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Temporal Risk Spreading in Insect Populations Extends Beyond Dormancy Through Multiple Forms of Life-History Bet-Hedging

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ABSTRACT

Environmental uncertainty exposes insect lineages to temporal variation in survival, development, reproduction, and recruitment, yet risk spreading is often discussed primarily through diapause. This emphasis can obscure other life-history routes by which reproductive success may be distributed across times, developmental trajectories, offspring states, and locations. We develop an original non-empirical evolutionary framework in which temporal risk spreading is treated as a property of the distribution of life-history outcomes rather than as a synonym for dormancy. The framework distinguishes dormancy, developmental-duration variation, reproductive scheduling, dispersal, and offspring heterogeneity while preserving the critical difference between environmentally induced variation and evolutionary bet-hedging. We argue that a trait qualifies as a candidate bet-hedging mechanism only when its biological consequences are evaluated across the environmental grain and temporal scale relevant to lineage persistence, with explicit attention to arithmetic versus geometric fitness, cue reliability, trait heritability, plasticity, and among-offspring covariance. The framework therefore does not assume that phenotypic variance, diapause, dispersal, or reproductive diversification is intrinsically adaptive. Instead, it provides a testable architecture for determining when apparently different insect life-history strategies could generate functionally comparable forms of risk spreading under environmental uncertainty.

Keywords: Bet-hedging, Diapause, Phenology, Developmental timing, Oviposition, Dispersal

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Introduction

Insect populations encounter environmental variation at temporal scales extending from hours and days to seasons, years, and generations. Such variation can alter not only mean performance but also the variance of survival and reproduction among individuals carrying the same or similar genotypes. Evolutionary theory increasingly recognizes that this variance can itself be a target of selection rather than statistical noise surrounding an optimal mean phenotype [1]. Consequently, the evolutionary value of a life-history strategy cannot always be inferred from its arithmetic mean performance under a set of environments. Where reproductive success fluctuates among generations, long-term genotype success can depend on geometric mean fitness, creating conditions in which

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reduced variance or diversified phenotypes may be favoured even when they do not maximize expected performance in every generation [2].

This distinction also creates a scale problem. Selection acting on variance among generations can alter the evolutionary consequences of variance-sensitive decisions expressed within generations [3]. More generally, biological responses to environmental fluctuation can occupy a continuum rather than falling cleanly into a single category such as specialization, plasticity, or bet-hedging [4]. Environmental predictability is especially important because plastic responses require informative cues: theoretical work predicts that reduced predictability can weaken the advantage of phenotype–environment matching and favour lower plasticity under some conditions [5]. Thus, phenotypic diversity alone is insufficient evidence for bet-hedging.

In insect biology, diapause provides the most familiar example of temporally redistributing developmental and reproductive opportunity, but focusing on dormancy alone risks narrowing the inferential problem. Development rates vary among individuals and microhabitats; mating and oviposition occur across temporally heterogeneous conditions; dispersal redistributes descendants among spatially asynchronous environments; and parents can generate heterogeneous offspring states. This article therefore proposes that these processes be examined as potentially distinct routes to temporal risk spreading, while reserving the term bet-hedging for cases in which evolutionary criteria are met. The argument is not that all such traits are bet-hedging. Rather, it is that dormancy should be treated as one member of a broader candidate set whose evolutionary interpretation requires explicit tests of environmental uncertainty, fitness variance, covariance among descendants, and alternative explanations such as plasticity, constraint, or simple environmental forcing.

Bet-hedging and environmental uncertainty

Bet-hedging is fundamentally a lineage-level interpretation of phenotypic strategy. Under fluctuating environments, strategies that reduce variance in reproductive success can outperform strategies with higher arithmetic mean success if occasional poor generations disproportionately reduce long-term multiplication [2]. Variance control may therefore involve either relatively invariant phenotypes that limit exposure to extreme failure or production of heterogeneous phenotypes that prevent all descendants from sharing the same fate [1]. These correspond broadly to conservative and diversifying forms of bet-hedging, but the distinction concerns fitness distributions rather than superficial differences in phenotype.

Temporal scale is crucial. A strategy that appears risk-prone within one reproductive episode may coexist with or be modified by selection operating across generations [3]. Likewise, different adaptations to environmental fluctuation can grade into one another as predictability, environmental grain, and the structure of phenotype–environment matching change [4]. This means that the same observable trait cannot be assigned a universal evolutionary label independently of its environmental context.

Plasticity creates the most important alternative explanation. If temperature, photoperiod, host condition, density, or another cue reliably predicts the state that affects future fitness, cue-responsive phenotype production may increase environmental matching without constituting diversifying bet-hedging. Conversely, declining predictability can reduce the evolutionary benefit of plasticity [5]. The proposed framework therefore separates three questions: whether a trait varies, whether that variation tracks information about the environment, and whether residual or deliberately diversified variation reduces lineage-level fitness variance. Only the third question, evaluated against appropriate alternatives and across relevant generations, supports a bet-hedging interpretation.

This distinction also prevents retrospective adaptive storytelling. Developmental noise, physiological limits, imperfect cue perception, maternal condition, genotype-by-environment interaction, and microhabitat variation can all generate phenotypic variance. Demonstrating variance is therefore an observation; demonstrating risk spreading requires linking that variance to differential environmental outcomes; and demonstrating evolutionary bet-hedging further requires evidence that the resulting distribution of outcomes improves long-term lineage performance under uncertainty. These levels of inference remain separate throughout the framework.

Dormancy as one risk-spreading mechanism

Diapause is an obvious starting point because it can remove insects from periods unsuitable for active development or reproduction. Across insects, however, diapause strategies vary in their reaction norms, and geographic patterns of diapause incidence and timing provide evidence that phenotypic plasticity and bet-hedging-like diversification may respond differently to environmental predictability [6]. Temperature change further complicates

interpretation because altered thermal regimes can act directly through physiological stress and development while also producing indirect consequences through phenological mismatch, host quality, and natural enemies [7]. Dormancy therefore spreads risk only within a larger ecological system of seasonal opportunities and hazards.

Other compensatory responses reinforce this point. Tropical and widespread butterflies can differ in behavioural and morphological thermoregulation, allowing activity under variable thermal environments without invoking dormancy [8]. Seasonal timing itself can evolve in relation to cue regimes. In the wall brown butterfly *Lasiommata megera*, northern range-margin populations repeatedly evolved diapause induction responses to longer local daylengths during northward range expansion [9]. Yet experiments on several of the same populations found no corresponding evidence of local adaptation to cold-winter survival, metabolic suppression, or supercooling points [10]. Adaptation of one seasonal trait therefore does not demonstrate generalized adaptation across the entire overwintering phenotype.

Diapause is more precisely a stage-specific, programmed developmental arrest used by many insects to bridge unfavourable seasons, and its induction, maintenance, termination, and post-diapause phases should not be conflated [11]. Prolonged diapause can extend developmental delay beyond a single adverse season, and in univoltine or semivoltine life cycles this temporal structure has been explicitly discussed in relation to plasticity and bet-hedging [12]. Nevertheless, prolonged development is not automatically a diversifying strategy: delays may be obligatory consequences of developmental schedules, resource acquisition, or cue requirements.

Physiological evidence further limits simple interpretations. Molecular responses associated with winter survival include transcriptional reorganization, cryoprotectant production, energetic storage, stress signalling, and repair, but these mechanisms vary substantially among species and remain taxonomically biased in the available evidence [13]. Nutritional status also influences diapause phenotypes in context-dependent ways. Poor-quality food can promote diapause in some systems whereas acute food deprivation can avert it, and evidence that stored nutrients generally regulate diapause duration remains insufficient [14]. Hence, the energetic state associated with dormancy cannot be reduced to a universal threshold or mechanism.

The timing of dormancy itself may impose costs. In *Pieris napi*, experimentally longer and warmer prewinter periods increased mass loss and were followed by reduced post-winter survival and lower adult dry mass among survivors [15]. This demonstrates that entering a dormant state does not simply eliminate temporal risk; it can shift risk to energetic depletion accumulated during an extended inactive interval. In invasive *Aedes albopictus*, genetically differentiated critical photoperiods for diapause corresponded closely to location-specific growing-degree-day deadlines beyond which another generation could no longer be completed, linking anticipatory photoperiodic timing to seasonal developmental opportunity [16].

Evidence for evolutionary responses to changing winters is consequently uneven. Phenological traits currently provide stronger examples of evolutionary shifts than many other winter-relevant physiological traits, even where variation suggests evolutionary potential [17]. In *Pararge aegeria*, population differences in diapause duration did not map simply onto winter length; instead, the observed pattern was more consistent with selection associated with warm autumn conditions and voltinism, although inference was necessarily limited by the number and identity of populations compared [18].

These findings motivate a narrower interpretation of dormancy in the proposed framework. Diapause can spread exposure across time when individuals or offspring do not all resume development in the same seasonal window, but dormancy can also be a highly predictable cue-responsive programme, an obligatory developmental feature, or a physiological response with substantial energetic costs. The evolutionary question is therefore not whether an insect diapauses, but whether variation in entry, duration, termination, or incidence creates a distribution of reproductive outcomes that buffers uncertain future conditions in a way not better explained by adaptive plasticity or constraint.

Variable developmental duration

Developmental duration provides a second candidate route to temporal risk spreading because variation in the completion of egg, larval, pupal, or nymphal development alters when individuals encounter later environmental states. Importantly, much of this variation can arise without any evolved diversification mechanism. Fine-scale microclimatic differences only metres apart can generate substantial differences in developmental phenology, emergence synchrony, and voltinism within a single *Pieris napi* population [19]. An observed spread of emergence dates may therefore reflect environmental heterogeneity rather than genotype-level diversification.

Thermal reaction norms nevertheless make developmental timing biologically structured rather than arbitrary. In *P. napi*, development times measured across naturally variable microclimates could be predicted from laboratory thermal-performance relationships when non-linearity, spatially explicit microclimate, and temporal temperature variation were incorporated [20]. This result cautions against treating all environmentally generated developmental variance as an independent response to fluctuation. Temperature-sensitive development can itself account for heterogeneous phenological shifts among insect populations, while extrapolation from laboratory conditions to field phenology remains dependent on realistic environmental variation and explicit links to fitness [21].

The population consequences of altered timing are also not unidirectional. Earlier seasonal development can create favourable demographic opportunities in some circumstances while producing developmental traps in others, as demonstrated for a multivoltine butterfly under climate change [22]. The proposed framework therefore distinguishes developmental asynchrony from developmental bet-hedging. The former is any spread in developmental completion times; the latter would require evidence that some heritable or systematically generated component of that spread reduces covariance in descendant failure across uncertain temporal states and increases long-term lineage success. Developmental variability caused entirely by predictable thermal exposure, by contrast, is better interpreted as environmental forcing or plasticity unless additional evolutionary evidence is available.

Reproductive scheduling

Risk can also be redistributed after maturation. Reproduction is itself thermally contingent: temperature can change activity, mate competition, signalling, mating costs, and the behavioural interactions that determine reproductive outcomes [23]. Thus, spreading mating or oviposition across time could expose reproductive attempts to partially independent thermal conditions. Whether this constitutes bet-hedging, however, depends on whether temporal diversification exceeds what is expected from cue-responsive behaviour, reproductive physiology, or simple limitation of available mates and substrates.

The timing of environmental stress relative to reproductive events can strongly alter consequences. In the burying beetle *Nicrophorus vespilloides*, experimentally imposed heatwaves had different effects depending on whether they occurred before, during, or after mating, with the strongest reproductive costs occurring when exposure coincided with mating [24]. Reproductive timing can therefore change the fitness consequence of the same physical stressor over intervals of only a few days.

Oviposition extends this problem by distributing offspring simultaneously across time and space. Female *Gryllus lineaticeps* altered egg laying in response to thermal and spatial heterogeneity; increased availability of oviposition sites changed the distribution of eggs, and warmer sites produced a trade-off between faster development and lower embryo survival [25]. The study explicitly characterized part of this behaviour as spatial bet-hedging, but its temperature sensitivity also illustrates why risk spreading and plasticity may operate jointly rather than as mutually exclusive categories.

Maternal preference may additionally fail when historical cue–fitness relationships break down. Female *Melitaea cinxia* tended to select microhabitats associated with drought-stressed host plants that often supported larval survival, yet during an exceptionally dry year this preference was associated with extremely poor offspring survival [26]. Such results show that repeated use of an apparently favourable reproductive state need not protect populations under environmental extremes. The framework consequently treats reproductive spreading as a candidate hedge only when diversification among reproductive bouts, dates, or oviposition sites reduces shared failure under unpredictable conditions rather than merely tracking currently favourable cues.

Dispersal and spatial diversification

Dispersal can convert spatial heterogeneity into temporal insurance when descendants occupying different locations subsequently experience partly independent sequences of environmental conditions. In wing-polymorphic insects, alternative dispersal phenotypes are produced through developmental systems linking environmental perception, signalling, morphology, and physiology [27]. Because winged and wing-reduced morphs often differ in mobility and reproductive allocation, dispersal polymorphism provides a plausible mechanism for dividing descendants between local exploitation and movement to alternative environments.

Yet polymorphism alone does not identify bet-hedging. A phylogenetic analysis of aphids showed that male wing polymorphisms are evolutionarily transient and have arisen repeatedly, while their distribution is associated with ecological traits including host alternation and host breadth [28]. Such macroevolutionary patterns establish

repeated evolution of alternative dispersal phenotypes but do not demonstrate that within-generation morph mixtures maximize geometric mean fitness under stochastic environments.

Spatial diversification is also constrained by landscape structure. Fine-scale landscape-genomic analysis of the Alpine butterfly *Lycaena tityrus* found high regional gene flow but detectable effects of anthropogenic barriers on genetic connectivity [29]. Consequently, the theoretical capacity of dispersal to spread temporal risk depends on realized movement and demographic independence among destinations, not simply on possession of wings or a dispersal morph.

Mechanistically, environmental conditions such as nutrition and crowding can bias insects toward reproductive or migratory phenotypes through endocrine and cellular signalling pathways associated with wing polyphenism [30]. Such condition-dependent switching is more naturally interpreted as plasticity when the inducing cue provides reliable information about the relative value of dispersal. A bet-hedging interpretation instead requires a residual diversified component, or a mixed strategy, whose advantage arises because future habitat quality cannot be reliably predicted at the time the dispersal phenotype is determined.

The proposed framework therefore treats dispersal as a bridge between spatial and temporal risk spreading. Spatial separation matters evolutionarily when it reduces covariance among future reproductive outcomes: offspring that disperse to different patches may encounter distinct future temperatures, resources, competitors, enemies, or disturbance histories. Conversely, movement into strongly correlated environments provides little temporal insurance, and dispersal into consistently inferior habitats can lower rather than stabilize lineage fitness. Determining whether dispersal functions as bet-hedging thus requires coupling dispersal phenotype, destination structure, environmental covariance, reproductive trade-offs, and multigenerational fitness rather than inferring strategy from movement alone.

Offspring heterogeneity

Offspring heterogeneity can arise through mechanisms that differ fundamentally in their evolutionary interpretation. In *Drosophila*, environmental predictability influenced within- and transgenerational plasticity of heat tolerance across life stages and climatic regions, demonstrating that parental and developmental environments can structure descendant phenotypes without requiring a bet-hedging explanation [31]. The first question is therefore whether heterogeneous offspring states represent information-dependent phenotype matching or diversification under uncertainty.

Nongenetic transmission can itself be contingent. In field crickets, inherited behavioural variability differed according to context and sex, indicating that transmission of phenotypic variance need not be uniform among traits or descendants [32]. Parental condition can also generate offspring differences: interacting maternal and paternal sugar consumption altered offspring life-history and physiological traits in *Drosophila melanogaster* [33]. Such parental effects establish pathways through which offspring phenotypes can vary, but they do not by themselves demonstrate that parents have evolved to diversify descendant risk.

The proposed framework consequently separates generated heterogeneity from risk-spreading heterogeneity. Generated heterogeneity includes offspring differences attributable to predictive transgenerational plasticity, parental condition, developmental noise, microenvironmental variation, or other processes whose fitness consequences remain unresolved. Risk-spreading heterogeneity is reserved for cases in which descendants occupy states that differ meaningfully in their exposure to uncertain future environments. These states might involve developmental timing, diapause propensity, dispersal phenotype, thermal tolerance, or reproductive schedule, but no particular trait is required.

A stronger bet-hedging interpretation requires another step: heterogeneity must reduce covariance in failure among descendants sufficiently to improve long-term lineage performance despite possible costs to mean performance [1]. For example, environmentally generated differences in emergence time can spread descendants across dates [19], but the pattern remains environmental forcing unless diversification has an evolved component with appropriate multigenerational fitness consequences. Likewise, spatially distributed oviposition can diversify developmental environments [25], yet cue-responsive site choice may remain ordinary plasticity.

The analytical problem is therefore not whether offspring differ, but why they differ, whether their future exposures are sufficiently independent, and what happens to lineage fitness across repeated environmental sequences. **Table 1** separates these states without treating phenotypic variance as an adaptive score.

Table 1. Offspring heterogeneity states that require different evolutionary interpretations in temporal risk-spreading analysis

State production		Interpretation boundary		Evidence for temporal risk spreading	
Origin of heterogeneity	**Observed descendant pattern**	**Evolutionary interpretation allowed**	**Alternative explanation**	**Fitness or exposure test**	**Standing in this article**
Cue-aligned transmission					
Cue-aligned transgenerational state	Descendant phenotype changes with parental or developmental environmental information	Potential anticipatory phenotype matching	Reliable cues can generate plasticity without diversification for uncertain futures	Test whether offspring state predicts the future environment better than alternative phenotypes	Evidence-supported pattern; classification proposed
Parental-condition pathways					
Context- or sex-dependent variance transmission	Phenotypic variability is transmitted differently among contexts or sexes	Conditional inheritance of variance	Trait- and sex-specific inheritance prevents population-wide generalization	Partition transmission, fitness and environmental exposure by sex and behavioural context	Evidence-supported pattern; interpretation boundary proposed
Biparental condition-dependent state	Maternal and paternal environments jointly modify descendant phenotype	Parental environmental effect	Physiological carry-over may explain heterogeneity without adaptive risk spreading	Separate parental condition effects from evolved diversification under uncertainty	Evidence-supported pattern; classification proposed
Developmental microenvironment					
Microenvironment-generated developmental spread	Cohort members experience different temperatures or developmental opportunities	Environmentally generated asynchrony	Spatial microclimate variation can create temporal spread without heritable diversification	Control developmental environment before attributing variance to a diversification strategy	Evidence-supported distinction
Residual diversification					
Residual diversified offspring state	Offspring remain distributed among alternative states after predictable cue effects are controlled	Candidate diversifying bet-hedging	Developmental noise, constraint and incomplete cue perception remain alternatives	Test whether diversification lowers covariance in descendant failure and increases long-term lineage performance	Proposed bet-hedging candidate state
Cross-domain descendant diversification	Offspring differ simultaneously in timing, dormancy, movement or later reproductive opportunity	Potential multicomponent risk spreading	Correlated traits may duplicate rather than diversify exposure	Quantify covariance among trait states and among environmental outcomes rather than counting trait differences	Proposed integrative state

The distinction becomes clearer when offspring states are placed between their developmental origins and their eventual environmental outcomes. **Figure 1** therefore represents heterogeneity as an inference problem rather than equating greater phenotypic spread with stronger bet-hedging.

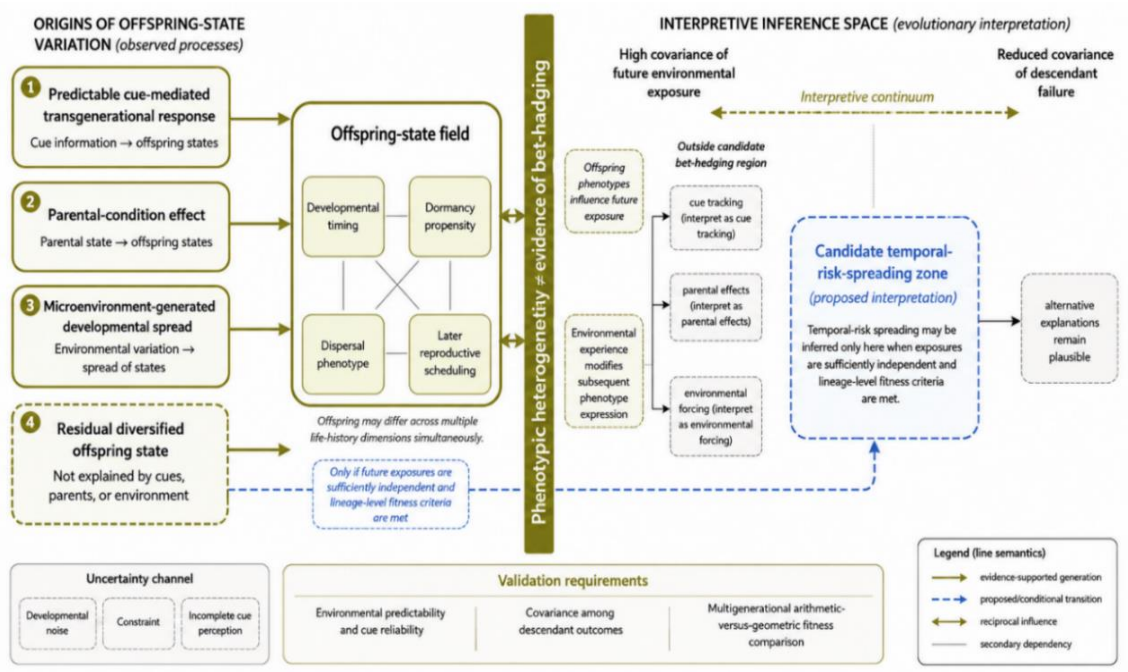


Figure 1. When offspring diversity becomes temporal risk spreading: state boundaries between cue tracking, environmental forcing and diversified future exposure

Integrated temporal risk-spreading architecture

The evidence considered above indicates that temporal risk spreading cannot be inferred from a single trait category. The proposed architecture instead recognizes five analytically distinct routes: dormancy, developmental-duration variation, reproductive scheduling, dispersal-mediated spatial diversification, and offspring heterogeneity. Their common property is not phenotype type but their potential to distribute descendant success across environmental states that are imperfectly correlated in time or space.

These routes are not assumed to be additive. A prolonged diapause may already generate substantial temporal separation [12], whereas additional variation in emergence timing may contribute little if all descendants subsequently encounter the same environmental episode. Conversely, spatial diversification through oviposition or dispersal may reduce covariance among future conditions even when developmental timing is similar [25]. The framework therefore places covariance of descendant outcomes, rather than number of variable traits, at its centre.

The architecture also allows one trait to change the value of another. Developmental timing can alter whether diapause is entered [16]; microclimate can change both phenology and voltinism [19]; dispersal phenotype can redistribute exposure among habitats [27]; and parental environments can alter descendant physiological states [31]. These relationships support integration at the level of exposure structure, but they do not establish that any particular multicomponent combination evolved through bet-hedging.

Accordingly, the proposed architecture defines temporal risk spreading as a functional outcome that may arise through different biological mechanisms. Bet-hedging is a narrower evolutionary interpretation requiring evidence that diversification or conservative variance control improves long-term lineage performance under environmental uncertainty.

Distinguishing bet-hedging from plasticity

Plasticity and bet-hedging can generate superficially similar population-level variation. The key difference lies in information. When an environmental cue reliably predicts conditions affecting fitness, phenotype adjustment can increase environment matching; declining predictability can alter the selective value of such plasticity [5]. In diapause systems, reaction-norm shape and environmental predictability can likewise produce combinations of plastic and diversified responses rather than a categorical either–or distinction [6].

The proposed framework therefore applies a sequential inference rule. First, determine whether observed variation can be explained by current environmental exposure, parental condition or predictable cues. Second, identify whether a residual diversified component remains after those effects are accounted for. Third, determine whether

alternative states encounter sufficiently different future environments to reduce correlated failure. Finally, compare long-term fitness consequences rather than assuming that reduced variance is advantageous.

This rule also excludes several common overinterpretations. Thermal reaction norms that accurately predict field development [20] support environmental dependence, not automatically bet-hedging. Repeated evolution of dispersal polymorphism [28] demonstrates evolutionary lability, not geometric-mean-fitness advantage. Transgenerational plasticity [31] demonstrates inherited environmental effects but can remain predictive plasticity. Bet-hedging should therefore be treated as a hypothesis requiring stronger evidence than phenotypic heterogeneity itself.

Future tests

Future tests should connect phenotype production to multigenerational environmental sequences. Common-garden or pedigree designs can first partition genetic, parental and immediate environmental contributions to variance. Experimental manipulation of cue reliability can then test whether individuals track informative environments or maintain diversification when prediction becomes unreliable. Developmental timing, diapause state, oviposition choice and dispersal should be measured jointly where possible because apparently independent traits may expose descendants to the same environmental episode.

Fitness analysis should distinguish arithmetic mean success from variance and geometric mean performance [2]. Particularly informative designs would quantify covariance among siblings or descendant groups exposed to different dates or locations rather than treating phenotypic variance itself as the response. Replicated environmental sequences are essential because a strategy cannot be classified as risk spreading from one favourable or adverse year.

Comparative tests across populations differing in seasonality or environmental predictability could examine whether diversification changes systematically with uncertainty, while reciprocal or transplant designs could separate local cue adaptation from generalized winter or stress tolerance [9]. Individual-based or demographic models could then ask when empirically measured trait distributions are sufficient to alter lineage persistence without converting model predictions into evidence of adaptation.

The strongest future test is therefore falsification-oriented: a proposed bet-hedging mechanism should lose support if cue-responsive plasticity, environmental forcing, developmental constraint or parental condition explains the same pattern without a long-term variance-reduction advantage.

Conclusion

Temporal risk spreading in insects is broader than dormancy but narrower than phenotypic variation. Developmental timing, reproductive scheduling, dispersal and offspring heterogeneity can redistribute exposure across future environments, yet none constitutes bet-hedging by definition. The proposed framework separates observable heterogeneity, exposure diversification and evolutionary interpretation, placing covariance among descendant outcomes and long-term lineage fitness at its core. This structure accommodates multiple life-history mechanisms while preserving critical boundaries among environmental forcing, plasticity, parental effects, constraint and bet-hedging. Its value is therefore not to classify insect traits prematurely, but to specify what evidence would be required before temporal diversification can be interpreted as an evolved strategy for surviving environmental uncertainty.

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