Eurasia Specialized Veterinary Publication

Entomological Research Letters

ISSN: 3108-639X

2026, Volume 6, Issue 1, Page No: 1-10
Copyright CC BY-NC-SA 4.0
Available online at: www.esvpub.com/

Sequencing, Memory, and Interaction Among Stressors Determine Insect Life-History Responses to Compound Environmental Extremes

Peter Nilsson[1]*, Eva Johansson[1], Lars Andersson[2]

[1]Department of Stress Sequencing and Compound Extremes, Faculty of Life Sciences, Swedish University of Agricultural Sciences, Uppsala, Sweden.
[2]Department of Memory and Interaction Among Stressors, Faculty of Biosciences, Lund University, Lund, Sweden.

***E-mail** ✉ peter.nilsson@slu.se

ABSTRACT

Insects increasingly encounter environmental extremes as temporally structured sequences rather than isolated exposures. Yet compound-stress studies are often interpreted primarily from stressor identity and concurrent interaction, leaving exposure order, inter-event interval, retained physiological state, and recovery insufficiently integrated. This article develops a conceptual architecture for sequence-sensitive compound stress. The framework treats each exposure as an event acting on a biological state inherited from prior conditions, while separating persistent injury, acclimatory state, cross-generational carry-over, and recovery-dependent residual effects. Evidence from thermal extremes, pesticides, nutrition, infection, and ecological resource pathways shows that interaction direction can depend on genotype, sex, taxon, life stage, nutritional context, endpoint, and recovery history. The architecture therefore rejects the assumption that synergy or antagonism is an intrinsic property of a stressor pair. Instead, interaction is interpreted as conditional on the state in which the second event is received. The framework also distinguishes stressor duration from inter-event interval and survival from reproductive, developmental, immune, and behavioral consequences. Its central output is a proposed state-transition logic for organizing sequence-sensitive responses without implying a universal mechanism or predictive model. This structure identifies experimentally testable distinctions while preserving taxonomic, temporal, mechanistic, and laboratory-to-field boundaries.

Keywords: Compound environmental extremes, Exposure order, Physiological memory, Recovery dynamics, Multiple stressors, Insect life history.

Received: 22 December 2025
Revised: 24 February 2026
Accepted: 28 February 2026

How to Cite This Article: Nilsson P, Johansson E, Andersson L. Sequencing, Memory, and Interaction Among Stressors Determine Insect Life-History Responses to Compound Environmental Extremes. Entomol Appl Sci Lett. 2026;6(1):1-10.
https://doi.org/10.51847/vOQHBB9LPV

Introduction

Insects are exposed to climatic, chemical, nutritional, and biotic pressures that can co-occur or follow one another over ecologically relevant timescales. A recent synthesis of insect responses to global-change drivers found that interactive evidence remains much less developed than evidence for individual drivers [1]. Broader conservation synthesis likewise emphasizes that insect declines arise within environments shaped by multiple anthropogenic and climatic pressures rather than single isolated causes [2]. Field measurements in managed honey bees further show that stressor networks differ among crops and regions, demonstrating that the combinations experienced by organisms are context dependent even before biological interaction is inferred [3].

The central inferential problem is that a stressor set does not uniquely specify a biological exposure history. Heat followed by infection, pesticide exposure followed by drought, or repeated heat events separated by partial

© 2026 Entomological Research Letters

recovery can expose the same organism to nominally similar drivers while acting on different physiological states. Concurrent factorial designs can estimate departures from an additive or other reference expectation, but they do not by themselves resolve whether a prior event has altered the state in which a later event is received. This distinction matters because persistence may reflect reversible acclimation, unresolved injury, resource depletion, developmental transition, or cross-generational carry-over, each of which has different implications.

This article therefore proposes a sequence-sensitive interpretation of compound environmental extremes. The framework separates stressor identity, order, interval, physiological memory, interaction class, recovery, and life-history consequence. It does not assume that these dimensions share a universal mechanism or that laboratory responses predict population outcomes. Instead, it uses them as analytically separable coordinates for asking when two nominally identical stressor combinations should, and should not, be expected to produce equivalent biological responses.

Materials and Methods

This study is a non-empirical conceptual synthesis rather than a new experiment or quantitative meta-analysis. The architecture was constructed by assigning evidence to the specific temporal, physiological, ecological, or methodological distinction that it can support. A systematic review of 2396 freshwater multiple-stressor experiments shows that interaction studies differ substantially in experimental architecture and in how combined effects are interpreted [4]. This methodological heterogeneity motivates explicit separation of exposure structure from biological inference.

The proposed architecture represents exposure as a sequence of events acting on a changing organismal state. Each event is described by stressor identity, intensity where known, duration, developmental timing, and whether it occurs alone, simultaneously, or asynchronously relative to another event. Order is treated independently from total exposure because an asynchronous microcosm experiment showed that reversing heatwave and pesticide timing altered longer-term community responses and recovery [5]. That study involved zooplankton rather than insects and is used here only to justify the design principle that order requires explicit testing, not to infer insect physiology.

A second distinction separates the exposure phase from the recovery phase. In a freshwater mesocosm, insecticide effects under heatwave or elevated-temperature conditions differed across treatment and recovery periods and propagated through community and food-web responses [6]. Because that evidence is community-level and not insect-specific at every endpoint, the present framework uses it only to support temporal partitioning of observation. Accordingly, the architecture does not assign a fixed recovery coefficient or interaction class. It asks instead whether the biological state immediately before a subsequent event can be distinguished from both the original baseline and the state measured during the first event.

Results and Discussion

“Stressor identity” is more specific than an environmental label. Heat exposure, for example, can interact with water balance, and thermal injury may be confounded with dehydration when humidity, duration, and access to water are not considered [7]. The proposed architecture therefore distinguishes the nominal driver from the proximate load actually imposed on the organism whenever the evidence permits that distinction.

Biological identity also conditions compound effects. In Drosophila melanogaster, temperature and low-dose insecticide exposure produced a synergistic mortality response in a susceptible genotype but an additive response in a resistant genotype, with sex further modifying outcomes [8]. More broadly, evidence across temperature–insecticide, insecticide–nutrition, and nutrition–pesticide–virus experiments shows that the direction or magnitude of compound responses can depend on genotype, sex, pollinator taxon, or nutritional context [8–10]. Interaction class therefore cannot be treated as a stable property of two stressor names independently of the state and identity of the exposed organism.

The identity of a later challenge can also reveal carry-over from an earlier one. A simulated heatwave altered immune traits and subsequent resistance to infection in Bombus impatiens workers [11]. This finding supports a sequence-sensitive biological premise but not a universal immune mechanism: the measured response is restricted to a particular host, life stage, thermal regime, and infection context. Stressor identity must therefore include both the external driver and the response system through which that driver is evaluated.

Exposure order

Order becomes biologically meaningful when the first exposure changes the conditions under which the second operates. In a sequential study of the collembolan Folsomia candida, prior teflubenzuron exposure reduced tolerance to drought and altered aspects of cold response, whereas no corresponding interaction with heat was detected for the tested endpoints [12]. Collembola are not Insecta, so this result is retained only as a hexapod comparator demonstrating that sequential effects can be stressor specific rather than uniformly sensitizing.

Direct insect evidence shows that timing relative to development can alter the consequences of an extreme. In an aphid system, the timing and duration of heat events changed life-history and projected population responses [13]. The important inference is not that one chronological order is universally most damaging, but that order must be indexed to biological state. A heat event before reproduction, during juvenile development, or after maturation may constitute different sequences even when meteorological exposure is similar.

The proposed framework therefore treats A→B, B→A, simultaneous A+B, and repeated A→A histories as distinct exposure architectures. An observed order effect should not be attributed to "memory" unless cumulative dose, developmental progression, survivor selection, and incomplete repair remain plausible alternatives and are evaluated where possible.

Exposure interval

Exposure interval is not interchangeable with event duration. In Drosophila suzukii, thermal limits depended strongly on stress duration, and reproductive performance deteriorated under conditions that were less restrictive for survival [14]. In contrast, larval Osmia lignaria exposed to simulated heatwaves showed strong temperature effects on mortality and development, while the tested difference in event duration did not significantly alter mortality [15]. These results caution against assuming that longer exposure necessarily increases every endpoint monotonically.

Interval asks a different question: what biological processes occur after one event ends but before the next begins? In Tribolium castaneum, fertility and reproductive-tissue responses recovered over timescales that differed with life stage and endpoint after experimental heat exposure [16]. Those recovery periods are not transferable as universal insect values, but they establish that the same elapsed interval can represent incomplete recovery for one trait and substantial recovery for another.

Accordingly, the proposed architecture defines interval relative to the recovery kinetics of the state variable under study rather than by clock time alone. A short interval may preserve residual injury or inducible protection; a longer interval may permit repair, developmental transition, or decay of acclimatory state. This formulation makes interval mechanistically agnostic: it specifies what must be measured without presuming why the state persists.

Physiological memory

Physiological memory is used here narrowly to mean that a prior exposure leaves a retained biological state that biases a later response after the initiating conditions have changed. This definition excludes persistence alone. Lasting tissue injury, selective mortality, irreversible developmental change, and genetic adaptation can all produce history dependence without constituting physiological memory in this sense.

Across heat-acclimation, comparative thermal-sensitivity, and transgenerational host–parasitoid experiments, prior thermal environments left measurable effects on later or descendant responses [17–19]. In the rice leaffolder Cnaphalocrocis medinalis, multigenerational heat acclimation was associated with persistent molecular and physiological differences and enhanced subsequent heat response after the initiating thermal conditions were removed, although the effect decayed across recovery generations [17]. Warm acclimation also reduced modeled sensitivity to ecologically relevant heat exposure across four Drosophila species, with the magnitude varying among species [18]. Transgenerational thermal effects in a Drosophila host–parasitoid system altered later ecological vulnerability, showing that environmental history can propagate beyond a single individual's immediate response [19]. None of these observations establishes a universal memory mechanism or guarantees that persistence is beneficial under another environmental sequence.

Cross-generational persistence requires an additional boundary. In Culex pipiens, parental warming and pesticide treatments modified offspring responses [20]. Such carry-over cannot automatically be equated with genetic adaptation, stable epigenetic inheritance, or beneficial acclimation. Parental condition, developmental carry-over, selection among exposed individuals, and other mechanisms can produce similar descendant patterns unless they are experimentally separated.

The proposed framework consequently separates four forms of history dependence: retained acclimatory state, persistent damage state, recovery-dependent residual state, and cross-generational carry-over. Physiological memory sensu stricto requires evidence that a retained state changes a later response; persistence alone is insufficient. Developmental-state shifts and survivor selection are retained as competing explanations because both can create apparent tolerance or sensitization without demonstrating within-individual memory.

Because these states can generate superficially similar later phenotypes while requiring different biological interpretations and experiments, the distinctions are compressed in **Table 1** rather than treated as interchangeable manifestations of "memory."

Table 1. Physiological history states that can mimic or generate memory-dependent responses to sequential environmental extremes

Recorded evidence	Biological context	Interpretation for a lat within its limit	Discriminating check
Retained acclimatory state			
Evidence-supported category; placement within the sequence architecture is proposed			
Prior exposure is followed by altered later sensitivity after initiating conditions change	Within-individual or experimentally retained thermal state	Earlier exposure modifies the state in which the next event is received Benefit may be species-, environment-, or endpoint-specific, and persistence can decay	Measure state after exposure and before re-challenge; compare contrasting exposure histories
Persistent damage state			
Proposed interpretive distinction			
Impairment remains after the first event	Reproductive, cellular, developmental, or physiological injury	Later vulnerability may reflect unresolved injury rather than memory Damage can mimic sensitization; persistence alone does not demonstrate response bias	Measure injury directly and test whether second-event response exceeds consequences of residual damage
Recovery-dependent residual state			
Evidence-supported recovery pattern; state classification is proposed			
State variable moves toward baseline during the inter-event interval	Traits recovering at different rates between events	Biological coupling between events depends on how much recovery has occurred Different traits may recover on different timescales; elapsed time alone does not establish recovery	Sample multiple recovery points and multiple endpoints
Cross-generational carry-over			
Evidence-supported category; integration into the state architecture is proposed			
Descendant phenotype differs according to exposure history	Parental or ancestral environmental exposure	A later-generation event is received in a history-conditioned state Does not by itself establish genetic adaptation, stable epigenetic inheritance, or fitness benefit	Separate parental condition, selection, developmental effects, and heritable mechanisms where feasible
Developmental-state shift			
Proposed failure-mode category			
Later response differs because the organism is no longer in the same developmental state	Exposure sequence crosses maturation or another stage transition	Apparent memory may instead reflect ontogenetic change Chronological interval and biological transition become confounded	Compare matched developmental states or explicitly incorporate stage transition
Survivor-selection state			
Proposed failure-mode category			
Remaining cohort appears more tolerant during later exposure	First event disproportionately removes susceptible individuals	Apparent protection can arise without within-individual acclimation Population composition changes between events	Track individuals or account for selective mortality before re-challenge

Synergy and antagonism

Synergy and antagonism are not treated here as inherent attributes of a stressor pair. They are interpretations relative to an explicit reference expectation and can change with biological state, endpoint, or sequence. Repeated heat exposure in male Drosophila melanogaster produced stage-dependent fertility loss and recovery dynamics [21]. Such accumulation demonstrates history dependence, but it should not be called synergistic unless the combined response departs from a defined additive or other justified expectation.

Compound responses can also emerge through more than one pathway. Experimental heatwaves altered bumblebee foraging both through direct effects on bees and through reduced floral nectar production [22]. A response attributed to "heat" may therefore combine organismal exposure with stress-induced changes in resources or ecological partners. Conversely, behavioral compensation can buffer one endpoint without protecting another: thermoregulatory behavior in a plant-feeding insect enhanced survival but did not preserve reproductive performance to the same extent [23].

The proposed framework consequently classifies interaction only after specifying the receiving state, comparator, pathway, and endpoint. Apparent synergy may represent unresolved injury carried into a second event; apparent antagonism may reflect acclimation or behavioral buffering; and an unchanged survival response may coexist with substantial reproductive cost. These alternatives are analytically distinct because they imply different mechanisms and validation experiments.

The resulting logic requires a visual representation that keeps exposure history, retained state, interaction interpretation, and life-history endpoint separate while preserving the cross-links among them. **Figure 1** therefore compresses the decision boundaries that determine when a compound response may be interpreted as state-conditioned synergy, buffering, cumulative injury, or an unresolved alternative.

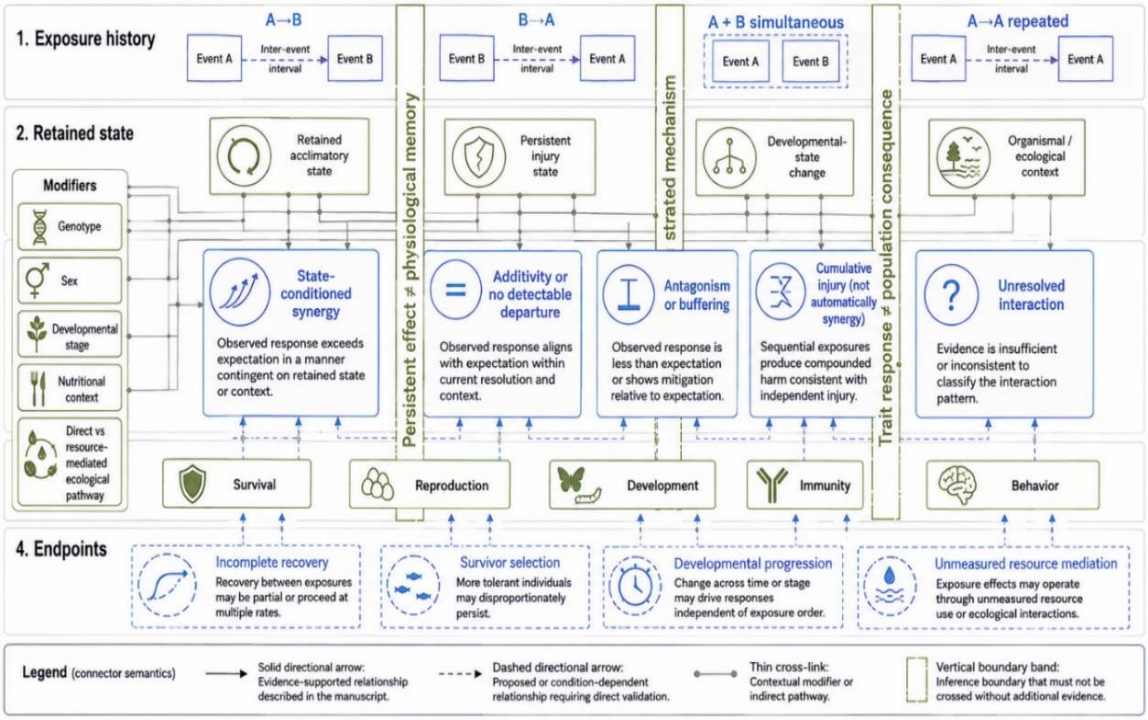


Figure 1. State-conditioned interaction field separating synergy, buffering, cumulative injury, and endpoint divergence during compound environmental extremes

Recovery between events

Recovery is not equivalent to the absence of exposure. It is a state transition whose rate and completeness depend on the trait being measured. Successive experimental heatwaves in Tribolium castaneum substantially exacerbated male reproductive impairment and were associated with sperm damage and offspring effects [24]. This supports cumulative reproductive injury when a new event arrives before relevant processes have returned to baseline, but it does not establish that every repeated event is synergistic or that transgenerational effects share one mechanism.

Endpoint dependence also complicates recovery. Temperature changed pesticide–behavior relationships in bees, but the direction and magnitude differed among behaviors and treatments [25]. More broadly, insect heat responses involve heterogeneous physiological and behavioral mechanisms rather than one canonical recovery pathway [26]. A single "recovered/not recovered" classification is therefore insufficient when survival, fertility, cognition, immunity, or behavior return toward baseline at different rates.

The proposed architecture treats recovery as event-specific and trait-specific. A later stressor can arrive during persistent damage, partial repair, retained acclimation, or an apparently restored state in which an unmeasured process remains altered. **Table 2** distinguishes these recovery states because their biological consequences and experimental tests differ even when the elapsed interval is identical.

Table 2. Recovery states that determine whether sequential extremes remain biologically coupled

Biological context, observable basis, and consequence for the next event	Recovery-between-events with evidential standing	Interpretive limit and decisive check
Reproductive, cellular, or physiological damage **Measured impairment remains before re-exposure** **Later event acts on residual damage and may intensify loss**	Incomplete injury repair Evidence-supported pattern; state label proposed	Accumulation can mimic synergy Measure damage immediately before each event and compare with single-event controls
Survival, fertility, behavior, immunity, or development recover at different rates **One endpoint returns toward baseline while another remains altered** **Sequence may appear resolved for one trait but coupled for another**	Trait-specific recovery Evidence-supported pattern; integration proposed	Endpoint choice can reverse interpretation Follow multiple traits across the same interval
Prior exposure leaves reversible tolerance or altered sensitivity **Later response differs despite removal of initiating condition** **Subsequent stress may be buffered**	Retained acclimatory protection Evidence-supported category; sequence role proposed	Protection may decay or become costly in another context Re-challenge across several recovery intervals
Food, host, floral, or microhabitat conditions change after the first extreme **External resource state differs before the next exposure** **Organismal response reflects both internal and environmental recovery**	Ecological-resource recovery Evidence-supported pathway; classification proposed	Resource mediation may be misattributed to physiology Measure environmental and organismal states in parallel
Coarse endpoint returns to baseline but another state variable is unmeasured **No obvious deficit in the chosen endpoint** **Later vulnerability or protection may still differ**	Apparent recovery with hidden state Proposed failure-mode category	False recovery caused by insensitive measurement Pair terminal outcomes with intermediate state measurements

Life-history consequences

The consequences of environmental extremes extend across levels from physiology and behavior to development and reproduction, and their directions are not uniformly negative. Climatic extremes can generate both population outbreaks and breakdowns depending on species interactions, life history, and environmental context [27]. The sequence framework therefore avoids compressing all endpoints into a single "stress severity" axis.

Post-event conditions can also modify carry-over. In Lobesia botrana, developmental stage and post-stress food quality influenced responses to heatwave exposure, showing that recovery environment can alter later developmental and immune consequences [28]. Acute heat exposure in Bombus terrestris impaired learning and short-term memory under laboratory conditions [29], demonstrating that sublethal behavioral functions may be affected even when demographic consequences remain unmeasured.

Accordingly, survival, reproduction, development, immunity, cognition, and behavior are retained as separate outcome channels. Trait change is not automatically interpreted as a fitness or population effect. Higher-level

consequences require direct demographic, field, or interaction evidence rather than extrapolation from a single organismal endpoint.

Sequence-sensitive compound-stress model

The central synthesis is a proposed state-transition model in which the response to event n depends on both the incoming event and the biological state inherited from prior history. Conceptually, response can be written as $R_n = f(E_n, S_{n-1}, \text{interval}, \text{context}, \text{endpoint})$, where E_n denotes the current exposure and S_{n-1} denotes the measurable pre-event state. This notation is organizational, not a fitted equation, and it assigns no universal coefficients or thresholds.

The pre-event state cannot be assumed identical across populations. Comparative work on Mediterranean water beetles shows intraspecific and interspecific variation in thermal tolerance associated with climatic background [30]. Likewise, climate-change evidence for honey bees is geographically and methodologically uneven, limiting transfer from well-studied regions and systems [31]. Developmental stage and sex further alter reproductive sensitivity to heat in Drosophila melanogaster [32]. Baseline state must therefore be parameterized only with variables justified for the taxon and endpoint under study.

Within the proposed model, the first event transforms S_0 into S_1; the interval then allows persistence, decay, repair, developmental transition, or ecological-resource change; the next event acts on that altered state. A sequence effect is inferred only when histories with matched current exposure produce different responses and plausible alternatives such as cumulative dose, selective mortality, or stage progression are considered. Physiological memory is one possible state-transition mechanism, not a default explanation.

The model generates testable contrasts rather than predictions with established accuracy. Informative experiments should compare A→B, B→A, simultaneous A+B, repeated A→A, and relevant single-stressor controls while manipulating the inter-event interval. Intermediate measurements are required to determine whether the state before the second exposure reflects damage, acclimation, recovery, or another process. Replication across life stages, sexes, populations, and field-relevant environments is necessary before transferability can be claimed.

This framework shifts compound-extreme interpretation from stressor combinations to exposure histories. The distinction is important because multiple-stressor research varies substantially in design [4], while insect studies demonstrate that developmental timing [13], recovery dynamics [16], retained heat-acclimated states [17], and repeated-event reproductive injury [21] can all change the biological meaning of a later exposure. These observations support sequence sensitivity as a plausible organizing principle, but they do not validate one universal mechanism.

The proposed concept of physiological memory is intentionally restrictive. A later response must be biased by a retained state after the initiating exposure has changed; persistent impairment alone is insufficient. This boundary prevents unresolved tissue damage, survivor selection, developmental change, and cross-generational environmental effects from being collapsed into a single memory category. It also avoids treating transgenerational persistence as evidence of genetic adaptation without direct inheritance and selection evidence.

Interaction terminology requires similar discipline. Genotype can change an apparent temperature–insecticide response from synergistic to additive [8], while behavioral thermoregulation can protect survival without equivalently protecting reproduction [23]. Thus synergy, antagonism, and buffering should be assigned to a specified comparator and endpoint rather than to the stressor pair itself. Reproductive limits can reveal vulnerability missed by survival-based measures across experimental and comparative thermal studies [14, 23, 33]. Parratt et al. showed that male-sterilizing temperatures corresponded with Drosophila species distributions more closely than lethal limits [33], but this association does not establish sterility as the sole determinant of geographic range.

The framework also clarifies why interval matters. Clock time is biologically informative only relative to processes occurring during that interval. If reproductive tissue remains damaged, a second event arrives during incomplete recovery; if inducible protection persists, it arrives during acclimation; if a stage transition occurs, it reaches a different developmental system. Similar intervals can therefore connect events strongly for one trait and weakly for another.

Several boundaries remain substantial. Most direct sequence evidence comes from laboratory or semi-natural systems and a limited taxonomic subset. Field stressor networks in honey bees are region- and crop-dependent [3], and broader multiple-stressor designs remain heterogeneous [4]. Mechanisms inferred from Drosophila, Tribolium, bees, aphids, or mosquitoes should not be transferred across Insecta without comparative validation.

Non-insect comparators used to motivate asynchronous design likewise provide methodological logic rather than insect-specific biological evidence.

The proposed model is therefore best regarded as a hypothesis-generating architecture. Its strongest tests will manipulate order and interval independently, measure the state between events, follow multiple life-history endpoints, and distinguish within-individual persistence from developmental, ecological, and cross-generational alternatives. Such experiments would determine when sequence adds explanatory information beyond stressor identity and cumulative exposure, and when a simpler dose-based interpretation is sufficient.

Conclusion

Compound environmental extremes are biologically defined not only by which stressors occur, but by when they occur, what state remains from previous exposure, and how much recovery is possible before the next event. The proposed sequence-sensitive architecture separates stressor identity, order, interval, physiological memory, interaction, recovery, and endpoint-specific consequence so that these dimensions are not conflated. It treats synergy and antagonism as conditional interpretations, physiological memory as a narrowly defined retained-state effect, and recovery as a trait-specific transition rather than an exposure-free period. The framework does not claim universal mechanisms or predictive performance. Its value is to make alternative explanations explicit and to specify experiments capable of distinguishing cumulative damage, acclimation, developmental change, ecological mediation, and genuine history-dependent response.

Acknowledgments: None

Conflict of Interest: None

Financial Support: None

Ethics Statement: None

References

1. Vidal MC, Abarca M, Backe K, Curé AE, Finke DL, Koltz AM, et al. What do we know about insect responses to global change? A review of meta-analyses on global change drivers. Insect Conserv Divers. 2025;18(6):900-16. doi:10.1111/icad.12845
2. Farooq M, Khan U, Adnan M, Younas W, Qureshi NB, Yasir QM, et al. Global decline of aquatic and terrestrial insects driven by climate change and anthropogenic impacts: The interaction of multiple stressors and disruption of niche conservatism. Biol Conserv. 2025;308:111181. doi:10.1016/j.biocon.2025.111181
3. French SK, Pepinelli M, Conflitti IM, Jamieson A, Higo H, Common J, et al. Honey bee stressor networks are complex and dependent on crop and region. Curr Biol. 2024;34(9):1893-903.e3. doi:10.1016/j.cub.2024.03.039
4. Orr JA, Macaulay SJ, Mordente A, Burgess B, Albini D, Hunn JG, et al. Studying interactions among anthropogenic stressors in freshwater ecosystems: A systematic review of 2396 multiple-stressor experiments. Ecol Lett. 2024;27(6):e14463. doi:10.1111/ele.14463
5. García-Astillero A, Polazzo F, Rico A. Combined effects of heat waves and pesticide pollution on zooplankton communities: Does the timing of stressor matter? Ecotoxicol Environ Saf. 2024;282:116751. doi:10.1016/j.ecoenv.2024.116751
6. Hermann M, Polazzo F, Cherta L, Crettaz-Minaglia M, García-Astillero A, Peeters ETHM, et al. Combined stress of an insecticide and heatwaves or elevated temperature induce community and food web effects in a Mediterranean freshwater ecosystem. Water Res. 2024;260:121903. doi:10.1016/j.watres.2024.121903
7. Sinclair BJ, Saruhashi S, Terblanche JS. Integrating water balance mechanisms into predictions of insect responses to climate change. J Exp Biol. 2024;227(10):jeb247167. doi:10.1242/jeb.247167
8. Martelli F, Alves AN, Yang YT, Batterham P, Wedell N. Genotype and sex affect the combined impact of temperature and low-dose insecticide exposure on insect survival. Insect Sci. 2025;32(6):2079-91. doi:10.1111/1744-7917.13475

9. Birkenbach M, Straub F, Weihermüller P, Wilfert L, Ayasse M, Kuppler J. Insecticide and low food quality treatments reduce health and pollination services of two key pollinator taxa. J Appl Ecol. 2024;61(11):2786-97. doi:10.1111/1365-2664.14788
10. Hsieh EM, Dolezal AG. Nutrition, pesticide exposure, and virus infection interact to produce context-dependent effects in honey bees (Apis mellifera). Sci Total Environ. 2024;949:175125. doi:10.1016/j.scitotenv.2024.175125
11. Tobin KB, Mandes R, Martinez A, Sadd BM. A simulated natural heatwave perturbs bumblebee immunity and resistance to infection. J Anim Ecol. 2024;93(2):171-82. doi:10.1111/1365-2656.14041
12. Xie L, Slotsbo S, Damgaard C, Holmstrup M. Exposure to teflubenzuron reduces drought tolerance of collembolans. Chemosphere. 2024;361:142448. doi:10.1016/j.chemosphere.2024.142448
13. Zhao F, Xing K, Hoffmann AA, Ma CS. The importance of timing of heat events for predicting the dynamics of aphid pest populations. Pest Manag Sci. 2019;75(7):1866-74. doi:10.1002/ps.5344
14. Ørsted M, Willot Q, Olsen AK, Kongsgaard V, Overgaard J. Thermal limits of survival and reproduction depend on stress duration: A case study of Drosophila suzukii. Ecol Lett. 2024;27(3):e14421. doi:10.1111/ele.14421
15. Melone GG, Stuligross C, Williams NM. Heatwaves increase larval mortality and delay development of a solitary bee. Ecol Entomol. 2024;49(3):433-44. doi:10.1111/een.13317
16. Sales K, Vasudeva R, Gage MJG. Fertility and mortality impacts of thermal stress from experimental heatwaves on different life stages and their recovery in a model insect. R Soc Open Sci. 2021;8(3):201717. doi:10.1098/rsos.201717
17. Quan PQ, Guo PL, He J, Liu XD. Heat-stress memory enhances the acclimation of a migratory insect pest to global warming. Mol Ecol. 2024;33(17):e17493. doi:10.1111/mec.17493
18. Baeza Icaza A, Poblete Ahumada G, Rezende EL, Peralta-Maraver I. Warm acclimation reduces the sensitivity of Drosophila species to heat stress at ecologically relevant scales. J Anim Ecol. 2025;94(5):896-907. doi:10.1111/1365-2656.70018
19. Bright NL, Chen J, Terry JCD. Transgenerational effects increase the vulnerability of a host-parasitoid system to rising temperatures. J Anim Ecol. 2025;94(10):2089-102. doi:10.1111/1365-2656.70112
20. 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
21. Meena A, De Nardo AN, Maggu K, Sbilordo SH, Roy J, Snook RR, et al. Fertility loss and recovery dynamics after repeated heat stress across life stages in male Drosophila melanogaster: Patterns and processes. R Soc Open Sci. 2024;11(10):241082. doi:10.1098/rsos.241082
22. Hemberger JA, Rosenberger NM, Williams NM. Experimental heatwaves disrupt bumblebee foraging through direct heat effects and reduced nectar production. Funct Ecol. 2023;37(3):591-601. doi:10.1111/1365-2435.14241
23. Leith NT, Miller EA, Fowler-Finn KD. Thermoregulation enhances survival but not reproduction in a plant-feeding insect. Funct Ecol. 2024;38(6):1344-56. doi:10.1111/1365-2435.14546
24. Sales K, Vasudeva R, Dickinson ME, Godwin JL, Lumley AJ, Michalczyk Ł, et al. Experimental heatwaves compromise sperm function and cause transgenerational damage in a model insect. Nat Commun. 2018;9(1):4771. doi:10.1038/s41467-018-07273-z
25. Kenna D, Graystock P, Gill RJ. Toxic temperatures: Bee behaviours exhibit divergent pesticide toxicity relationships with warming. Glob Chang Biol. 2023;29(11):2981-98. doi:10.1111/gcb.16671
26. González-Tokman D, Córdoba-Aguilar A, Dáttilo W, Lira-Noriega A, Sánchez-Guillén RA, Villalobos F. Insect responses to heat: Physiological mechanisms, evolution and ecological implications in a warming world. Biol Rev Camb Philos Soc. 2020;95(3):802-21. doi:10.1111/brv.12588
27. Harvey JA, Heinen R, Gols R, Thakur MP. Climate change-mediated temperature extremes and insects: From outbreaks to breakdowns. Glob Chang Biol. 2020;26(12):6685-701. doi:10.1111/gcb.15377
28. Iltis C, Louâpre P, Vogelweith F, Thiéry D, Moreau J. How to stand the heat? Post-stress nutrition and developmental stage determine insect response to a heat wave. J Insect Physiol. 2021;131:104214. doi:10.1016/j.jinsphys.2021.104214
29. Gérard M, Amiri A, Cariou B, Baird E. Short-term exposure to heatwave-like temperatures affects learning and memory in bumblebees. Glob Chang Biol. 2022;28(14):4251-9. doi:10.1111/gcb.16196

30. Pallarés S, Garoffolo D, Rodríguez B, Sánchez-Fernández D. Role of climatic variability in shaping intraspecific variation of thermal tolerance in Mediterranean water beetles. Insect Sci. 2024;31(1):285-98. doi:10.1111/1744-7917.13241
31. Zapata-Hernández G, Gajardo-Rojas M, Calderón-Seguel M, Muñoz AA, Yáñez KP, Requier F, et al. Advances and knowledge gaps on climate change impacts on honey bees and beekeeping: A systematic review. Glob Chang Biol. 2024;30(3):e17219. doi:10.1111/gcb.17219
32. Meena A, Maggu K, De Nardo AN, Sbilordo SH, Eggs B, Al Toma Sho R, et al. Life stage-specific effects of heat stress on spermatogenesis and oogenesis in Drosophila melanogaster. J Therm Biol. 2024;125:104001. doi:10.1016/j.jtherbio.2024.104001
33. Parratt SR, Walsh BS, Metelmann S, White N, Manser A, Bretman AJ, et al. Temperatures that sterilize males better match global species distributions than lethal temperatures. Nat Clim Chang. 2021;11(6):481-4. doi:10.1038/s41558-021-01047-0