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Larval History Can Persist Across Complete Metamorphosis Through Mechanisms of Physiological Carry-Over and Developmental Constraint

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ABSTRACT

Complete metamorphosis reorganizes insect anatomy so extensively that larval and adult phenotypes are often treated as developmentally decoupled. This interpretation is useful but incomplete. Adult construction still depends on resources, physiological states, surviving or newly generated tissues, endocrine decisions, and microbial interactions established before or during metamorphosis. We argue that larval history can therefore influence adults without requiring simple preservation of larval structures or persistent exposure to the original environmental factor. Evidence from holometabolous insects indicates several non-equivalent forms of continuity: transfer of energetic reserves, persistence of metabolic or transcriptional states, selective cellular survival and tissue remodeling, endocrine gating of developmental commitments, and taxon-specific microbial or symbiont transmission. Conversely, extensive histolysis, regulatory reprogramming, microbiota turnover, and de novo tissue construction can weaken or erase larval effects. We propose that carry-over across complete metamorphosis should be interpreted as bounded developmental dependence rather than as either developmental continuity or complete reset. This perspective separates persistence of an initiating substrate from persistence of its downstream consequences and generates experimentally distinguishable mechanisms for later testing.

Keywords: Holometaboly, Carry-over effects, Developmental constraint, Ecdysone, Transstadial microbiome, Insect metamorphosis

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Introduction

Complete metamorphosis creates one of the sharpest ontogenetic transitions in animal development. During the pupal interval, many larval structures undergo histolysis or substantial reorganization while imaginal tissues differentiate into adult organs, producing ecological and morphological separation between juvenile and adult stages [1]. This transformation has encouraged a powerful but potentially misleading shorthand: larval conditions act before metamorphosis, adult traits act after it, and the pupal transition substantially resets the organism.

A carry-over effect requires a narrower inference. It occurs when conditions experienced during an earlier stage alter phenotype or performance expressed later, but the causal pathway need not involve uninterrupted persistence of the original exposure, molecule, tissue, or phenotype [2]. Accordingly, a larval-history effect may survive metamorphosis because material is physically retained, because an induced physiological state changes the

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resources entering metamorphosis, because developmental timing constrains later construction, or because a transient perturbation changes subsequent regulatory trajectories. These mechanisms differ experimentally and should not be conflated.

Direct evidence already shows why the distinction matters. Manipulating the bacterial environment of *Aedes aegypti* larvae can alter developmental and adult traits, including body size and components of adult immune or vector-related phenotype, while other adult outcomes can remain unchanged [3]. Such results establish cross-stage dependence under the tested conditions; they do not establish a universal microbial memory or show that the same bacteria necessarily persist into adulthood.

This perspective develops a mechanistic alternative to the binary choice between continuity and reset. We treat complete metamorphosis as an active transformation through which some components of larval history are transmitted, some are converted into different state variables, and others are erased. The central proposed distinction is between substrate continuity, in which a material, organism, cell, or state itself survives the transition, and effect continuity, in which the initiating factor disappears but its downstream consequences remain detectable. The following sections examine how reserves, molecular states, tissue architecture, endocrine gating, and microbial associations can generate or constrain these forms of carry-over.

The metamorphic reset assumption

The reset assumption is most defensible at the level of structures that are dismantled and reconstructed. It becomes weaker when generalized to organismal state. Metamorphosis consumes resources accumulated during larval growth, operates through regulatory systems established before pupation, and builds adult phenotype within developmental conditions partly inherited from the juvenile stage. Thus, anatomical discontinuity does not imply physiological independence.

Experiments with gnotobiotic mosquitoes illustrate this distinction. Different bacterial conditions during larval development induced metabolic and transcriptional differences, and some consequences extended into adults even though metamorphosis intervened between the manipulated larval environment and the adult phenotype [4]. The admissible conclusion is that larval microbial conditions altered developmental state sufficiently to affect later biology. It is stronger than a simple association but does not require persistence of the larval bacterial community itself.

Indeed, microbial turnover supplies an important counterexample to literal continuity. Comparative evidence across insects undergoing complete metamorphosis indicates that the gut transition can produce substantial restructuring of microbial communities [5]. A strong adult carry-over phenotype therefore cannot automatically be interpreted as evidence that its initiating microbiota crossed the pupal stage intact.

Trait-level decoupling is equally important. In *Aedes albopictus*, larval nutrition and temperature affected developmental variables, body-size measures, adult longevity, and immune-gene expression in non-equivalent ways; commonly used body-size measurements themselves did not provide an interchangeable representation of developmental history [6]. Carry-over is consequently better treated as a mapping from a prior state to particular later traits than as a property of the whole adult.

An even clearer separation between substrate and effect comes from larval infection. In *Tribolium castaneum*, consequences of larval parasite exposure remained detectable in adult immune expression even though the parasite itself was cleared during metamorphosis [7]. This is effect continuity without persistence of the original biological agent. It also shows why detecting an adult molecular signature cannot, by itself, identify which component crossed the metamorphic boundary.

We therefore reject neither developmental decoupling nor metamorphic reorganization. Instead, we propose that the "reset" should be decomposed into separate questions: what is destroyed, what survives, what is rebuilt from inherited resources, what regulatory states are re-established, and which earlier perturbations alter the conditions under which rebuilding occurs. A mechanistic account of carry-over begins only after those questions are separated.

Persisting reserves and molecular states

The most direct route by which larval history can influence an adult is through resources accumulated before feeding ceases. Metamorphosis is energetically expensive, and teneral adults emerge with reserves whose quantity and composition reflect acquisition and allocation during juvenile development. In male *Aedes aegypti*, larval diet and temperature affected developmental performance and teneral energetic reserves, with corresponding

differences in later longevity and thermal tolerance under the tested rearing regimes [8]. Such evidence supports resource-mediated carry-over while remaining bounded to the environmental combinations, sex, and mosquito system examined.

Resource continuity is not merely a question of total mass. Developmental diet can leave molecular signatures after eclosion. In *Drosophila melanogaster*, global expression profiles of young adults were influenced more strongly by developmental diet than by the adult diet imposed over the early post-eclosion interval, with sex-dependent features of the response [9]. This demonstrates persistence of developmental influence on the adult transcriptome, not permanent transcriptional memory. Adult feeding, aging, reproductive state, and other environmental inputs can subsequently modify the expression landscape.

Larval crowding provides another example in which the later phenotype cannot be reduced to a retained quantity of stored nutrient. Adult *Drosophila simulans* developing under different larval densities showed expression differences involving energy metabolism, locomotor processes, and reproductive investment [10]. Because crowding simultaneously changes nutrient availability, competition, developmental time, waste exposure, and social environment, the adult transcriptome identifies a carry-over state but does not isolate a single causal mediator.

Stress can also alter the allocation landscape entering metamorphosis. Chronic exposure to predator cues during larval development produced adult effects in two *Anopheles* species that varied with species, sex, and measured trait, including differences in size, reserves, oxidative physiology, and reproductive characteristics [11]. The heterogeneity is mechanistically informative: a common larval challenge need not create a common adult syndrome.

Similarly, larval food restriction in a nectar-feeding butterfly reduced adult body size and mass-adjusted flight metabolic capacity without producing an equivalent reduction in every adult life-history outcome; longevity, for example, did not simply mirror the energetic effects [12]. Developmental resource limitation can therefore constrain an adult performance component while other traits are buffered, compensated, or governed by different limiting processes.

These observations motivate a proposed distinction between reserve carry-over and state carry-over. Reserve carry-over involves material quantities available to metamorphosis and the teneral adult, including lipid, carbohydrate, protein, or other stored resources. State carry-over describes an induced molecular or physiological configuration—such as transcriptional, metabolic, immune, or redox state—that changes later developmental operation. The two may interact, but neither should be inferred from the other. A small adult may reflect developmental resource limitation, endocrine timing, tissue-specific growth regulation, or combinations of these mechanisms; conversely, substantial molecular carry-over can exist without a conspicuous change in body size.

Tissue remodeling and developmental constraint

Complete metamorphosis combines destruction, selective persistence, remodeling, and de novo construction rather than applying a single developmental operation to every tissue. Neural circuits provide a particularly clear example. Analysis of the *Drosophila* mushroom body shows that some larval neurons and circuit elements can survive metamorphosis and become extensively repurposed within the adult network while other components are eliminated [13]. Cellular continuity therefore need not imply functional continuity: the same cell can occupy a different circuit role after remodeling.

Programmed cell death is the complementary process. Widespread elimination of larval cells contributes to metamorphic reconstruction of the insect nervous system and other tissues, under strong developmental and hormonal regulation [14]. Any proposed carry-over mechanism based on persistent cells must consequently demonstrate that the relevant cell population escapes elimination or influences a surviving lineage before disappearing.

Metabolic tissues similarly expose the inadequacy of a simple persistence-versus-replacement dichotomy. In *Drosophila*, metamorphic remodeling of larval fat body and mobilization of stored lipid are regulated processes that can be experimentally shifted, including through signaling pathways interacting with the ecdysone-driven transition [15]. Larval fat body can thus contribute resources during reconstruction even when adult adipose organization is not merely the unchanged larval organ.

Developmental constraint can also arise in tissues destined to form adult structures. Ecdysone regulates final wing-disc growth through TORC1-dependent control in *Drosophila*, linking systemic developmental signaling to tissue-specific growth termination [16]. The implication for carry-over is bounded but important: an adult structure can

encode earlier developmental conditions because its precursor tissue experienced a constrained growth trajectory before metamorphosis, not because the final adult tissue literally preserved the larval phenotype.

Imaginal tissues additionally possess temporally restricted capacities for repair. Ecdysone-dependent maturation of the *Drosophila* wing-disc epithelial barrier contributes to closure of the regeneration checkpoint as development advances [17]. Earlier damage and developmental delay can therefore interact with a changing window of tissue competence. This establishes a mechanism by which timing itself becomes a constraint on subsequent construction.

Adult morphogenesis also depends on cellular capacities that operate during pupation. Disrupting mitochondrial function in *Tribolium castaneum* compromises wing development during metamorphosis [18]. Such evidence does not show that ordinary variation in larval mitochondrial state necessarily predicts adult wings, but it establishes that metamorphic construction is conditional on physiological machinery whose impairment can limit the adult outcome.

At the regulatory level, metamorphosis involves extensive reconfiguration of transcriptional programs and chromatin accessibility [19]. This makes indiscriminate claims of persistent "epigenetic memory" particularly problematic. A stage-specific chromatin or transcriptional difference is not proof that the same regulatory state survives metamorphosis. Demonstrating persistence requires lineage- or state-resolved measurements spanning the transition and excluding reconstruction of a similar adult state by a different mechanism.

Finally, recent developmental work demonstrates that adult *Drosophila* adipose tissue is generated de novo during metamorphosis, while larval adipose cells can remain transiently present around adult emergence [20]. Replacement and persistence can therefore coexist within one physiological system. The relevant mechanistic question is not simply whether a tissue survives, but whether larval cells, stored substrates, signaling states, or constraints on precursor development influence the adult tissue produced.

We consequently define developmental constraint here as a bounded dependence of adult construction on states or commitments established earlier in ontogeny. It does not imply irreversibility, maladaptation, or evolutionary constraint in the population-genetic sense. A developmental history constrains an adult outcome only when later development operates within a state space altered by that earlier history.

Endocrine carry-over

Endocrine regulation provides a mechanism through which larval state can influence adult construction without requiring the persistence of a larval hormone concentration into adulthood. Ecdysteroid production and secretion coordinate the transition into metamorphosis and interact with other developmental signals controlling when tissues leave larval growth and enter pupal differentiation [21]. The relevant carry-over variable is therefore often the timing, threshold, or downstream consequence of endocrine activation rather than persistent hormone molecules.

Neuropeptides and peptide hormones connect nutritional and physiological information to the prothoracic gland and the control of ecdysone production in *Drosophila* [22]. This architecture creates a plausible conversion step: larval nutritional or physiological state can be translated into altered endocrine timing, which then changes the developmental context encountered by multiple tissues.

Specific experimental pathways illustrate the principle. Neuropeptide F signaling can modify developmental timing and body-size regulation through interactions with the endocrine growth axis [23]. Likewise, EGFR signaling in the prothoracic gland contributes to regulation of ecdysone biosynthesis, developmental timing, and final body size [24]. Neither pathway should be treated as a universal transstadial memory mechanism; rather, each shows how information present during larval growth can alter the schedule within which adult precursors develop.

Nutritional history can also become developmentally gated. Nutrient-dependent endocycling in steroidogenic tissue contributes to the timing of metamorphic commitment in *Drosophila*, linking nutritional state with the endocrine machinery that governs developmental transition [25]. Once a commitment threshold has been passed, continued development can become less dependent on subsequent nutrient availability, a principle captured by work on critical weight and metamorphic commitment in holometabolous insects [26]. This introduces temporal asymmetry: the same environmental perturbation can have different consequences depending on whether it occurs before or after an endocrine commitment point.

Endocrine carry-over must therefore be distinguished from endocrine mediation. Demonstrating that ecdysone controls a tissue process establishes endocrine mediation. Demonstrating carry-over additionally requires

evidence that an earlier larval condition altered the endocrine state or its downstream developmental consequences and that this difference accounts for later phenotype. The proposed mechanism has at least four separable stages: larval-state loading, endocrine gating, tissue-specific response, and adult expression. Failure to measure the intermediate stages leaves alternative mechanisms—including direct resource limitation or tissue-autonomous effects—open.

Because these endocrine scenarios permit different strengths of inference, a comparative adjudication is more informative than treating every hormone-associated adult phenotype as equivalent.

Table 1. Distinguishing endocrine mediation from genuine developmental carry-over across metamorphosis

Before or at commitment		**Meaning after the gate**		**Discrimination**	
Metamorphic gate state	**Biological basis**	**Carry-over inference**	**Competing route**	**Temporal or intervention test**	**Synthesis standing**
Larval-state loading					
Larval-state loading	Nutritional, metabolic, stress, or other larval state changes before endocrine commitment	Earlier condition could alter the endocrine context entering metamorphosis	Resource limitation may act directly without endocrine mediation	Manipulate larval state while measuring endocrine output and downstream tissue response	Proposed mechanistic category
Endocrine commitment					
Endocrine-gate shift	Altered timing or production of metamorphic endocrine signal	Developmental history is converted into a changed transition schedule	Hormonal change may correlate with, rather than mediate, the initiating condition	Restore endocrine timing independently of the larval exposure and test phenotypic rescue	Evidence-supported mechanism; carry-over inference requires exposure linkage
Commitment-threshold crossing	Nutrient-responsive developmental state becomes less sensitive to later nutrition after commitment	Timing of exposure can determine whether larval conditions remain consequential	Threshold position and physiological meaning may differ among taxa	Apply equivalent perturbations before and after the measured commitment point	Evidence-supported principle with taxonomic limits
Tissue response and remodeling					
Tissue-response constraint	Imaginal or remodeling tissue responds to endocrine state through growth, maturation, or competence pathways	Adult morphology can encode earlier endocrine timing through precursor-tissue development	Tissue-autonomous nutrient or damage signaling may produce similar phenotype	Alter tissue response while holding systemic endocrine conditions constant	Evidence-supported developmental mechanism
Remodeling-state conversion	Endocrine transition mobilizes or reorganizes larval tissues and stored resources	Larval material can influence adult construction after being transformed rather than preserved intact	Material contribution does not establish persistence of larval tissue identity	Track tissue fate and resource flux simultaneously across pupation	Proposed carry-over interpretation grounded in remodeling evidence
Post-gate decoupling					
Post-gate decoupling	Later developmental program proceeds despite removal or reversal of the original larval condition	Metamorphosis can attenuate an earlier exposure while retaining selected downstream consequences	Apparent absence of carry-over may be trait-specific rather than organism-wide	Measure multiple downstream traits across the same staged perturbation	Proposed boundary state

The corresponding visual architecture separates what is observed from what must be inferred, because an endocrine association becomes evidence of carry-over only when the initiating larval state, the developmental gate, and the later consequence can be linked without collapsing alternative routes.

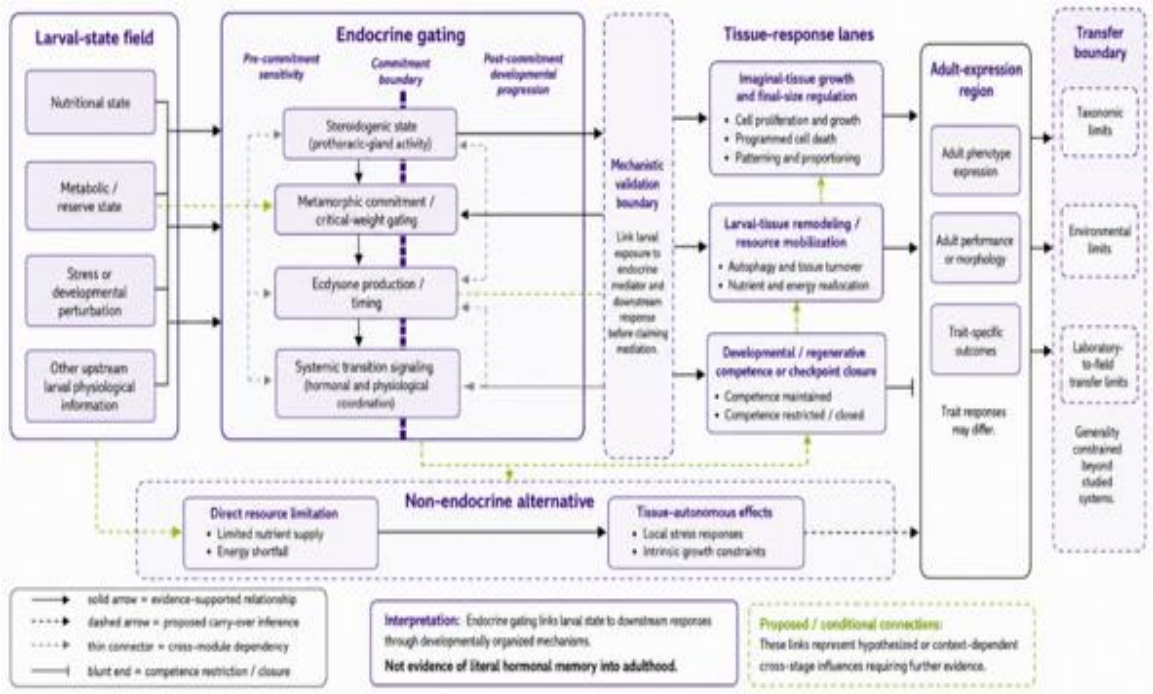


Figure 1. Endocrine gating converts larval state into bounded developmental dependence rather than hormonal memory

Microbiome and symbiont continuity

Microbial continuity across metamorphosis represents a particularly stringent test of the distinction between persistence and reconstruction because gut remodeling can directly disrupt the habitat occupied by larval microorganisms. The relevant mechanisms range from complete or partial microbial loss to persistence in protected locations, reacquisition from the environment, and specialized host structures that maintain obligate partners.

Microbial associates can nevertheless influence development before this transition occurs. In mosquito larvae, a prolific gut-associated fungus can alter developmental and later fitness-related characteristics, demonstrating that a larval microbial interaction can change the physiological conditions from which metamorphosis proceeds [27]. An adult consequence arising from such exposure should not automatically be equated with transstadial persistence of the microorganism; host-mediated metabolic or developmental effects provide an alternative route. Studies following bacterial communities across stages show that some taxa can be detected on both sides of metamorphosis while overall community composition changes. Work in *Anopheles albimanus* supports transstadial occurrence for portions of the gut bacterial assemblage but also documents substantial developmental restructuring [28]. Detection of the same bacterial taxon in larvae and adults is therefore compatible with continuity, but without strain-resolved tracking it may not distinguish direct survival from environmental reacquisition or replacement by closely related organisms.

Environmental contribution further complicates inference. Comparisons among *Aedes albopictus* breeding water, larvae, and adults reveal pronounced stage-associated microbial structuring alongside connections with the larval habitat [29]. The adult microbiome is consequently not interpretable as a simple archived record of the larval gut. Stage-specific filtering, metamorphic turnover, environmental exposure, and host physiology can all contribute to the adult assemblage.

Some insect–symbiont systems have evolved—or, more conservatively at the mechanistic level, possess—specialized anatomical arrangements that directly protect continuity. In *Lagria* beetles, host morphological adaptations facilitate maintenance and transmission of ectosymbiotic bacteria during metamorphosis [30]. This provides unusually strong evidence for substrate continuity because the mechanism addresses the physical problem created by tissue reorganization. It also shows why conclusions from such systems should not be generalized to insects lacking comparable symbiont-bearing structures.

Conversely, adult microbial function does not prove larval-to-adult transmission. Experimental work in *Aedes aegypti* shows that the adult gut microbiota can be important for normal fecundity, longevity, and vector

competence [31]. Those functional dependencies establish the importance of adult microbial state, but the origin of that state remains a separate question.

The microbiome literature therefore requires at least three mechanistically distinct interpretations. Microbial substrate continuity occurs when the same microbial lineage is retained across metamorphosis. Host-state carry-over occurs when larval microbes alter host physiology and the host consequence persists even if the microbes do not. Microbiome reconstruction occurs when metamorphosis substantially removes the larval community and the adult assemblage is generated through stage-specific filtering or reacquisition. These categories are proposed as inference tools rather than universal biological classes.

Crucially, the three routes can coexist. A minority symbiont may be actively retained while most gut taxa turn over; larval microbial metabolism may alter stored resources even when the responsible organisms disappear; and adults may subsequently acquire microorganisms whose functional effects are independent of larval exposure. Accordingly, sequencing larvae and adults at isolated time points is rarely sufficient to establish mechanism. Strong tests require lineage-resolved microbial tracking across pupation, simultaneous measurement of host physiological state, and experimental separation of microbial persistence from reacquisition. Complete metamorphosis can thus interrupt microbial continuity while still permitting the biological consequences of larval microbial history to cross the developmental boundary.

Adult phenotypic expression

Adult phenotype is the point at which larval carry-over becomes observable, but observation alone does not identify the mechanism that crossed metamorphosis. In *Drosophila melanogaster*, larval diet altered adult reproductive output without producing a corresponding survival effect, and these nutritional effects remained analytically distinguishable from consequences of injury and infection [32]. Adult expression is therefore endpoint-specific rather than a unitary property of the organism.

This pattern is consistent with experiments in which developmental food stress affected adult body size and flight metabolic capacity without an equivalent effect on longevity [12]. Similarly, adult physiology can depend strongly on conditions encountered after eclosion: *Aedes aegypti* requires an adult gut microbiota for several normal life-history and vector-related functions [31]. Consequently, present adult state may preserve, amplify, compensate for, mask, or override developmental differences.

We propose three observational categories. Persistent expression occurs when larval treatments remain associated with an adult trait. Apparent convergence occurs when treatment groups become indistinguishable for the measured adult endpoint despite earlier differences. Conditional expression describes the hypothesis that apparently converged adults may diverge when challenged by later energetic, immune, reproductive, or environmental demand. The third category is explicitly proposed and requires experimental testing. None of these observational patterns alone establishes persistence of a particular substrate, causal mediator, fitness consequence, or adaptive value.

Mechanistic carry-over model

We propose a metamorphic carry-over state-space model in which larval history enters metamorphosis through partially separable resource, molecular/immune, developmental-endocrine, tissue, and symbiotic states. Metamorphosis then acts through selective retention, destruction, remodeling, resource conversion, endocrine gating, de novo construction, and microbial loss or reacquisition.

These operations generate mechanistically distinct outcomes. Retained-substrate carry-over requires physical continuity, illustrated most clearly when symbionts are actively maintained during metamorphosis [30]. Transformed-state carry-over occurs when the initiating substrate disappears but a downstream host state remains, as demonstrated by adult immune consequences after larval parasite clearance [7]. Gated developmental constraint occurs when earlier state changes the timing or conditions under which later construction proceeds, consistent with nutrient-sensitive control of metamorphic commitment [25]. Reconstructed adult state becomes plausible where the relevant adult structure is built de novo [20] or where microbial turnover is extensive [5].

Two additional states are proposed. Compensated convergence describes disappearance of an initially detectable difference because remodeling, adult resource acquisition, or physiological compensation narrows treatment differences. Latent carry-over describes an earlier difference that is not detectable under baseline adult conditions but becomes expressed under a later demand. Neither state should be inferred from a single adult endpoint.

Because identical adult observations can arise through different metamorphic routes, a state-and-response ledger is needed to distinguish what persisted from what merely remained consequential.

Table 2. State-dependent interpretation of persistence, conversion, reconstruction and apparent convergence across complete metamorphosis

Material continuity		
Metamorphic continuity dimension	**Retained substrate**	**Transformed state**
Cross-stage basis	Cell, reserve, symbiont or other substrate survives transition	Initiating factor disappears after altering host physiology
Adult observation	Adult difference with demonstrable continuity	Adult difference remains
Mechanism permitted	Physical continuity can mediate carry-over	Effect continuity without substrate continuity
Discriminating test	Fate-track substrate and perturb it selectively	Remove initiating factor and track candidate host state
Alternative	Survival alone does not prove phenotypic causation	Another persistent mediator may explain the effect
Standing	Evidence-supported mechanism class	Evidence-supported mechanism class
Gating and reconstruction		
Metamorphic continuity dimension	**Gated constraint**	**Reconstructed state**
Cross-stage basis	Earlier state changes developmental timing or commitment	Adult tissue or community is rebuilt
Adult observation	Adult construction differs	Weak or absent substrate continuity
Mechanism permitted	Larval history altered the developmental conditions of metamorphosis	Adult state need not preserve larval material
Discriminating test	Stage perturbation around commitment plus mediator manipulation	Combine lineage tracking with state measurements
Alternative	Direct resource limitation may mimic gating	Reconstructed state may still inherit resources or constraints
Standing	Evidence-supported mechanism class; integration proposed	Evidence-supported reconstruction; interpretation conditional
Convergence and latent expression		
Metamorphic continuity dimension	**Compensated convergence**	**Latent carry-over**
Cross-stage basis	Earlier difference becomes undetectable by adulthood	Baseline adults converge but later demand reveals divergence
Adult observation	Apparent adult similarity	Demand-dependent adult difference
Mechanism permitted	Reset cannot be distinguished from compensation by endpoint alone	Earlier history may remain functionally concealed
Discriminating test	Repeated measurements across metamorphosis	Pre-specified adult challenge after matched baseline phenotype
Alternative	Low sensitivity or adult environmental override	New adult exposure may itself generate divergence
Standing	Proposed state	Proposed state

A state-space representation further separates adult similarity from mechanistic reset and makes explicit where evidence-supported developmental routes end and proposed interpretation begins.

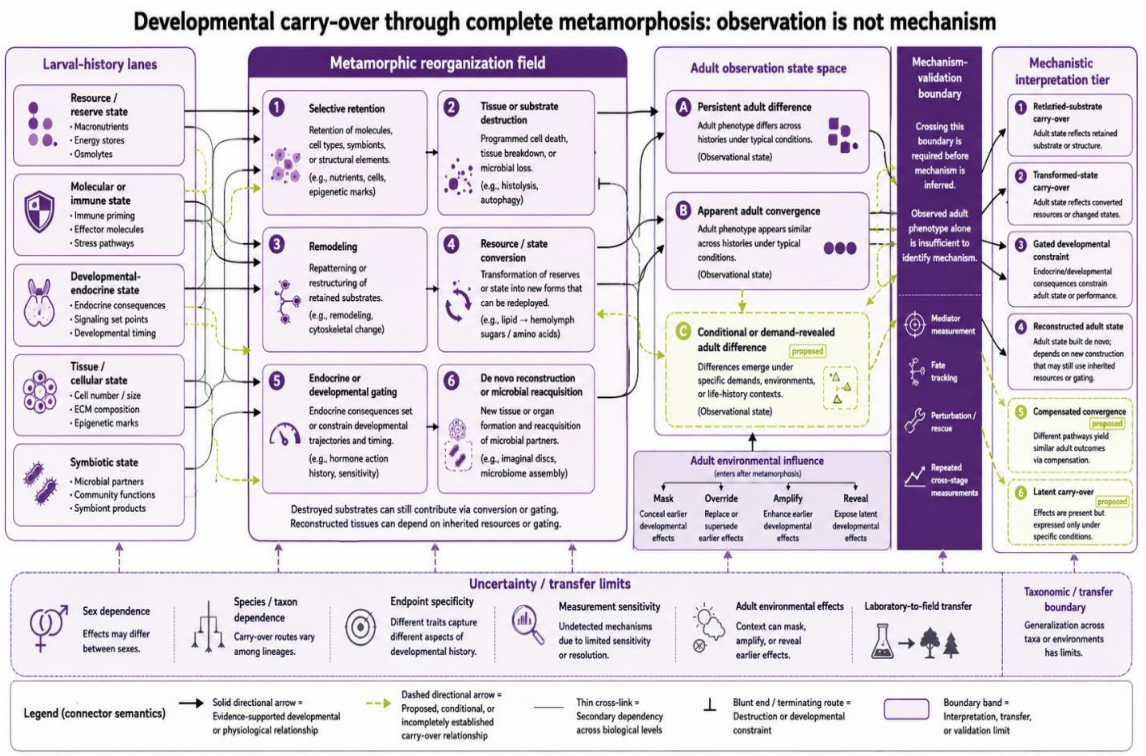


Figure 2. State-space separation of retained history, transformed history, reconstruction and masked carry-over across metamorphosis

Falsifiable predictions

The proposed model generates experimentally separable predictions. First, if adult effects are reserve-mediated, experimentally equalizing the relevant pre-pupal reserve should attenuate the downstream difference; persistence after successful reserve equalization would weaken that mechanism [8]. Second, if developmental gating is causal, equivalent perturbations delivered before and after a measured commitment transition should produce different downstream consequences, and manipulating the candidate endocrine mediator should alter that pattern [25].

Third, substrate continuity and effect continuity predict different fate trajectories. An immune consequence may persist after the initiating parasite has disappeared [7], whereas microbial substrate continuity requires lineage-resolved evidence that the same organism crosses metamorphosis rather than being reacquired [28]. Finally, the proposed latent-carry-over state predicts that treatment groups showing matched baseline adult phenotypes may diverge under a pre-specified adult challenge. Failure of such divergence across appropriately powered, mechanistically relevant challenges would count against that particular hypothesis rather than being reinterpreted post hoc as concealed carry-over.

Results and Discussion

Complete metamorphosis should not be treated as either a universal reset or a transparent bridge between larval and adult phenotype. The evidence supports a more selective interpretation. Some substrates persist, some effects survive after their initiating substrate disappears, and some adult systems are rebuilt while remaining dependent on resources or developmental commitments established earlier.

The proposed state-space model therefore assigns stronger evidential status to demonstrated mediator continuity than to cross-stage association alone. De novo adult adipose construction, for example, establishes tissue reconstruction but does not show that larval resource history is irrelevant to the construction process [20]. Conversely, extensive microbiota turnover limits claims of microbial continuity without excluding host-state effects initiated during larval life [5].

Generality remains constrained. Much mechanistic evidence comes from Drosophila, Aedes, Anopheles and a limited set of beetle and lepidopteran systems. Sex, species, nutritional environment, adult conditions and measured endpoint can alter whether carry-over is visible. Laboratory manipulations additionally isolate

developmental pathways more cleanly than field environments, where adult feeding, microbial acquisition, thermal conditions, infection and reproduction continually modify phenotype.

Future tests should therefore combine repeated measurements across the metamorphic boundary with fate or lineage tracking, crossed larval and adult environments, targeted mediator manipulation, and multiple adult endpoints. Adult body size, transcription, immunity, metabolism, reproduction and survival should not be treated as interchangeable proxies. Nor should developmental plasticity be described as adaptive without direct evidence linking the measured response to fitness under the relevant ecological conditions. Carry-over is most informative when formulated as a mechanism-specific, falsifiable developmental dependency rather than as a retrospective label for any larval–adult association [2].

Conclusion

Complete metamorphosis can profoundly reorganize an insect without making the adult independent of larval history. Persistence may reside in retained substrates, transformed physiological states, developmental commitments, resource constraints or protected symbiotic associations; alternatively, reconstruction and compensation can erase detectable differences. The proposed state-space model separates these routes from the adult patterns they produce and treats apparent convergence as mechanistically ambiguous rather than synonymous with reset. Testing carry-over therefore requires identifying what crosses the metamorphic boundary, what is transformed there, and which adult conditions reveal or override its consequences. This evidence-bounded approach makes developmental continuity experimentally discriminable without assuming universal persistence or mechanistic memory.

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