Eurasia Specialized Veterinary Publication

Entomological Research Letters

ISSN: 3108-639X

2024, Volume 4, Issue 1, Page No: 36-44
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Available online at: www.esvpub.com/

Complete Metamorphosis Does Not Fully Erase Earlier Biological States Because Cross-Stage Constraints and Compensation Persist

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ABSTRACT

Complete metamorphosis creates a profound developmental discontinuity between juvenile and adult insects, but discontinuity is not equivalent to biological erasure. Current interpretations often emphasize either metamorphic decoupling, in which larval and adult stages gain developmental or evolutionary independence, or carry-over effects, in which juvenile conditions remain detectable in adults. Treating these as competing alternatives obscures an important possibility: metamorphosis may selectively reset some components of biological state while retaining, remodeling, reconstructing, or compensating for others. This theoretical synthesis develops a proposed selective-state transformation view of holometabolous development. It distinguishes states that can be effectively reset from constraints that survive metamorphosis, then separates compensation from true restoration. Evidence from developmental regulation, neural remodeling, microbiome turnover, nutritional and thermal stress, regeneration, and adult performance indicates that persistence is carrier-, trait-, and context-dependent. Apparent adult recovery in one phenotype may therefore coexist with altered structure, metabolism, immunity, energetics, or repair history in another. The framework does not assume that persistent differences are adaptive, costly, or mechanistically identical across taxa. Instead, it treats metamorphic decoupling as incomplete, multidimensional, and testable through longitudinal measurements that distinguish disappearance of a state from transformed expression or compensated consequence.

Keywords: Holometaboly, Metamorphic decoupling, Developmental carry-over, Compensatory growth, Physiological constraint, Life-stage plasticity.

Received: 21 March 2024
Revised: 24 May 2024
Accepted: 26 May 2024

How to Cite This Article: Collins G, Evans O, Williams N, Brooks E. Complete Metamorphosis Does Not Fully Erase Earlier Biological States Because Cross-Stage Constraints and Compensation Persist. Entomol Appl Sci Lett. 2024;4(1):36-44. https://doi.org/10.51847/qzDgsGTr92

Introduction

Complete metamorphosis reorganizes the insect body at a scale that can make larval and adult phenotypes appear biologically discontinuous. Larval organs may be remodeled, replaced, or integrated into newly constructed adult structures, while the intervening pupal stage reorganizes tissue relationships and functional priorities. This extensive reconstruction is central to holometaboly, but it does not logically establish that all earlier biological states are erased [1]. The distinction matters because a structure can disappear while consequences of its prior condition remain, and an adult trait can appear normal even if the developmental route to that state has changed.

One influential interpretation of complete metamorphosis is that separating growth-dominated juvenile stages from differentiation-dominated adult construction can relax conflicts between rapid biomass accumulation and functional specialization. Comparative and theoretical work supports a conditional association between

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holometabolous development, rapid growth, and temporal separation of growth from differentiation [2]. Yet the value of decoupling does not imply perfect independence between stages. Rather, it raises a sharper question: which components of juvenile state become free to reset, which remain constrained by developmental history, and which are altered sufficiently that their persistence can only be detected through another adult phenotype?

Evidence for stage-specific molecular organization strengthens this problem. Whole-transcriptome profiles in the pine sawfly Neodiprion lecontei support substantial larval–adult differentiation in gene-expression architecture [3]. In Drosophila melanogaster, cold exposure elicits strongly stage-specific transcriptomic responses, and RNA interference reveals that genes affecting cold tolerance can differ between larvae and adults [4]. Quantitative-genetic analysis of cold hardiness in the same species likewise found no significant cross-stage genetic correlation for that trait, despite evidence that some physiological mechanisms remain shared [5]. These findings support decoupling at defined molecular or performance levels, not whole-organism independence.

This article therefore addresses an inferential gap between "metamorphosis resets biology" and "juvenile effects carry over." It proposes that complete metamorphosis should instead be treated as a selective state transformation. Under this view, earlier biological states may follow different fates: some are replaced or reconstructed, some survive through retained cellular or structural carriers, some persist only through downstream physiological consequences, and some are phenotypically masked by compensation. These categories are proposed analytical distinctions rather than validated universal states. Their purpose is to prevent adult similarity from being equated automatically with developmental erasure and adult difference from being equated automatically with direct persistence of a juvenile mechanism.

Metamorphic decoupling theory

Metamorphic decoupling is strongest when the biological machinery defining one stage is actively replaced by machinery defining the next. In Drosophila, sequential developmental identity is regulated by the transcriptional regulators Chinmo, Broad, and E93. Functional evidence indicates that these factors form a mutually inhibitory sequence in which larval identity is actively maintained and then relinquished as metamorphic programs emerge [6]. Such regulatory switching demonstrates that stage identity is not merely the passive consequence of elapsed time. It is actively reconstructed, creating a mechanistic basis for genuine discontinuity in selected developmental programs.

The broader developmental architecture of holometaboly reinforces that discontinuity. Adult construction is extensively deferred, and larval and adult morphologies can therefore specialize around very different functional demands [7]. This developmental separation can increase the apparent autonomy of stages, but "decoupling" should not be treated as a scalar property of the whole organism. A gene-regulatory program, a tissue, a microbial community, a neural lineage, and a stored energetic resource do not necessarily cross metamorphosis by the same route. The relevant unit of analysis is therefore the biological carrier of state.

Neural development provides a direct example of selective retention within otherwise radical reorganization. Mapping of Drosophila memory circuitry shows that individual larval neurons and circuit components can have different metamorphic fates, including persistence and remodeling into adult organization [8]. This does not establish that larval memory content itself survives. It instead shows that metamorphosis can preserve selected cellular substrates while altering their connections and functional context. At the level of circuit architecture, persistence and transformation can therefore coexist.

The microbiome provides a contrasting case in which stage transition can promote much stronger replacement. Across 18 insect species, larva-to-adult microbiota turnover was greater in holometabolous than hemimetabolous insects, particularly in community composition rather than simple alpha diversity [9]. This supports the idea that metamorphosis can facilitate a major ecological reset of gut-associated communities. However, even here, turnover is not equivalent to universal sterilization, and the extent of reset depends on post-metamorphic recolonization, host biology, and symbiotic mode.

These examples motivate a refinement of metamorphic decoupling theory. The proposed refinement is that decoupling should be defined at the level of state-bearing components and their transition rules, not inferred from external stage contrast alone. Complete metamorphosis may therefore create a strong developmental boundary while still allowing selected information, material, or constraint to cross that boundary by persistence, remodeling, or downstream propagation. Decoupling and carry-over are thus not opposite outcomes; they can occur simultaneously at different biological levels.

Developmental states that can be reset

A developmental state is effectively reset when the adult condition is reconstructed with little dependence on the corresponding larval state, even if the organism as a whole retains other consequences of larval history. Honey bees provide a useful microbial example. Experimental perturbation of larval microbiota did not prevent newly emerged adults from converging on characteristic adult core communities after adult inoculation and social transmission [10]. The result supports effective decoupling of larval and adult microbiome states in that system, not wholesale erasure of all host consequences of larval experience.

The degree of microbial resetting varies among insects because microbiota are embedded in different developmental and ecological relationships with their hosts. Reviews of postembryonic host–microbe crosstalk emphasize that developmental strategy, nutritional symbiosis, endocrine interactions, and microbial transmission routes influence whether microbial associations persist or are rebuilt [11]. Consequently, "microbiome reset" is not a single mechanism. In some taxa, destruction or remodeling of gut structures may remove microbial habitat; in others, intracellular or vertically transmitted symbionts may follow different trajectories. Adult recolonization can also reconstruct a community that resembles a species-typical endpoint without retracing the larval route that preceded it.

Cell replacement offers a second form of reset. Drosophila larval epidermal cells normally disappear during development, but experimentally forcing them to persist into the adult epidermis reveals that their behavior changes in the adult context. Cells retained into adulthood acquire aging-associated phenotypes that are not produced simply by prolonging larval life [12]. This experiment is artificial and should not be read as evidence that ordinary larval epidermal states survive metamorphosis. Its importance is conceptual: replacement and retention expose the same cell lineage to fundamentally different state trajectories. A state can disappear because its carrier is removed, whereas a retained carrier can be reprogrammed by the post-metamorphic environment.

The proposed framework therefore distinguishes reset from absence of detectable carry-over. Reset requires evidence that a relevant state-bearing component has been replaced, reconstructed, or rendered functionally independent of its earlier condition. A null adult phenotype alone is insufficient because the measured endpoint may be insensitive to an altered developmental history. Strong evidence for reset should combine knowledge of what is physically or functionally reconstructed with measurements showing that plausible earlier-state differences no longer explain the adult state.

Constraints that survive metamorphosis

The opposite trajectory occurs when larval conditions alter resources, structures, regulatory states, or developmental decisions that cannot be fully rewritten during metamorphosis. In Aedes aegypti, experimentally manipulated larval bacterial environments produced distinct developmental transcriptomic responses and adult differences in lipid storage and starvation resistance [13]. These results do not require the bacteria themselves to persist across metamorphosis. The lasting constraint can instead reside in host physiology established under the earlier microbial environment.

Thermal stress provides another example. Larval heat exposure in Drosophila melanogaster was followed by adult changes in feeding, locomotion, energy stores, and expression of insulin-signaling-associated genes [14]. The evidence supports a cross-stage association between juvenile stress and selected adult phenotypes, but it does not prove that altered insulin signaling is the sole causal mediator. More generally, a constraint can survive because a juvenile perturbation changes the amount, allocation, or developmental timing of material available for adult construction even when the molecular response that initiated the change has ended.

Not all adult traits have the same capacity for post-metamorphic revision. Experimental manipulation of larval and adult nutrition in Drosophila showed that larval diet strongly shaped structural traits such as wing and femur size, whereas adult nutrition more readily altered body mass [15]. This distinction illustrates why adult "recovery" must be defined trait by trait. Post-eclosion feeding can change labile stores and mass without necessarily rewriting dimensions fixed during juvenile growth.

Equally important, plausible carry-over effects can be absent. In female Drosophila, larval protein-to-carbohydrate balance affected adult reproduction, but did not produce corresponding effects on adult survival or on survival consequences of injury and bacterial infection [16]. Such null results prevent the concept of persistence from becoming unfalsifiable. A juvenile exposure may constrain one adult function while leaving another indistinguishable from controls.

Measurement choice can further alter interpretation. In Aedes albopictus, larval diet and temperature affected development, body size, immunity, and adult longevity, but different size metrics showed different relationships with later survival [17]. Therefore, a single morphological proxy cannot be assumed to summarize the entire developmental history. The proposed framework treats cross-stage constraint as multidimensional: evidence must specify what was altered, where it is measured, and whether the adult effect reflects direct persistence, indirect resource limitation, or a later compensatory response.

Compensatory growth and repair

Persistence of an earlier constraint does not imply that development is passive. Holometabolous insects possess substantial capacity to repair damage, modify growth, and redistribute resources before adult emergence. Compensation is therefore central to interpreting apparent decoupling because it can reduce visible differences without restoring the original developmental state.

Imaginal-disc regeneration in Drosophila demonstrates that repair can recruit programs distinct from ordinary development. Single-cell and functional analyses identified Ets21C as a regulator that sustains a regeneration-specific blastema program while being dispensable for normal development [18]. This provides direct evidence that developmental systems can enter a repair state with transcriptional logic that is not merely accelerated baseline growth. It does not establish that every form of environmental compensation uses the same mechanism, but it shows why compensation should be treated as an active biological process rather than as simple return to a pre-existing trajectory.

Compensation can also be incomplete and function-specific. In the butterfly Speyeria mormonia, larval food restriction produced smaller adults, did not trigger increased adult feeding, and reduced flight metabolic capacity, while lifespan was unchanged and fecundity was comparatively buffered [19]. The pattern is inconsistent with a simple continuum from “damaged” to “fully recovered.” Different adult functions can occupy different positions after the same juvenile stress.

A related pattern occurs in female Aedes albopictus. Larval nutrient deprivation prolonged development, permitting eventual emergence, yet adult metabolic profiles remained altered [20]. Developmental delay can therefore compensate one problem—insufficient resource acquisition for metamorphosis—while preserving a metabolic signature of the earlier environment. Importantly, persistent difference is not synonymous with hidden damage; altered adult metabolism may have neutral, beneficial, or detrimental consequences depending on context.

Repair itself may require cross-tissue coordination. In Drosophila, neuronal tachykinin-like receptor signaling affected repair of damaged wing imaginal discs through kynurenine-related metabolism, even though the receptor manipulation did not disrupt normal development [21]. This injury-specific dependence shows that compensation can create systemic relationships not required during unperturbed growth. The broader inference is limited but important: a compensated adult phenotype may embody additional developmental work, altered resource allocation, or repair-specific signaling that is invisible if only the final structure is measured.

The proposed synthesis therefore separates compensation from erasure. Erasure implies that a relevant earlier state no longer constrains the adult condition; compensation implies that the organism reaches a partially or apparently normalized endpoint despite that earlier constraint. The distinction predicts that compensated phenotypes should often become distinguishable when multiple adult traits, energetic demands, or challenges are measured rather than a single endpoint. Whether such differences constitute costs, neutral alternative states, or context-dependent benefits is a separate empirical question.

Hidden costs of compensation

Compensation complicates interpretation because a phenotype can approach an apparently normal adult endpoint while differences remain in functions not captured by that endpoint. Nutritional history in Aedes aegypti, for example, altered combinations of adult fecundity, survival, immune-gene expression, and dengue-virus susceptibility depending on whether nutritional stress occurred during larval or adult life [22]. These outcomes do not show that a visually normal mosquito contains a universal latent deficit, nor do they establish transmission consequences in nature. They demonstrate instead that developmental history can remain detectable in functionally distinct adult systems after metamorphosis.

The magnitude and direction of such differences are not taxonomically fixed. Chronic larval predator exposure produced long-lasting physiological and life-history effects in Anopheles coluzzii and Anopheles gambiae, but

the responses differed between the two closely related species [23]. Compensation should therefore not be represented as a species-independent reserve that simply repairs a standard amount of developmental damage. The organismal route from juvenile stress to adult phenotype depends on the biological architecture available to a particular taxon and on which traits are measured.

Sex can impose another boundary. A meta-analysis spanning 85 insect species found that females were, on average, more sensitive than males to early food stress, with much of that pattern associated with larger female body size; consistent female-biased sensitivity was not evident in species lacking marked sexual size dimorphism [24]. Thus, developmental compensation may conceal unequal vulnerability among individuals even when population means suggest substantial recovery.

The proposed synthesis defines a hidden cost of compensation narrowly: an adult difference that remains consequential for another measured function after one focal phenotype has been partially normalized. It does not treat every persistent difference as a cost. Distinct larval perturbations have produced adult metabolic signatures in experimentally studied Diptera [13, 14], but whether those signatures are detrimental, neutral, or beneficial depends on subsequent demands. A cost is therefore demonstrated only when an independently relevant endpoint is impaired or when additional resources, time, or repair processes required for compensation are themselves measured.

This distinction also prevents three common interpretive errors: equating adult resemblance with complete reset, equating physiological difference with damage, and extrapolating a laboratory carry-over effect to population performance. Because these errors arise from different evidence patterns, a reconciliation matrix is more informative than a linear summary. The table below separates the observable adult state from the inference that can legitimately be drawn from it.

Table 1. Resolving apparently recovered adult phenotypes when compensation and cross-stage constraint remain biologically distinct

Biological setting	Evidence · Permitted interpretation	Principal uncertainty or boundary condition	Research or test · Evidential standing
Endpoint normalization with unresolved state history			
Any holometabolous system in which only one adult trait is measured	Adult focal trait approaches control values after juvenile perturbation The measured endpoint has converged; developmental erasure is not established	Unmeasured metabolic, immune, neural, structural, or reproductive differences may or may not remain	Measure orthogonal adult traits and relevant developmental intermediates Proposed synthesis
Trait-selective compensation			
Nutritional and energetic carry-over systems	One adult function is buffered while another remains altered Compensation is function-specific rather than whole-organism restoration	Different traits may have distinct developmental determination and energetic priority	Jointly quantify structure, reserves, performance, survival, and reproduction Evidence-supported pattern; classification proposed
Compensated development with retained physiological signature			
Development prolonged or growth reorganized before emergence	Emergence or adult formation is achieved, but metabolic or physiological state differs Successful completion of metamorphosis can coexist with persistent state differences	Persistent difference is not necessarily detrimental	Test adult consequences under standardized and challenged conditions Evidence-supported pattern; interpretation proposed
Context-revealed liability			
Adult challenge exposes a difference not evident under benign conditions	Stress, infection, flight demand, or resource limitation separates previously similar adults Earlier-state consequences may be conditional on later demand	Challenge response can reflect adult plasticity rather than juvenile history alone	Use factorial juvenile-history × adult-environment designs Proposed synthesis
Taxon- or sex-contingent compensation			
Closely related species or sexually dimorphic populations	Similar juvenile exposure generates different adult effects among taxa or sexes Compensation cannot be assigned a universal magnitude or direction	Exposure dose, body size, ecology, and developmental timing may differ simultaneously	Replicate across sex, species, developmental stage, and ecological context

			Evidence-supported boundary; classification proposed
Apparent hidden cost without demonstrated impairment			
Any system showing an adult molecular or metabolic difference	Adult state differs from control but performance consequence is unknown A persistent difference is established; a cost is not	Directional fitness or functional consequence remains unmeasured	Avoid cost terminology until an independent adverse endpoint is demonstrated Proposed inference rule

The same logic can be visualized across biological scales rather than as a single juvenile-to-adult arrow. **Figure 1** is needed because compensation can simultaneously normalize one adult endpoint, preserve another difference, and encounter taxonomic or environmental boundaries that cannot be represented faithfully in a simple developmental pipeline.

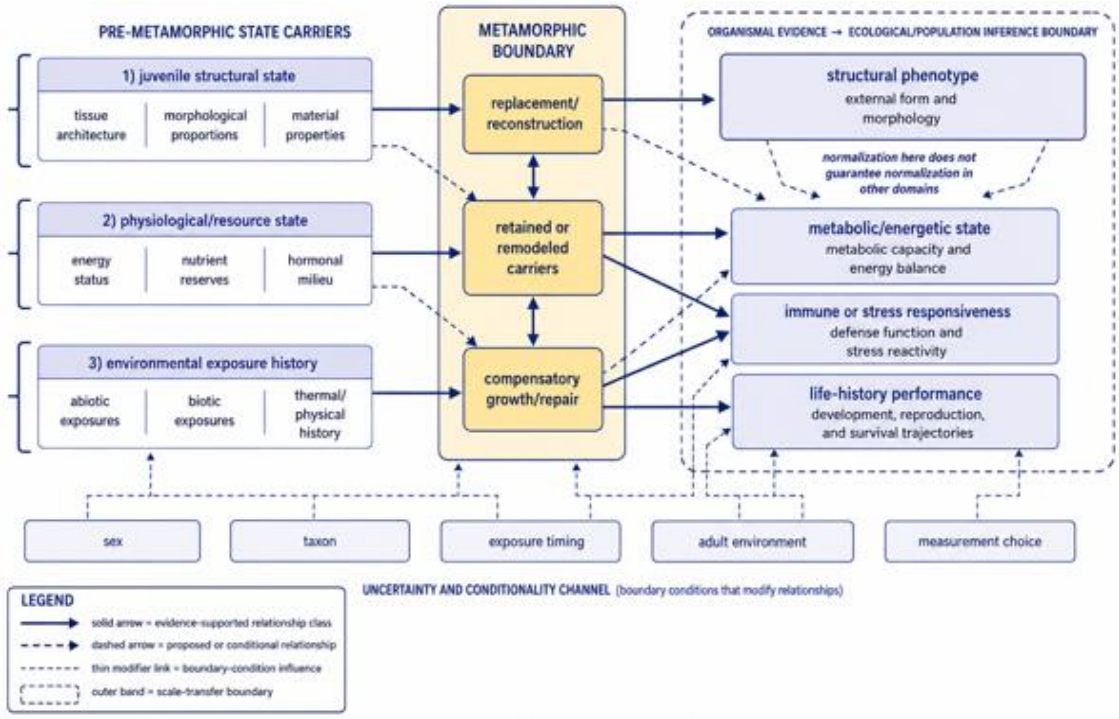


Figure 1. Adult normalization can conceal distinct cross-stage constraints without proving developmental erasure

Adult expression of earlier states

Adult expression of developmental history can occur even when the intervening metamorphic transformation is extensive. A manipulative field experiment with the Colorado potato beetle, Leptinotarsa decemlineata, showed that heat-wave effects depended strongly on ontogenetic timing and host-plant context: neonatal exposure altered juvenile performance and later adult survival, whereas comparable later-stage exposures produced weaker or absent carry-over, and herbivory and crop yield did not mirror the organismal effects [25]. This field evidence is important precisely because it establishes both persistence and its limits.

Adult expression also need not resemble the juvenile perturbation. Larval microbial and thermal environments can later appear as adult metabolic or behavioral differences [13, 14]. Likewise, selected neural or cellular components can cross metamorphosis through persistence or remodeling rather than straightforward preservation of their larval functional state [8, 12]. The proposed synthesis therefore distinguishes carrier persistence from phenotypic recurrence. An earlier state may influence adulthood even when the measured adult phenotype differs qualitatively from the juvenile phenotype that initiated the trajectory.

Adult expression should consequently be described at the level actually measured: structural, metabolic, neural, immune, reproductive, behavioral, or survival-related. Evidence at one level cannot establish continuity at another.

Limits of metamorphic decoupling

Metamorphic decoupling is neither complete nor absent. Transcriptomic, physiological, and quantitative-genetic studies demonstrate substantial stage differentiation while simultaneously leaving room for shared mechanisms and trait-specific constraints [3–5]. The concept therefore loses precision if "decoupling" is used to mean that larval and adult biology are globally independent.

Null outcomes are equally important. Larval diet can affect reproduction without changing survival, and morphometric proxies can disagree in their association with adult longevity [16]. Measurement discordance in mosquitoes further shows that conclusions about persistence can depend on which adult proxy is selected [17]. Such results argue against searching for a single universal index of cross-stage constraint.

Boundary conditions are also biological rather than merely statistical. Predator-induced carry-over differs between related mosquito species [23], sensitivity to early food stress varies with sex and sexual size dimorphism [24], and heat-wave carry-over in the field depends on exposure stage and host-plant environment [25]. Laboratory findings should therefore be transferred to field performance only when the relevant ecological demands are measured rather than assumed.

Finally, absence of an observed adult difference cannot by itself establish reset, because an earlier state may have been compensated, transformed, or expressed in an unmeasured domain. Conversely, detecting an adult difference does not identify its mechanism. The theoretical distinction is therefore evidential: decoupling concerns how far biological state dependence is reduced across metamorphosis, whereas persistence concerns which specific information, material, constraint, or consequence remains detectable and by what route.

Testable predictions

The proposed selective-state transformation synthesis generates five predictions.

First, biological components that are physically replaced or reconstructed should, other factors being equal, show greater potential for functional resetting than components whose cells, structures, or molecular states persist. Testing this prediction requires longitudinal identification of the relevant carrier rather than comparison of larval and adult endpoints alone.

Second, compensation should increase discordance among adult traits when a juvenile constraint cannot be fully removed. Experiments should therefore measure development time, structural dimensions, energetic reserves, locomotor or reproductive performance, and stress responses together rather than treating one normalized endpoint as evidence of recovery.

Third, adult similarity in a focal trait should not reliably predict absence of developmental-history effects elsewhere. This prediction is falsifiable through prespecified multivariate adult phenotyping; it should be rejected where independently measured systems converge consistently after juvenile perturbation.

Fourth, cross-stage effects should vary systematically with exposure timing, sex, taxon, and adult environment. Factorial experiments that manipulate these dimensions can determine whether apparent heterogeneity reflects genuine biological interaction or merely differences in stress intensity and measurement.

Fifth, strong claims of developmental erasure should require direct evidence that relevant earlier-state information is no longer carried forward. Adult convergence alone is insufficient when the underlying carrier, resource state, or compensatory trajectory remains unmeasured. Conversely, evidence of persistent difference should not be labelled a cost unless functional impairment is independently demonstrated.

Conclusion

Complete metamorphosis creates exceptional developmental discontinuity, but discontinuity is not equivalent to universal erasure. The synthesis developed here proposes that earlier biological states can be replaced, remodeled, propagated indirectly, or compensated, with different outcomes occurring simultaneously within one organism. This view reconciles strong evidence for stage decoupling with equally credible evidence for cross-stage constraint without weakening either concept.

Its central implication is methodological as much as theoretical: inference should follow the biological carrier and the measured endpoint. Adult resemblance does not prove reset, adult difference does not prove direct persistence, and compensation does not automatically imply hidden cost. Resolving these alternatives requires multilevel, longitudinal, taxonomically explicit experiments that separate developmental state, metamorphic transformation, adult expression, and ecological consequence.

Acknowledgments: None

Conflict of Interest: None

Financial Support: None

Ethics Statement: None

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