complete metamorphosis of insects. Philos Trans R Soc Lond B Biol Sci. 2019;374(1783):20190063. doi:10.1098/rstb.2019.0063
3. Moore MP, Martin RA. On the evolution of carry-over effects. J Anim Ecol. 2019;88(12):1832-44. doi:10.1111/1365-2656.13081
4. Karpova EK, Bobrovskikh MA, Burdina EV, Adonyeva NV, Deryuzhenko MA, Zakharenko LP, et al. Larval stress affects adult Drosophila behavior and metabolism. J Insect Physiol. 2024;159:104709. doi:10.1016/j.jinsphys.2024.104709
5. Rawat K, Bhambore AA, Isvaran K. Don't leave the past behind: Larval experience shapes pupal antipredator response in Aedes aegypti. Behav Ecol. 2025;36(2). doi:10.1093/beheco/araf001
6. Roux O, Renault D, Mouline K, Diabaté A, Simard F. Living with predators at the larval stage has differential long-lasting effects on adult life history and physiological traits in two anopheline mosquito species. J Insect Physiol. 2021;131:104234. doi:10.1016/j.jinsphys.2021.104234
7. Mackay AJ, Yan J, Kim CH, Barreaux AMG, Stone CM. Larval diet and temperature alter mosquito immunity and development: Using body size and developmental traits to track carry-over effects on longevity. Parasit Vectors. 2023;16(1):434. doi:10.1186/s13071-023-06037-z
8. Meier CJ, Martin LE, Hillyer JF. Mosquito larvae exposed to a sublethal dose of photosensitive insecticides have altered juvenile development but unaffected adult life history traits. Parasit Vectors. 2023;16(1):412. doi:10.1186/s13071-023-06004-8
9. Jorissen S, Janssens L, Verheyen J, Stoks R. Synergistic survival-related effects of larval exposure to an aquatic pollutant and food stress get stronger during and especially after metamorphosis and shape fitness of terrestrial adults. Environ Pollut. 2023;326:121471. doi:10.1016/j.envpol.2023.121471
10. Morimoto J, Wenzel M, Derous D, Henry Y, Colinet H. The transcriptomic signature of responses to larval crowding in Drosophila melanogaster. Insect Sci. 2023;30(2):539-54. doi:10.1111/1744-7917.13113
11. Lhomme P, Khallaf Ali M, Larsson M, Anderson P. A sensitive period for the induction of host plant preference in a generalist herbivorous insect. Anim Behav. 2020;169:1-8. doi:10.1016/j.anbehav.2020.08.020
12. Tüzün N, Stoks R. Carry-over effects across metamorphosis of a pesticide on female lifetime fitness strongly depend on egg hatching phenology: A longitudinal study under seminatural conditions. Environ Sci Technol. 2017;51(23):13949-56. doi:10.1021/acs.est.7b04399
13. Morrow H, Mirth CK. Timing Drosophila development through steroid hormone action. Curr Opin Genet Dev. 2024;84:102148. doi:10.1016/j.gde.2023.102148
14. Truman JW, Riddiford LM. The evolution of insect metamorphosis: A developmental and endocrine view. Philos Trans R Soc Lond B Biol Sci. 2019;374(1783):20190070. doi:10.1098/rstb.2019.0070
15. Truman JW, Riddiford LM. Drosophila postembryonic nervous system development: A model for the endocrine control of development. Genetics. 2023;223(3). doi:10.1093/genetics/iyac184
16. Diamandi JA, Duckhorn JC, Miller KE, Weinstock M, Leone S, Murphy MR, et al. Developmental remodeling repurposes larval neurons for sexual behaviors in adult Drosophila. Curr Biol. 2024;34(6):1183-93.e3. doi:10.1016/j.cub.2024.01.065

17. Truman JW, Price J, Miyares RL, Lee T. Metamorphosis of memory circuits in Drosophila reveals a strategy for evolving a larval brain. Elife. 2023;12. doi:10.7554/eLife.80594
18. Giraud E, Varet H, Legendre R, Sismeiro O, Aubry F, Dabo S, et al. Mosquito-bacteria interactions during larval development trigger metabolic changes with carry-over effects on adult fitness. Mol Ecol. 2022;31(5):1444-60. doi:10.1111/mec.16327
19. Guilhot R, Xuéreb A, Lagmairi A, Olazcuaga L, Fellous S. Microbiota acquisition and transmission in Drosophila flies. iScience. 2023;26(9):107656. doi:10.1016/j.isci.2023.107656
20. Hammer TJ, Moran NA. Links between metamorphosis and symbiosis in holometabolous insects. Philos Trans R Soc Lond B Biol Sci. 2019;374(1783):20190068. doi:10.1098/rstb.2019.0068
21. Harrison RE, Yang X, Eum JH, Martinson VG, Dou X, Valzania L, et al. The mosquito Aedes aegypti requires a gut microbiota for normal fecundity, longevity and vector competence. Commun Biol. 2023;6(1):1154. doi:10.1038/s42003-023-05545-z
22. Silva FWS, Viol DL, Elliot SL. Juvenile responses to immune challenges are not carried through to subsequent life stages in an insect. Sci Rep. 2024;14(1):21456. doi:10.1038/s41598-024-72546-1
23. Perrotta BG, Kidd KA, Marcarelli AM, Paterson G, Walters DM. Effects of chronic metal exposure and metamorphosis on the microbiomes of larval and adult insects and riparian spiders through the aquatic-riparian food web. Environ Pollut. 2025;371:125867. doi:10.1016/j.envpol.2025.125867
24. Sniegula S, Raczyński M, Golab MJ, Johansson F. Effects of predator cues carry over from egg and larval stage to adult life-history traits in a damselfly. Freshw Sci. 2020;39(4):804-11. doi:10.1086/711374
25. Niitepõld K, Boggs CL. Carry-over effects of larval food stress on adult energetics and life history in a nectar-feeding butterfly. Ecol Entomol. 2022;47(3):391-9. doi:10.1111/een.13124
26. Leith NT, Macchiano A, Tenhumberg B, Smith I, Woods JP, Fowler-Finn KD. Carryover effects on reproduction can buffer against mortality-driven population declines at elevated developmental temperatures. Ecol Lett. 2025;28(11). doi:10.1111/ele.70264
27. Velikaneye BA, Kozak GM. Timing-dependent effects of elevated temperature on reproductive traits in the European corn borer moth. J Evol Biol. 2024;37(9):1076-90. doi:10.1093/jeb/voae092
28. Alston MA, Lee J, Moore ME, Kingsolver JG, Willett CS. The ghost of temperature past: Interactive effects of previous and current thermal conditions on gene expression in Manduca sexta. J Exp Biol. 2020;223(7). doi:10.1242/jeb.213975
29. Vanslembrouck A, Jansen S, De Witte J, Janssens C, Vereecken S, Helms M, et al. Larval competition between Aedes and Culex mosquitoes carries over to higher arboviral infection during their adult stage. Viruses. 2024;16(8):1202. doi:10.3390/v16081202
30. Bahrami R, Quaranta S, Perdomo HD, Bonizzoni M, Khorramnejad A. Carry-over effects of Bacillus thuringiensis on tolerant Aedes albopictus mosquitoes. Parasit Vectors. 2024;17(1):456. doi:10.1186/s13071-024-06556-3
31. Stratton SM, Green DA 2nd. Diapause history has lasting effects on adult brain gene expression in monarch butterflies. J Insect Physiol. 2025;167:104893. doi:10.1016/j.jinsphys.2025.104893
32. Liu J, He XZ, Wang Q. Male larval experience of cues from adult rivals alters lifetime sperm investment patterns in a sperm heteromorphic moth, Ephestia kuehniella. Insect Sci. 2023;30(6):1773-83. doi:10.1111/1744-7917.13204
33. Braem S, Van Dyck H. Larval and adult experience and ecotype affect oviposition behavior in a niche-expanding butterfly. Behav Ecol. 2023;34(4):547-61. doi:10.1093/beheco/arad022
34. Buchner S, Hsu SK, Nolte V, Otte KA, Schlötterer C. Effects of larval crowding on the transcriptome of Drosophila simulans. Evol Appl. 2023;16(10):1671-9. doi:10.1111/eva.13592