Eurasia Specialized Veterinary Publication

Entomological Research Letters

ISSN: 3108-639X

2022, Volume 2, Issue 1, Page No: 60-70
Copyright CC BY-NC-SA 4.0
Available online at: www.esvpub.com/

Maternal Effects Function as Cross-Generational Environmental Forecasts That Can Improve or Misguide Offspring Life-History Programming

Ahmed Ben Ali[1]*, Samira Gharbi[1], Nabil Jebali[2]

[1]Department of Maternal Effects and Transgenerational Plasticity, Faculty of Sciences, University of Tunis, Tunis, Tunisia.
[2]Department of Offspring Programming and Environmental Forecasting, Faculty of Biosciences, University of Sfax, Sfax, Tunisia.

***E-mail** ✉ ahmed.benali@fst.rnu.tn

ABSTRACT

Maternal effects are a major route by which environmental variation experienced in one generation can influence the phenotype of the next, but their evolutionary interpretation is often stronger than the evidence permits. In insects, mothers can alter offspring development, diapause, thermal tolerance, immunity, morphology, allocation, and stress responses through behavioral, endocrine, nutritional, molecular, and immune routes. Yet cross-generational phenotype change does not by itself demonstrate that maternal information predicts offspring environments or that induced phenotypes improve fitness. This evolutionary perspective develops a proposed cross-generational forecast model in which maternal effects qualify as candidate environmental forecasts only when three conditions are considered jointly: parent–offspring environmental correspondence, transmission fidelity, and context-dependent efficacy of offspring programming. Evidence from thermal regimes, diapause systems, nutrition, toxicants, pathogens, and maternal condition shows both potentially beneficial and misleading cross-generational effects. The framework therefore distinguishes information transfer from physiological carry-over, mechanism from adaptive interpretation, trait response from fitness consequence, and laboratory matching from field generalization. It predicts that maternal programming should be most informative when cue reliability is high, mechanisms transmit relevant information faithfully, and offspring encounter environments in which the induced phenotype improves performance. Conversely, environmental mismatch, altered cue structure, stress intensity, or nonforecast maternal influences may produce neutral or harmful consequences. The article uses these distinctions to organize testable predictions for mechanistic and evolutionary validation in insects.

Keywords: Maternal effects, Transgenerational plasticity, Environmental predictability, Insect life history, Developmental programming, Diapause

Received: 27 March 2022
Revised: 18 May 2022
Accepted: 20 May 2022

How to Cite This Article: Ben Ali A, Gharbi S, Jebali N. Maternal Effects Function as Cross-Generational Environmental Forecasts That Can Improve or Misguide Offspring Life-History Programming. Entomol Res Lett. 2022;2(1):60-70. https://doi.org/10.51847/Ljp3CT2pj

Introduction

Maternal effects provide a route through which environmental information, physiological state, and resource allocation in mothers can alter offspring phenotype. In insects, such effects occur across developmental, physiological, immune, and behavioral systems, but their interpretation requires more than demonstrating that offspring from different maternal environments differ. Cross-generational plasticity can involve multiple sender–receiver steps, distinct mechanisms, and effects that persist for different numbers of generations. A useful evolutionary interpretation must therefore separate the existence of a maternal effect from the proposition that the effect carries information about future conditions [1].

© 2022 Entomological Research Letters

This distinction becomes especially important in changing environments. Human-altered conditions can weaken historical relationships between cues and future environments, disrupt the transmission of information, or expose offspring to combinations of stressors not encountered in the parental generation [2]. Population-genetic models likewise show that maternal control is favored only under particular combinations of predictive information and response costs; in other parameter spaces, within-generation plasticity or alternative strategies may be favored instead [3]. Consequently, the relevant question is not whether mothers influence offspring, but whether maternal influence improves the match between offspring phenotype and the environment actually encountered.

The empirical literature does not justify treating that match as universal. Meta-analysis has reported beneficial transgenerational effects across organisms, but subsequent critique has questioned whether the available designs consistently satisfy strict criteria for adaptive transgenerational plasticity, while a predator-induced meta-analysis found substantial heterogeneity in magnitude and direction [4-6]. The productive synthesis is therefore neither that maternal effects are generally adaptive forecasts nor that adaptive forecasting is implausible. Rather, the evidence suggests that forecasting is conditional and must be demonstrated at several inferential levels.

This perspective focuses on insects because their short generation times, pronounced seasonality, discrete developmental stages, diapause systems, ecological specialization, and tractable physiological mechanisms make them particularly informative for resolving this problem. The central contribution is a proposed framework that treats maternal effects as candidate cross-generational forecasts only when environmental predictability, transmission mechanism, offspring response, and realized consequence are distinguished explicitly. The framework also retains nonforecast explanations—including maternal condition, direct carry-over, terminal investment, parent–offspring conflict, and developmental constraint—so that inherited phenotype change is not automatically assigned an anticipatory function.

Materials and Methods

This article is an evolutionary perspective. The analytical procedure was conceptual rather than statistical: evidence was organized according to whether it addressed environmental predictability, maternal information sources, transmission mechanisms, offspring phenotypic responses, mismatch, or evolutionary interpretation. Experimental and theoretical studies were used for different inferential purposes rather than pooled into a single evidence class.

A first methodological distinction separates genuine cross-generational responses from direct exposure, within-generation plasticity, selection, and uncontrolled carry-over. Multigenerational experimental design is critical because treatment of parents and offspring can otherwise confound inherited effects with direct environmental responses [7]. A second distinction concerns evolutionary interests. Maternal effects need not maximize offspring fitness when maternal and offspring optima diverge, so apparently organized developmental programming can arise under parent–offspring conflict rather than cooperative forecasting [8].

Forecasting logic was defined by environmental correspondence. Temporal autocorrelation is particularly important because maternal information is potentially useful only when parental conditions provide information about conditions descendants are likely to encounter. Analyses of environmental variation indicate that stronger temporal predictability can favor greater transgenerational plasticity, whereas weak correlation reduces its expected value [9]. Spatial structure, migration, and the costs of transmission can further alter whether inherited environmentally responsive states evolve or persist [10].

For the present synthesis, maternal effects are therefore sorted into four analytical classes rather than treated as a single mechanism: (i) candidate environmental information, such as thermal, seasonal, pathogenic, toxicological, or resource-related conditions; (ii) maternal physiological state, including age and nutritional condition, which may influence offspring without necessarily encoding predictive information; (iii) transmission routes, including endocrine, molecular, immune, epigenetic-associated, behavioral, and provisioning processes; and (iv) offspring consequences, including developmental timing, tolerance, immunity, allocation, survival, and reproduction. The resulting classification is interpretive, not taxonomic. It is designed to prevent level-of-analysis slippage between cue, mechanism, trait, and fitness.

Results and Discussion

Thermal conditions provide one of the clearest candidate information sources because temperature can vary predictably across seasons and generations. In *Drosophila mojavensis*, within- and transgenerational heat-tolerance plasticity differed across life stages and climatic regions, indicating that thermal information is filtered by both developmental stage and environmental history [11]. The result supports thermal predictability as a plausible forecast variable, but it does not imply that all populations or stages respond similarly.

Toxicant exposure shows why maternal state must remain analytically distinct from environmental information. In *Spodoptera littoralis*, maternal body mass influenced larval sensitivity to chlorpyrifos, demonstrating a maternal effect on stress susceptibility without establishing that mothers were transmitting an anticipatory pesticide signal [12]. In *Culex pipiens*, parental exposure to warming and pesticide stress produced transgenerational interactions that were frequently costly rather than uniformly protective [13]. Nutritional history adds another layer: in *Aedes aegypti*, parental nutritional condition altered reproductive allocation, illustrating how resource state can change offspring provisioning independently of environmental prediction [14].

Behavioral modification of offspring environments can also transmit maternal influence. In *Onthophagus taurus*, maternal behavioral plasticity moderated temperature effects on development, but the pattern varied among populations, showing that maternal environmental modification can buffer offspring while remaining context-specific [15]. Seasonal diapause systems provide especially informative tests because photoperiod and temperature can carry information about future conditions. In *Aphidius ervi*, transgenerational effects on diapause and life-history traits differed between strains and did not produce a single maternal rule [16]. In *Trichogramma telengai*, grandmaternal temperature influenced diapause induction only under restricted photoperiodic conditions, emphasizing that ancestral information can be contingent on another cue rather than acting independently [17].

Maternal age further demonstrates why inherited variation should not automatically be interpreted as forecasting. In *Drosophila melanogaster*, maternal and grandmaternal age affected offspring viability and performance, but effects varied with genotype and larval density [18]. Age therefore functions here as a maternal-state variable capable of changing offspring phenotype, not as evidence that mothers anticipate future environments. Taken together, these studies support a heterogeneous information landscape in which environmental cues, maternal condition, and allocation states can all produce cross-generational effects but differ in predictive meaning.

Transmission mechanisms

A maternal effect can only operate as a forecast if information or maternal state reaches offspring through a biologically effective route. Yet mechanism alone establishes transmission, not evolutionary purpose. The strongest evidence therefore comes from studies that manipulate candidate pathways while measuring descendant phenotypes.

Endocrine control provides a clear example. In *Nasonia vitripennis*, juvenile hormone was causally implicated in maternal regulation of offspring diapause: molecular and hormonal manipulation linked maternal endocrine state to a defined developmental outcome [19]. This demonstrates a causal transmission route in a specific wasp system, but it does not establish that juvenile hormone mediates maternal forecasting across insects.

Molecular provisioning can likewise alter offspring tolerance. Experimental manipulation of maternal Hsp23 loading in *Drosophila melanogaster* increased embryonic thermal tolerance, showing that maternally supplied molecules can directly alter descendant stress performance [20]. Because the study used targeted manipulation, it is strong evidence for a mechanism, while the natural environmental conditions under which such loading functions adaptively remain a separate question.

Epigenetic-associated molecular change occupies a more cautious evidentiary position. In *Leptinotarsa decemlineata*, insecticide exposure altered intergenerational DNA methylation patterns, supporting persistent molecular effects across generations without proving that methylation itself causes adaptive resistance [21]. In the pea aphid *Acyrthosiphon pisum*, manipulation of the l-DOPA/dopamine pathway demonstrated transgenerational regulation of cuticular melanization, providing stronger functional evidence for a maternal biochemical pathway but not for the fitness value of the resulting pigmentation [22].

Immune systems show that transmission routes can be both specific and heterogeneous. Long-lasting transgenerational antiviral immunity has been demonstrated in insect models; maternal immune challenge can protect mealworm eggs and early offspring; and mechanistic reviews emphasize that insect transgenerational immune priming can involve different maternal and paternal routes [23-25]. Transcriptomic work in *Manduca sexta* additionally shows that maternal immune exposure can alter offspring immune-related gene expression [26].

These findings justify treating immune information transfer as a mechanistic class while resisting the assumption that one pathway explains all systems.

Table 1 shows the same observation—an inherited molecular or physiological change—supports different evolutionary inferences depending on mechanism, taxon, and level of validation.

Table 1. Transmission mechanisms and the inferential boundaries between inherited information, offspring regulation, and forecasting

Across-generation mechanism			Forecast eligibility	
Transmission route	**Maternal/offspring system**	**Inherited substrate**	**Forecast meaning**	**Separation test**
Endocrine regulation	Parasitoid diapause	Hormone manipulation alters offspring developmental state	Direct maternal control of development. Evidence-supported mechanism.	Species- and pathway-specific. Link endocrine change to cue predictability and fitness
Molecular provisioning	Drosophila thermal tolerance	Manipulated maternal product changes embryo tolerance	Maternal products can alter offspring stress phenotype. Evidence-supported mechanism.	Natural regulation not established by manipulation alone. Measure naturally induced provisioning under matching and mismatching environments
Epigenetic-associated change	Beetle toxicant exposure	Intergenerational molecular-state differences	Environmental history can persist molecularly. Evidence-supported association.	Causality to phenotype or fitness unresolved. Functional perturbation plus descendant performance
Immune information transfer	Pathogen-exposed insects	Protection, immune assays, molecular responses	Pathogen-related information can affect descendants. Evidence-supported but heterogeneous.	Pathogen, taxon, and route specificity. Separate priming from vertical transmission and generalized stress
Behavioral/resource-mediated effect	Maternal environmental modification or provisioning	Maternal behavior or condition changes offspring environment/resources	Maternal state can alter developmental context. Evidence-supported class.	May not encode future conditions. Manipulate maternal state independently of cue reliability
Forecast linkage	Cross-level integration	Cue–mechanism–phenotype chain	Transmission may contribute to forecasting only if predictive value is demonstrated. Proposed synthesis.	Mechanism is not equivalent to adaptation. Test cue reliability, mechanism, and fitness jointly

Adaptive forecasting under environmental autocorrelation

Environmental autocorrelation is the central ecological condition that can convert a maternal cue into potentially useful information. If maternal and offspring environments are correlated, delayed information may help offspring prepare before they can sample the environment directly. If the relationship is weak or changes rapidly, the same inherited program may be irrelevant or harmful. This logic is supported theoretically by analyses of environmental predictability [9] and empirically by insect thermal studies in which developmental stage and climatic context alter the value of inherited thermal responses [11].

Evidence from variable thermal landscapes further shows that within- and transgenerational changes in thermal tolerance can arise rapidly but are accompanied by context-dependent performance and fitness consequences [27]. Such results support conditional forecasting rather than a universal buffering mechanism. A thermal response that improves performance under one regime may impose costs under another, and an effect measured in one stage may not predict adult performance.

Diapause systems reinforce this point. Maternal or grandmaternal temperature can influence diapause-related traits, but responses depend on strain and photoperiod [16]. Grandmaternal temperature effects in *T. telengai*, for example, were restricted to particular short-day conditions [17]. These systems therefore resemble multi-cue forecasting problems in which thermal information is interpreted against a photoperiodic background rather than acting as a standalone signal.

Figure 1 shows that forecasting emerges from nested relations among predictability, maternal information, mechanism, response, and validation rather than from a single maternal-effect arrow.

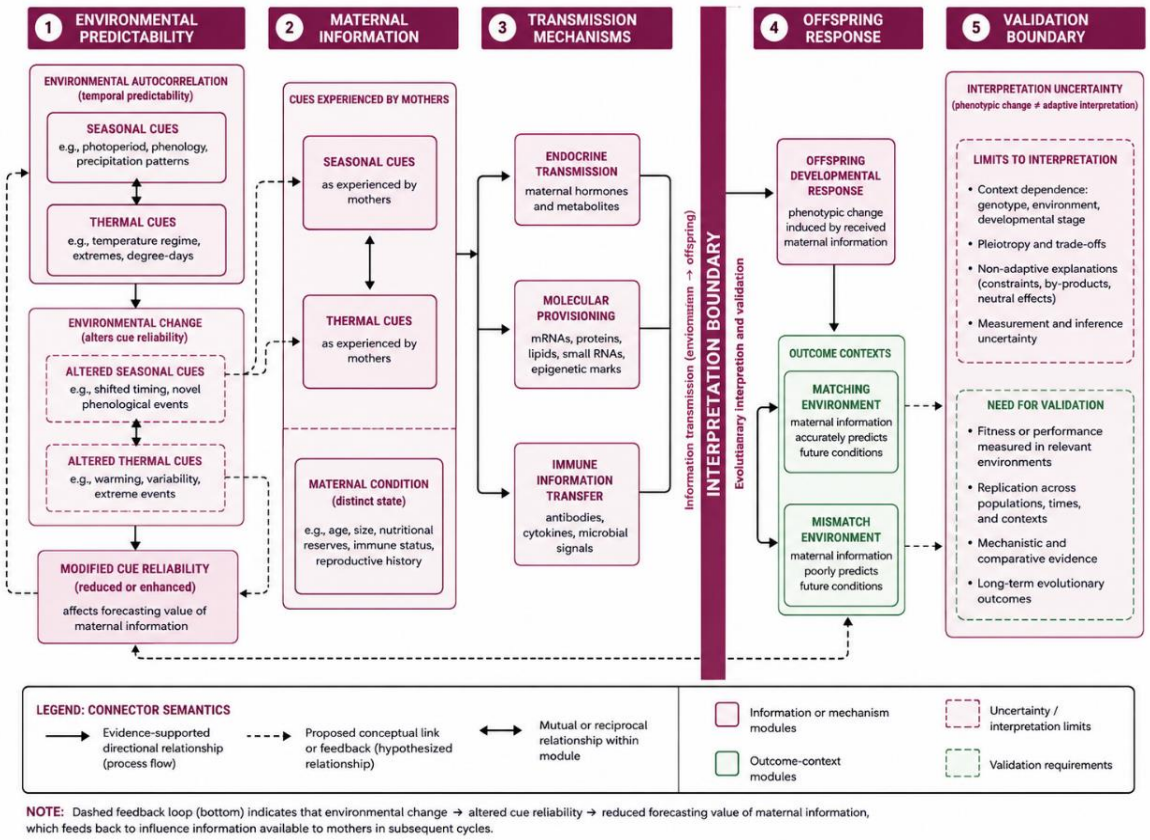


Figure 1. Environmental predictability and the conditional conversion of maternal information into offspring programming

Maladaptive programming under environmental mismatch

Forecast failure is most plausible when maternal information no longer corresponds to offspring conditions, but harmful offspring outcomes can also arise without forecasting. The interpretive problem is therefore to distinguish a misleading inherited prediction from stress damage, condition-dependent allocation, or another maternal effect. Anthropogenic toxicants illustrate this distinction. In *Callosobruchus maculatus*, pesticide exposure produced sex- and generation-specific life-history effects compatible with transgenerational plasticity and potential terminal investment [28]. The results make terminal investment a plausible competing interpretation rather than demonstrating that pesticide exposure generated a predictive maternal program. In *S. littoralis*, temperature altered insecticide sensitivity in a family-dependent transgenerational context, showing that the consequences of parental exposure can change with offspring environment [29].

Pathogen dose provides an especially strong example of sign reversal. In *Spodoptera exempta*, low parental viral exposure was associated with increased offspring resistance, whereas higher exposure produced greater susceptibility and transmission-related costs [30]. Thus, the same broad environmental category can yield beneficial or harmful descendant outcomes depending on exposure intensity. A binary "primed versus not primed" model would miss this transition.

Environmental mismatch should therefore be defined by discordance among parental information, transmitted state, offspring environment, and realized performance—not merely by exposure to different conditions. Maternal physiological state may also produce poor offspring performance without any predictive error. **Table 2** separates these cases so that harmful cross-generational effects are not automatically labeled maladaptive forecasts.

Table 2. Stage-specific mismatch states that distinguish misleading forecasts from nonforecast maternal effects

Mismatch mode	Offspring pattern	Why the forecast can fail	Alternative-strategy test and standing

Cue–environment discordance. Seasonal, thermal, toxicant, or pathogen systems	Maternal and offspring conditions differ	Previously useful information may become misleading. Historical cue reliability often unknown	Manipulate parent–offspring correlation independently of exposure severity. Proposed forecast-failure state.
Transmission without benefit. Mechanism is detected but offspring performance does not improve	Molecular or physiological inheritance without performance gain	Information is transmitted but program efficacy is absent. Trait measured may not reflect fitness	Cross mechanism state with matching and mismatching environments. Proposed model state.
Stress-induced vulnerability. Toxicant or combined-stressor systems	Offspring performance declines after parental exposure	Carry-over damage may mimic maladaptive programming. Mechanism may be unresolved	Separate parental damage from transmitted signaling. Evidence-supported possibility.
Dose-dependent reversal. Host–pathogen systems	Low exposure and high exposure produce opposite descendant effects	Forecast value can change with exposure intensity. Threshold is system-specific	Use exposure gradients rather than binary treatments. Evidence-supported in focal system.
Maternal-condition effect. Nutrition, age, activity, or allocation state	Offspring phenotype varies with maternal state	Cross-generational effect may be nonpredictive. Maternal state may covary with environment	Manipulate condition independently of environmental cue. Evidence-supported distinction.
Alternative evolutionary strategy. Terminal investment or conflict	Maternal phenotype changes offspring outcomes without clear matching benefit	Effect may optimize maternal rather than offspring returns. Fitness interests may differ	Measure maternal and offspring fitness jointly. Proposed interpretive category.

Offspring life-history consequences

The evolutionary significance of maternal effects is realized through offspring life-history consequences, but those consequences operate at several levels. Development time, diapause, body size, thermal tolerance, immune phenotype, survival, and reproductive allocation are not interchangeable endpoints. A change in one may be beneficial, costly, or neutral when integrated across the full life cycle.

Maternal condition provides a useful example. In *Pararge aegeria*, flight-induced maternal effects altered offspring performance during drought, showing that maternal physiological state and offspring resource stress can interact across generations [31]. This effect is biologically important even if it does not represent forecasting. It shows that offspring developmental outcomes can depend on maternal state under environmental stress.

Thermal and diapause studies similarly show that benefits are contingent. Rapid transgenerational changes in thermal tolerance can coexist with performance trade-offs [27], while diapause effects depend on strain, photoperiod, and generation [16]. Maternal age effects in *Drosophila* vary with genotype and density [18], and immune priming can be protective at early stages without establishing lifetime fitness benefit [24]. These examples reinforce a core distinction: trait response is evidence of cross-generational consequence, whereas adaptation requires an appropriate fitness comparison in the relevant environment.

Cross-generational forecast model

The proposed model organizes maternal effects as a sequence of four inferential gates rather than a single causal pathway. The first is forecast eligibility: does the maternal environment contain information about the offspring environment? Environmental autocorrelation is the principal theoretical condition. The second is transmission fidelity: does a mechanism convey a relevant maternal state to offspring? The third is program efficacy: does the induced offspring phenotype function appropriately under the environment encountered? The fourth is evolutionary validation: does the response improve fitness relative to relevant alternatives?

These gates permit discordance at multiple points. Reliable information may be transmitted weakly. A strongly transmitted state may induce an ineffective phenotype. A useful phenotype may encounter an unexpected environment. Alternatively, a cross-generational effect may bypass forecasting entirely because it arises from maternal condition, passive carry-over, terminal investment, conflict, or developmental constraint. The model therefore separates forecast failure from nonforecast maternal effects.

Table 3 shows these distinctions into a prediction-and-test ledger so that each conceptual state is tied to an observation that could, in principle, falsify the proposed interpretation.

Table 3. Prediction-and-test ledger for evaluating candidate cross-generational forecasts in insects

Forecast construction			Forecast challenge		
Forecast stage	**Observable requirement**	**Meaning if supported**	**Main alternative**	**Decisive test**	**Standing**
Forecast-eligible cue	Maternal environment predicts offspring environment	Maternal information could support anticipatory programming	Coincidental correlation	Manipulate parent–offspring environmental correlation	Proposed model state
Faithful transmission	Maternal cue changes a defined offspring mechanism or state	Relevant information reaches the offspring system	Maternal condition or passive carry-over	Perturb transmission while holding exposure constant	Proposed model state
Effective program	Induced phenotype performs better in the matching offspring environment	Programming is conditionally beneficial	General stress hardening or provisioning	Cross inherited state with match and mismatch environments	Proposed model state
Forecast failure	Response becomes neutral or harmful after environmental discordance	Maternal information misguides programming in that context	Stress damage or constraint	Measure mechanism, phenotype, and fitness along an environmental gradient	Proposed model state
Nonforecast maternal effect	Cross-generational phenotype changes without predictive correspondence	Maternal influence is real but not anticipatory	Allocation, age, conflict, terminal investment	Decouple maternal state from predictive environmental cues	Proposed model state
Evolutionarily validated forecast	Matching response improves relevant fitness and survives field/population transfer	Evidence supports adaptive forecast interpretation in that system	Laboratory-specific performance benefit	Replicate across populations, life stages, and natural conditions	Proposed validation endpoint

Figure 2 shows the model depending on triaging discordance rather than placing all maternal effects on one success–failure axis.

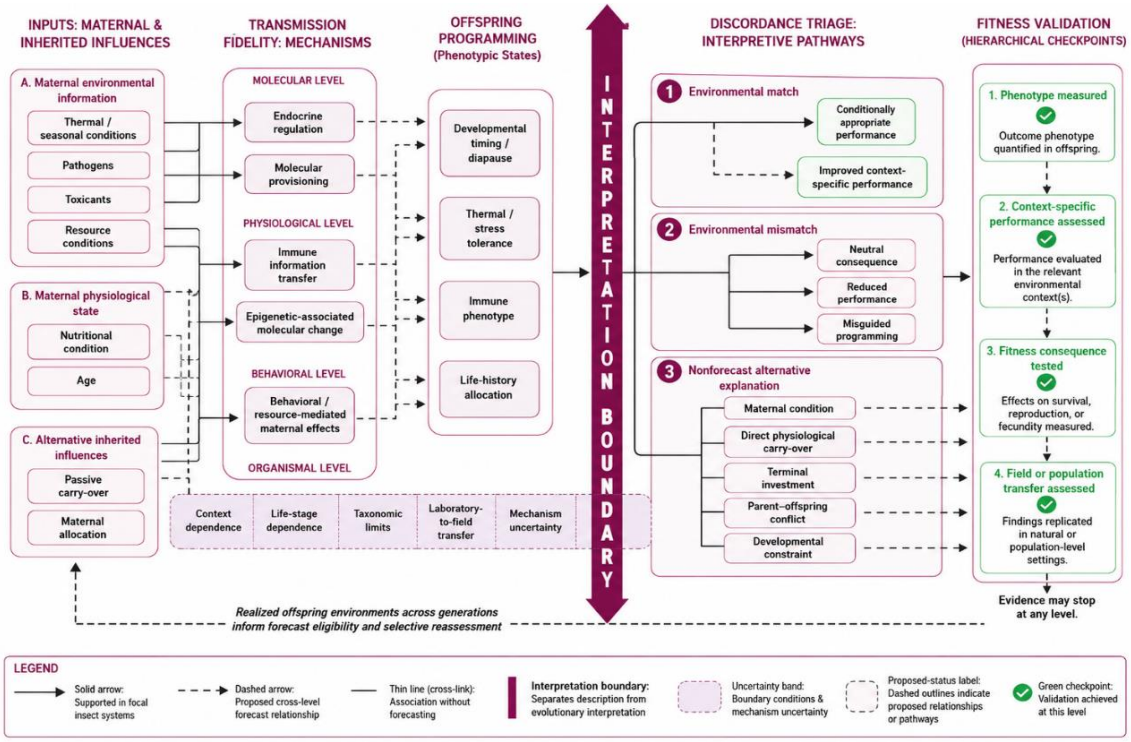


Figure 2. Where cross-generational information loses predictive value before offspring fitness can validate the forecast

The evidence supports a conditional view of maternal forecasting. Cross-generational effects are real and biologically consequential, yet the adaptive interpretation of those effects remains more demanding than the demonstration of inheritance. Broad meta-analysis has reported benefits across taxa [4], but stricter methodological critique has emphasized uncertainty about generality [5], and predator-induced responses show pronounced heterogeneity [6]. In insects, such heterogeneity is expected because cue reliability, developmental stage, physiology, generation time, and ecology differ markedly among systems.

Mechanistic evidence strengthens causal inference at the transmission level without automatically resolving evolutionary function. Juvenile hormone can causally regulate maternal diapause programming in *Nasonia* [19], and maternal Hsp23 loading can alter *Drosophila* thermal tolerance [20]. Epigenetic-associated changes, biochemical pathways, immune priming, and transcriptomic responses further demonstrate that maternal experience can reach offspring through multiple biological routes. Yet a molecular pathway establishes how a phenotype changes, not whether the change increases fitness under predicted conditions.

Laboratory-to-field transfer remains a major limitation. Many studies isolate thermal, toxicological, nutritional, or pathogenic variables under controlled conditions, whereas natural environments combine fluctuating cues and ecological interactions. Grandmaternal temperature effects that depend on photoperiod [17] and family-dependent toxicant responses modified by temperature [29] illustrate why single-factor laboratory responses cannot be assumed to persist under field complexity. Likewise, population or strain differences can change both the magnitude and direction of effects.

The proposed model therefore uses environmental predictability as a gate rather than a retrospective label. A beneficial offspring phenotype is not automatically evidence of forecasting if maternal condition or provisioning can explain it. Conversely, a harmful effect under novel conditions does not prove that the underlying maternal mechanism evolved maladaptively; environmental change may have broken a previously informative correlation. This distinction is especially important under rapid anthropogenic change, where cue reliability can shift faster than inherited response systems.

Taxonomic generalization should also remain restrained. The current evidence spans Diptera, Lepidoptera, Coleoptera, Hymenoptera, and Hemiptera, but mechanisms and outcomes differ among orders, species, strains, life stages, and sexes. The framework is therefore intended as an analytical architecture for comparison, not as a claim that all insects use the same forecasting system.

A further limitation is that maternal and offspring environments are often represented by coarse treatment categories even when insects integrate cues over different temporal windows. Photoperiod may indicate season, temperature may fluctuate over hours or days, and nutritional or pathogenic states can change within a developmental stage. Forecast accuracy therefore depends not only on whether two environments are labelled "matching," but also on whether the cue sampled by the mother corresponds to the spatial and temporal scale relevant to offspring development. The same reasoning applies to biological scale. Molecular persistence can be demonstrated before organismal consequences are known, whereas survival or fecundity differences can be measured without identifying the transmission route. Future work should deliberately connect these levels rather than treating evidence at one level as a substitute for another.

This scale-sensitive view also clarifies why null findings are informative. Failure to detect a maternal effect may indicate genuinely low forecast value, but it may also reflect measurement at the wrong life stage, insufficient environmental contrast, strain-specific thresholds, or compensatory offspring plasticity. The proposed model therefore treats absence, reversal, and context dependence as outcomes that constrain inference rather than as inconvenient exceptions.

Testable predictions

The framework generates testable predictions that distinguish forecast logic from generic maternal effects. First, the value of maternal programming should increase as parent–offspring environmental correlation increases, provided offspring cannot obtain equivalent information cheaply after development begins. Experiments should therefore manipulate predictability independently of stress severity rather than comparing only exposed and unexposed parents [9].

Second, mechanism-specific predictions require intervention. If an endocrine, molecular, immune, or provisioning pathway transmits predictive information, disrupting that route should reduce the context-dependent offspring benefit while leaving the parental environmental treatment otherwise unchanged. The strongest tests will measure pathway state, offspring phenotype, and performance in the same design.

Third, mismatch experiments should cross inherited state with both matching and mismatching offspring environments. The model predicts that genuinely predictive programming should show an environment-dependent performance advantage rather than a uniform benefit across all conditions. Dose gradients are especially important because pathogen evidence demonstrates that low and high parental exposure can reverse descendant outcomes [30].

Fourth, predictions should be replicated across life stages, sexes, populations, and field-realistic contexts. A response that appears beneficial in larvae may disappear or reverse in adults, and laboratory performance may fail to translate to reproduction or population persistence. These tests are necessary before describing a maternal effect as an evolutionarily validated forecast.

Conclusion

Maternal effects in insects can transmit environmental and physiological information across generations, but transmission is not synonymous with prediction and prediction is not synonymous with adaptation. Their evolutionary meaning depends on whether maternal conditions reliably anticipate offspring environments, whether relevant information is transmitted with sufficient fidelity, and whether the induced phenotype improves life-history performance under those conditions. The proposed cross-generational forecast model makes these requirements explicit while retaining maternal condition, carry-over, terminal investment, conflict, and developmental constraint as competing explanations. This framework therefore treats maternal effects as conditional information systems whose value can improve, disappear, or reverse as cue reliability, mechanism, developmental stage, and offspring environment change.

Acknowledgments: None

Conflict of Interest: None

Financial Support: None

Ethics Statement: None

References

1. Bell AM, Hellmann JK. An integrative framework for understanding the mechanisms and multigenerational consequences of transgenerational plasticity. Annu Rev Ecol Evol Syst. 2019;50:97-118. doi:10.1146/annurev-ecolsys-110218-024613
2. Donelan SC, Hellmann JK, Bell AM, Luttbeg B, Orrock JL, Sheriff MJ, et al. Transgenerational plasticity in human-altered environments. Trends Ecol Evol. 2020;35(2):115-24. doi:10.1016/j.tree.2019.09.003
3. Dury GJ, Wade MJ. When mother knows best: A population genetic model of transgenerational versus intragenerational plasticity. J Evol Biol. 2020;33(1):127-37. doi:10.1111/jeb.13545
4. Yin J, Zhou M, Lin Z, Li QQ, Zhang YY. Transgenerational effects benefit offspring across diverse environments: A meta-analysis in plants and animals. Ecol Lett. 2019;22(11):1976-86. doi:10.1111/ele.13373
5. Sánchez-Tójar A, Lagisz M, Moran NP, Nakagawa S, Noble DWA, Reinhold K. The jury is still out regarding the generality of adaptive 'transgenerational' effects. Ecol Lett. 2020;23(11):1715-8. doi:10.1111/ele.13479
6. MacLeod KJ, Monestier C, Ferrari MCO, McGhee KE, Sheriff MJ, Bell AM. Predator-induced transgenerational plasticity in animals: A meta-analysis. Oecologia. 2022;200(3-4):371-83. doi:10.1007/s00442-022-05274-w
7. Donelson JM, Salinas S, Munday PL, Shama LNS. Transgenerational plasticity and climate change experiments: Where do we go from here? Glob Chang Biol. 2018;24(1):13-34. doi:10.1111/gcb.13903
8. Kuijper B, Johnstone RA. Maternal effects and parent-offspring conflict. Evolution. 2018;72(2):220-33. doi:10.1111/evo.13403
9. Colicchio JM, Herman J. Empirical patterns of environmental variation favor adaptive transgenerational plasticity. Ecol Evol. 2020;10(3):1648-65. doi:10.1002/ece3.6022
10. Greenspoon PB, Spencer HG. The evolution of epigenetically mediated adaptive transgenerational plasticity in a subdivided population. Evolution. 2018;72(12):2773-80. doi:10.1111/evo.13619

11. Diaz F, Kuijper B, Hoyle RB, Talamantes N, Coleman JM, Matzkin LM. Environmental predictability drives adaptive within- and transgenerational plasticity of heat tolerance across life stages and climatic regions. Funct Ecol. 2021;35(1):153-66. doi:10.1111/1365-2435.13704
12. Bagni T, Siaussat D, Maria A, Couzi P, Maïbèche M, Massot M. A maternal effect influences sensitivity to chlorpyrifos pesticide in the pest moth Spodoptera littoralis. Ecotoxicol Environ Saf. 2020;204:111052. doi:10.1016/j.ecoenv.2020.111052
13. Tran TT, Janssens L, Dinh KV, Stoks R. Transgenerational interactions between pesticide exposure and warming in a vector mosquito. Evol Appl. 2018;11(6):906-17. doi:10.1111/eva.12605
14. Yanchula KZ, Alto BW. Paternal and maternal effects in a mosquito: A bridge for life history transition. J Insect Physiol. 2021;131:104243. doi:10.1016/j.jinsphys.2021.104243
15. Macagno ALM, Zattara EE, Ezeakudo O, Moczek AP, Ledón-Rettig CC. Adaptive maternal behavioral plasticity and developmental programming mitigate the transgenerational effects of temperature in dung beetles. Oikos. 2018;127(9):1319-29. doi:10.1111/oik.05215
16. Tougeron K, Devogel M, van Baaren J, Le Lann C, Hance T. Trans-generational effects on diapause and life-history-traits of an aphid parasitoid. J Insect Physiol. 2020;121:104001. doi:10.1016/j.jinsphys.2019.104001
17. Reznik SY, Voinovich ND, Samartsev KG. Grandmaternal temperature effect on diapause induction in Trichogramma telengai (Hymenoptera: Trichogrammatidae). J Insect Physiol. 2020;124:104072. doi:10.1016/j.jinsphys.2020.104072
18. Bloch Qazi MC, Miller PB, Poeschel PM, Phan MH, Thayer JL, Medrano CL. Transgenerational effects of maternal and grandmaternal age on offspring viability and performance in Drosophila melanogaster. J Insect Physiol. 2017;100:43-52. doi:10.1016/j.jinsphys.2017.05.007
19. Mukai A, Mano G, Des Marteaux L, Shinada T, Goto SG. Juvenile hormone as a causal factor for maternal regulation of diapause in a wasp. Insect Biochem Mol Biol. 2022;144:103758. doi:10.1016/j.ibmb.2022.103758
20. Lockwood BL, Julick CR, Montooth KL. Maternal loading of a small heat shock protein increases embryo thermal tolerance in Drosophila melanogaster. J Exp Biol. 2017;220(23):4492-501. doi:10.1242/jeb.164848
21. Brevik K, Bueno EM, McKay S, Schoville SD, Chen YH. Insecticide exposure affects intergenerational patterns of DNA methylation in the Colorado potato beetle, Leptinotarsa decemlineata. Evol Appl. 2021;14(3):746-57. doi:10.1111/eva.13153
22. Zhang Y, Wang XX, Tian HG, Zhang ZF, Feng ZJ, Chen ZS, et al. The l-DOPA/dopamine pathway transgenerationally regulates cuticular melanization in the pea aphid Acyrthosiphon pisum. Front Cell Dev Biol. 2020;8:311. doi:10.3389/fcell.2020.00311
23. Mondotte JA, Gausson V, Frangeul L, Suzuki Y, Vazeille M, Mongelli V, et al. Evidence for long-lasting transgenerational antiviral immunity in insects. Cell Rep. 2020;33(11):108506. doi:10.1016/j.celrep.2020.108506
24. Dhinaut J, Chogne M, Moret Y. Trans-generational immune priming in the mealworm beetle protects eggs through pathogen-dependent mechanisms imposing no immediate fitness cost for the offspring. Dev Comp Immunol. 2018;79:105-12. doi:10.1016/j.dci.2017.10.017
25. Vilcinskas A. Mechanisms of transgenerational immune priming in insects. Dev Comp Immunol. 2021;124:104205. doi:10.1016/j.dci.2021.104205
26. Roesel CL, Rosengaus RB, Smith W, Vollmer SV. Transcriptomics reveals specific molecular mechanisms underlying transgenerational immunity in Manduca sexta. Ecol Evol. 2020;10(20):11251-61. doi:10.1002/ece3.6764
27. Cavieres G, Rezende EL, Clavijo-Baquet S, Alruiz JM, Rivera-Rebella C, Boher F, et al. Rapid within- and transgenerational changes in thermal tolerance and fitness in variable thermal landscapes. Ecol Evol. 2020;10(15):8105-13. doi:10.1002/ece3.6496
28. Castano-Sanz V, Gomez-Mestre I, Garcia-Gonzalez F. Evolutionary consequences of pesticide exposure include transgenerational plasticity and potential terminal investment transgenerational effects. Evolution. 2022;76(11):2649-68. doi:10.1111/evo.14613
29. Bagni T, Siaussat D, Maria A, Couzi P, Maïbèche M, Massot M. The impact of temperature on insecticide sensitivity depends on transgenerational effects. Sci Total Environ. 2022;851(Pt 1):158140. doi:10.1016/j.scitotenv.2022.158140

30. Wilson K, Grzywacz D, Cory JS, Donkersley P, Graham RI. Trans-generational viral transmission and immune priming are dose-dependent. J Anim Ecol. 2021;90(6):1560-9. doi:10.1111/1365-2656.13476
31. Gibbs M, Van Dyck H, Breuker CJ. Flight-induced transgenerational maternal effects influence butterfly offspring performance during times of drought. Oecologia. 2018;186(2):383-91. doi:10.1007/s00442-017-4030-1