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Opinion

Transcending Global Change Biology Disciplinary Boundaries through Ecophysiotoxicology

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Abstract: This paper proposes the unification of ecophysiology and ecotoxicology into a novel research field, ecophysiotoxicology, underpinned by hormesis as a broadly generalizable biological principle through which low-dose stress commonly activates adaptive, compensatory, or protective responses, whereas higher doses cause inhibition or damage. Here, an adaptive response denotes an inducible biological adjustment and is not synonymous with heritable evolutionary adaptation or an unconditional net fitness benefit. The integration combines mechanistic tools from ecophysiology, including stressor-induced molecular, metabolic, and physiological adjustments, with ecological scaling and dose-response tools from ecotoxicology to predict multistressor effects across biological hierarchies. Hormesis provides the new theoretical bridge, placing natural and anthropogenic stressors on a shared dose-dependent continuum, while the two disciplines contribute complementary explanatory and predictive strengths. The synthesis addresses critical gaps in global change biology by incorporating stressor dissimilarity, exposure timing, eco-evolutionary dynamics, and explicit translation from mechanisms to population, community, and ecosystem outcomes. A concise workflow and two applied scenarios show how the proposed field is intended to support testable predictions, strengthen risk assessment, and evaluate priming or resilience-enhancing interventions without assuming that every stimulatory response is beneficial at every biological level.

Keywords: ecophysiology; ecotoxicology; hormesis; global change biology; multiple stressors; dose response; ecological scaling

1. Introduction

Ecophysiology and ecotoxicology are intrinsically linked disciplines. Ecophysiology is the study of how organismal physiological processes (e.g., respiration, photosynthesis, water balance, thermoregulation) are adapted to and influenced by their environment. Ecotoxicology is the study of toxic chemical effects on living organisms, especially at the levels of population, community, and ecosystem. In essence, their greatest strengths are complementary as ecophysiology supplies mechanistic tools for resolving stressor-induced molecular, metabolic, and physiological adjustments, whereas ecotoxicology supplies dose-response methods and ecological scaling to predict how such responses propagate across biological hierarchies. Together, these fields serve as pillars of global change biology. Global change biology defines the problem, namely the multifaceted, large-scale alterations to the Earth system, while ecophysiology and ecotoxicology provide the tools and concepts to diagnose and understand its biological effects. Global change biology relies on ecophysiological principles to understand and predict how species respond to changing abiotic drivers. In relation to ecotoxicology, global change biology

recognizes pollution as a primary driver of global change (e.g., nitrogen deposition and chemical pollution), while climate change can modify toxicant effects. Ecophysiology provides mechanistic explanations for effects that ecotoxicology measures and scales, all within the framework of global change biology. For example, climate warming and atmospheric mercury (Hg) deposition may occur together. Ecotoxicology asks how warming affects Hg bioaccumulation in fish. Ecophysiology provides mechanistic answers: warmer temperatures can increase metabolic rate and food consumption, thereby increasing Hg uptake, while thermal stress may reduce detoxification efficiency. Global change biology then synthesizes how a physical driver and a chemical pollutant jointly alter ecological risk, food-web dynamics, and contaminant transfer to top predators. Such an integrated assessment is valuable for research, conservation, and policy.

Global change biology poses complex, multistressor questions that cannot be answered by any single discipline. Ecotoxicology and ecophysiology are fundamental sub-disciplines within its umbrella, providing the mechanistic and predictive evidence needed to move from observing change to explaining it and forecasting its consequences. An ecotoxicologist or ecophysiologist may work entirely within the conventional boundaries of the respective field when the question does not concern global change. When either discipline addresses global-change questions, however, it contributes an essential part of a larger causal problem. Global change biology can therefore be viewed as a meta-discipline organized around a grand challenge, creating a clear imperative to integrate ecophysiology and ecotoxicology.

2. Ecophysiology versus Ecotoxicology: Where Global Change Comes

The shared foundation of the two fields is that both are sub-disciplines of ecology, with a common goal of understanding how organisms interact with their environment and respond to environmental stressors using field and laboratory approaches. Their traditional distinction concerned the nature of the stressor, as ecotoxicology focused primarily on anthropogenic chemical stressors, whereas ecophysiology focused mainly on natural physical and chemical stressors. Their central lines of inquiry were also assumed to differ: ecotoxicology emphasized harm and disruption from toxicants, often in support of policy and conservation, while ecophysiology emphasized the mechanisms of acclimation, adaptation, survival, and resilience under natural stressors. Scientific developments, especially during the past decade, now make this categorical separation increasingly difficult to sustain.

Evidence accumulated across toxicology, medicine, plant biology, entomology, microbiology, and ecology shows that hormesis is elicited by remarkably diverse stressor classes—from botanicals and other phytochemicals to pesticides, metals, radiation, and carcinogens—and across diverse organisms. Hormesis is therefore stressor-general, and its expression depends on dose, timing, prior exposure, endpoint, and biological condition [1–9]. Hormesis is an evolutionarily conserved and highly generalizable biological feature that facilitates stress adaptation in the global biosphere [7]. In the stress-biology and toxicological sense used here, an adaptive response is an induced compensatory or protective adjustment that increases the capacity of an organism to maintain function or withstand a subsequent challenge; it should not be confused with heritable evolutionary adaptation, nor does it guarantee a net benefit for every trait or level of organization [3]. An ecotoxicologist working with a toxicant may therefore observe adaptive responses when exposure lies within the low-dose region, as increasingly reported [8–14]. Conversely, an ecophysiologist interested in adaptive mechanisms may examine hormetic responses to anthropogenic chemicals [15,16]. The mechanisms underlying stimulatory and adaptive responses to anthropogenic chemicals overlap with those induced by natural chemicals and physical global-change factors such as heat, drought, and radiation [7,17–21]. Hormesis therefore challenges the traditional stressor boundary and provides a powerful theoretical bridge for unifying ecophysiology and ecotoxicology (Figure 1).

Different global-change factors can affect particular forms of life (e.g., plants) at different levels of the ecological hierarchy, and numerous factors act concurrently [22,23]. Their effects may propagate upward through the hierarchy, but buffering and compensatory mechanisms can attenuate that propagation. Concurrent buffering across spatial and temporal scales can diminish stressor impacts, while synergistic interactions among buffering processes may further increase the capacity of an ecosystem to withstand multiple co-occurring stressors [22]. The effects of multiple global-change factors therefore cannot be predicted reliably from single-factor effects. Nevertheless, approximately 80% of global-change studies examine one factor, 19% examine two, and fewer than 2% examine three or four [24]. Laboratory experiments show that an increasing number of concurrent factors (up to 10) produces progressively larger directional shifts in soil properties, processes, and microbial communities [24]. An increasing number of anthropogenic stressors, from pesticides to microplastics, can also amplify the damaging effects of climate change on ecosystem functioning [25]. Likewise, the positive contribution of high soil microbial diversity to ecosystem functioning under zero to four factors disappears when many factors act simultaneously, partly through reductions in fungal abundance and in a core cluster of interdependent bacterial and fungal taxa [26]. However,

stressor quality is as important as stressor number, because the cocktail effect may worsen as stressors become more dissimilar (or diverse), producing unexpectedly strong synergistic declines of microarthropod populations and losses of soil functions [27,28]. Both the number and dissimilarity of stressors are therefore essential to global change and environmental risk assessment, and integrating ecophysiology and ecotoxicology can help to clarify how these two dimensions jointly shape biological responses.

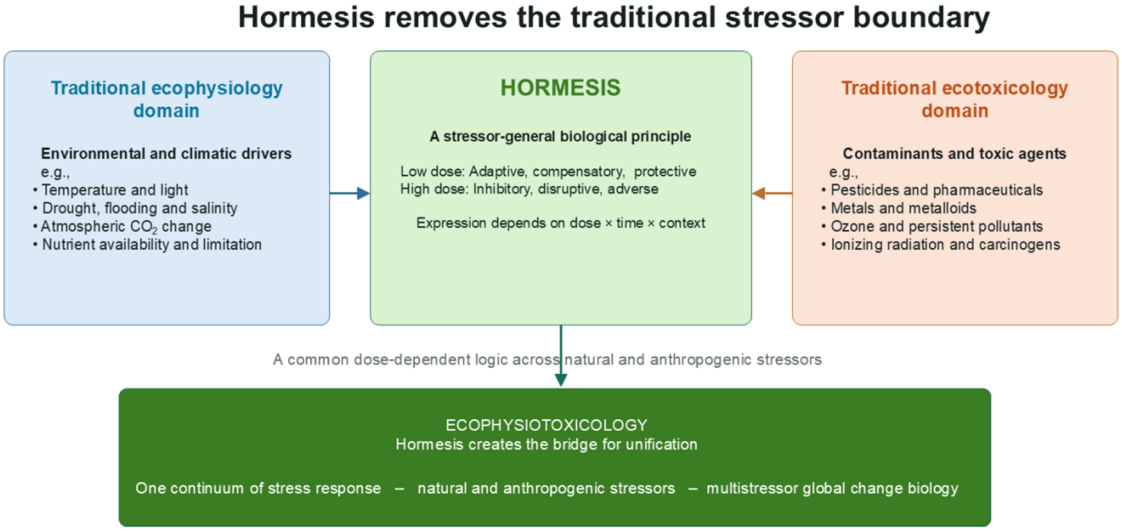

Figure 1. Hormesis as the theoretical bridge between ecophysiology and ecotoxicology. Natural and anthropogenic stressors are placed on a shared dose-dependent continuum. Across diverse stressor classes, low doses can activate compensatory, protective, or adaptive responses, whereas higher doses cause inhibition or adversity. This stressor-general logic dissolves the traditional categorical boundary without implying that every exposure or endpoint must display hormesis. Note that some agents such as ozone are studied by both fields, an overlap that further supports the argument that hormesis supplies a shared dose-response logic.

Ecotoxicologists increasingly examine chemical-mixture effects in the context of other global-change drivers, including acidity, heat, salinity, and soil properties [10,11,29–34]. Recent evidence also indicates that hormetic responses to anthropogenic contaminants can antagonize adverse effects of physical stressors; for example, low-dose pesticide exposure can counteract effects of a heat spike [10]. Ecotoxicology can therefore be strengthened by ecophysiological knowledge, as illustrated by ozone-flux metrics that incorporate detoxification in the apoplast [35]. Conversely, ecophysiology and ecology can benefit from ecotoxicological tools, including nonlinear dose-response and mixture models [13,36–39]. For example, many ecophysiologists studying ozone effects on vegetation already use ecotoxicological methods for risk assessment. Designs that include more doses, repeated observations, and multiple concurrent stressors also increase the likelihood of detecting hormesis, including responses that were not the original object of the experiment. Ecophysiologists and ecotoxicologists may thus be studying the same dose-dependent phenomena from different disciplinary starting points. The generality of hormesis suggests that ecotoxicology can be viewed as the ecophysiology of anthropogenic chemicals, and ecophysiology as the ecotoxicology of natural stressors. Unification makes that correspondence explicit and permits stressor dissimilarity and cross-hierarchy propagation to be addressed within one field.

The long-standing perception that natural and anthropogenic stressors belong to separate intellectual domains helped create a disciplinary chasm that has impeded scientific advancement [40]. Ecologists and biodiversity scientists are consequently being urged to treat chemical pollution as a global-change factor, while ecotoxicologists are being urged to adopt a more holistic, ecosystem-wide perspective [40]. At both local and global scales, the capacity of organisms to express hormetic adaptive responses provides a foundation for connecting these domains. Low-dose stress can stimulate protective adjustments across virtually all major classes of stressors, from localized environmental changes to planetary-scale drivers [1,4,15]. Global change biology, which spans the hierarchy from individuals to ecosystems, can therefore be examined through this integrative approach [23,41]. Developing a predictive science requires clarification of the mechanistic links between individual variation and adaptive behavior and the emergent properties of populations, communities, and ecosystems, especially under human intervention. Integrated Individual-Based Global Change Ecology was recently proposed to move beyond species-, population-, or community-level averages and recognize individual organisms as fundamental units of ecological processes [41]. Integrating ecophysiology's resolution of individual

variability with ecotoxicology's scaling of population, community, and ecosystem outcomes directly addresses this need.

2.1. Conceptual Boundaries and Main Innovation

Ecophysiotoxicology emerges beside (but not in place of) established neighboring areas. Environmental physiology and ecophysiology explain how environmental variation modifies organismal function, performance, and plasticity [42]. Conservation physiology applies physiological knowledge to conservation problems and management decisions [43]. Within ecotoxicology, physiological responses and biomarkers have long connected contaminant exposure with biological dysfunction [44]. These fields demonstrate that physiological and toxicological questions already overlap; overlap alone is therefore not the novelty claimed here.

The conceptual advance is the specific synthesis of four elements. First, hormesis serves as a formal, stressor-general bridge that places natural and anthropogenic stressors on the same dose-dependent continuum. Second, a mechanistic pillar from ecophysiology explains how molecular, metabolic, energetic, and physiological adjustments arise. Third, an ecological-scaling pillar from ecotoxicology predicts how these responses propagate (or fail to propagate) from organisms to populations, communities, and ecosystems. Fourth, the same architecture treats stressor number, dissimilarity, timing, and sequence as determinants of multistressor outcomes. Ecophysiotoxicology is thus defined not simply by interdisciplinary subject matter, but by hormesis-centered, cross-hierarchy prediction across natural and anthropogenic stressors (Figure 2).

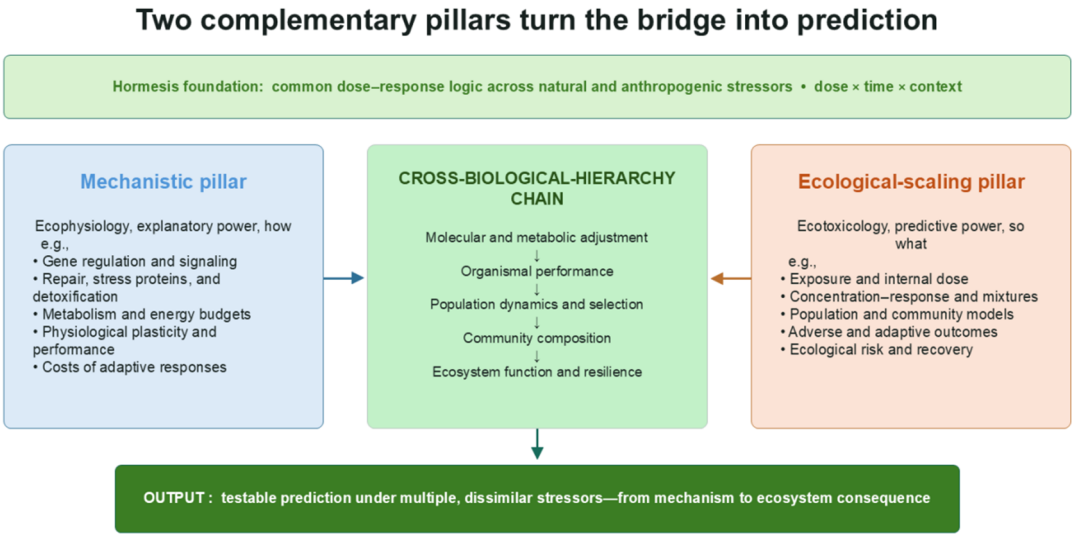

Figure 2. The two-pillar architecture of ecophysiotoxicology. The mechanistic pillar from ecophysiology provides explanatory power by resolving molecular, metabolic, energetic, and physiological adjustments. The ecological-scaling pillar from ecotoxicology contributes the dose-response and modeling tools used to link exposure and organismal responses to population, community, and ecosystem outcomes. Together, the two pillars provide a basis for developing and testing cross-hierarchy predictions. Hormesis supplies the common dose-response foundation across natural and anthropogenic stressors.

Three related terms require particular precision. Hormesis denotes a biphasic dose response with low-dose stimulation and high-dose inhibition relative to a stated reference [3]. Adaptive response denotes an inducible compensatory or protective adjustment [3,45]; evolutionary adaptation is reserved for heritable population change. Priming (or preconditioning) denotes an exposure-history effect in which an initial, usually mild, stress modifies the response to a later challenge [46–48]. A hormetic response may be adaptive in the first sense without increasing every component of fitness or every ecosystem function. This distinction answers a question of cross-scale interpretation without removing adaptation from the biological basis of hormesis.

2.2. Making Stressor Dissimilarity Explicit

The original concept of stressor dissimilarity can be operationalized without reducing it to a single chemical descriptor. Each stressor can be characterized along five domains: (1) physicochemical properties and exposure route; (2) dose, duration, timing, and bioavailability; (3) molecular initiating events or primary targets; (4) affected physiological pathways; and (5) standardized single-stressor response profiles. Mixed continuous and categorical

descriptors can be summarized using a prespecified distance such as Gower's distance, while retaining domain-specific distances to distinguish, for example, exposure dissimilarity from mechanistic dissimilarity [49]. This produces testable alternatives: similar stressors may converge on and saturate the same pathway, whereas dissimilar stressors may act independently or synergistically by affecting different processes that share a finite energy budget. Full factorial or response-surface designs can then test whether interaction strength changes with dissimilarity instead of invoking a cocktail effect after the outcome is known.

3. Ecophysiotoxicology Can Advance Global Change Biology

Building on the definition above, the proposed synthesis treats hormesis and adaptive response as principles that challenge the traditional boundary between ecophysiology and ecotoxicology. If hormesis is accepted as a general biological principle [2], the fundamental question is not whether an agent is intrinsically a toxicant, but how a biological system responds to that agent across dose, time, and context. The complementary strengths of the two fields then become essential components of the unified discipline. Ecophysiology supplies mechanistic tools for explaining hormetic responses, including repair-enzyme induction, stress-protein synthesis, immune activation, gene regulation, and metabolic reorganization. Ecotoxicology supplies the exposure framework, ecological context, and forecasting models needed to predict how these responses propagate through populations, communities, and ecosystems under multiple stressors. Environmental hormesis has been examined not only at sub-individual and individual levels but also in relation to population dynamics and communities [4,50–54], and its relevance to ecosystem responses under global change is increasingly recognized [55,56]. The same higher-level expression has also been described as ecosystem hormesis nearly 30 years ago, or ecohormesis recently, but it remains part of the broader environmental-hormesis framework [4,50,56,57].

The central purpose of ecophysiotoxicology is to create a predictive understanding of how ecological systems respond to environmental stress by integrating mechanisms across all levels of biological organization, from molecular to ecosystem scales. It is guided by the hormetic framework, used here as an empirical biphasic dose-response pattern rather than as a new universal numerical model [3,50]. The relevant parameters and scale are study-specific and should be estimated for the stated endpoint, exposure regime, and time window, while hormetic and monotonic alternatives are compared [18]. These estimates may then inform scale-appropriate models that link mechanisms to organismal or population outcomes and account for energetic and eco-evolutionary costs. The framework exhibits a continuous, integrative loop. It begins with ecophysiology's contribution of mechanistic insight through controlled experiments; these mechanisms are then used to forecast population-level effects and analyze community dynamics using ecotoxicological scaling tools. The process measures trade-offs and identifies adaptive phenotypes, ultimately leading to real-world impact assessments grounded in ecological realism. The aim is to predict eco-evolutionary dynamics under global change and to develop conservation strategies and ecological risk assessments that account not only for toxicity but also for acclimatory, adaptive, overcompensatory, and buffering responses [9]. This unified discipline may also facilitate related areas, including environmental phenomics, in which phenotype expresses the nexus between physiology and toxicology, and eco-toxicophysiomics, in which multi-omics tools resolve mechanisms below the individual level and connect them to responses at higher levels of organization.

Ecophysiotoxicology, built on the hormesis framework, permits general models of stress response to link mechanistic biological processes with ecological outcomes across scales. Two equally important and interconnected pillars support the field. A mechanistic pillar from ecophysiology provides explanatory power, resolving the biological mechanisms and causal pathways of stress responses (how?). An ecological-scaling pillar from ecotoxicology contributes dose-response and ecological-scaling tools that place those mechanisms in ecological context and help forecast outcomes for populations, communities, and ecosystems (so what?). Their integration seeks general models rather than separate observations, with the aim of predicting how multiple stressors affect a system from biochemical reactions to ecosystem processes. Ecophysiotoxicology thus synthesizes physiology and molecular biology, ecology, and toxicology. Among its aims are to use physiology and biochemistry to quantify the costs of adaptive responses, follow adaptive traits in wild populations, and resolve eco-evolutionary dynamics in stressed and contaminated environments. The field moves beyond compartmentalized areas by integrating their strengths. Its central goal is to explain how ecological systems perceive and respond to both natural and anthropogenic stressors and to predict how those responses propagate from molecules to organisms, populations, communities, ecosystems, and landscapes. The mechanistic pillar identifies causal pathways through gene regulation, protein synthesis, metabolic trade-offs, and energy budgets; then, the ecological-scaling pillar uses that understanding to forecast future ecological outcomes.

3.1. From the Two Pillars to Testable Prediction

Application follows a concise sequence (Figure 3). The analysis begins by defining the biological system under study, stressors, biological hierarchy, time window, and endpoint to be predicted. Exposure is then mapped as dose, duration, timing, sequence, bioavailability, and, where relevant, internal dose, and stressor dissimilarity is specified before the response is examined. Experiments span the dose range required to identify both the low-dose and high-dose limbs, include time and recovery, and compare hormetic with monotonic alternatives. The mechanistic pillar measures signaling, damage and repair, detoxification, energy allocation, and performance. The ecological-scaling pillar links those measurements to survival, reproduction, population dynamics, community composition, or ecosystem function through adverse-outcome-pathway, toxicokinetic–toxicodynamic, demographic, or individual-based models [58–60]. Predictions are finally evaluated against independent data from another mesocosm, field site, population, or sampling season.

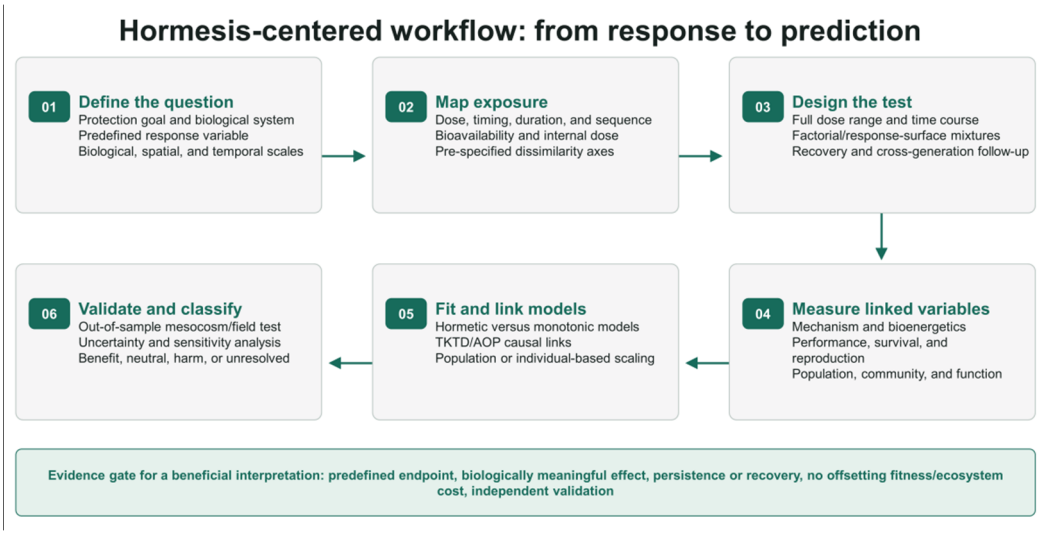

Figure 3. Operational workflow for ecophysiotoxicology. The workflow outlines how the hormesis-centered two-pillar concept could be used to develop and test cross-hierarchy predictions: define the question and endpoint; map exposure and stressor dissimilarity; span dose, time, and recovery; measure linked mechanisms and ecological responses; compare response and scaling models; and validate independently before interpretation or application. AOP: adverse outcome pathway; TKTD: Toxicokinetic-toxicodynamic model.

3.2. Context-Dependent Classification of Outcomes

Beneficial, neutral, and harmful are not intrinsic properties of a dose or response but conditional judgments relative to a defined biological and decision context [61]. This qualification is essential in hormesis because stimulation in one endpoint may coexist with inhibition or invariability in another, and apparent gains may carry delayed, cross-level, or transgenerational costs [12,18,51,62]. Before data collection, an analysis should therefore specify the reference condition, endpoint of interest, biological level, time horizon, management or protection objective, desired direction of change, minimum ecologically relevant effect or equivalence bounds, uncertainty criterion, and sentinel endpoints representing unacceptable trade-offs. A response is operationally beneficial only when it exceeds the prespecified meaningful-change threshold in the desired direction without an overriding adverse change in the protected or sentinel endpoints over the relevant period. Conversely, it is harmful when an adverse threshold is exceeded, a protected function declines, or an apparent gain is offset by unacceptable delayed or cross-level costs. It is neutral when the estimate and its uncertainty remain within prespecified equivalence bounds around the reference, instead of only when a conventional significance test is nonsignificant. Where endpoints or levels differ in sign, or uncertainty prevents assignment, the outcome should be reported as mixed or indeterminate rather than forced into one category. Classification should thus be stated explicitly as beneficial, neutral, or harmful for endpoint X, at biological level Y, over time T, relative to reference R and objective O. Prespecification makes the unavoidable context dependence transparent and prevents post hoc labeling.

3.3. Applied Examples

Consider warming combined with a pulsed pesticide exposure in fresh water. Ecophysiology supplies the mechanistic measurements: thermal performance, metabolic demand, pesticide uptake, detoxification, redox

regulation, energy reserves, and recovery. Ecotoxicology supplies the concentration-response design and the scaling from survival and reproduction to population or community outcomes. Hormesis generates a distinctive prediction: a low pesticide exposure may induce defenses that reduce injury from a later heat spike, whereas a higher dose, an altered sequence, or insufficient recovery may reverse the interaction. Such cross-protection has been reported within and across generations, but the unified framework further asks whether the molecular and organismal response predicts persistence at the population level [10].

A second example combines drought-rewetting cycles with pesticides, metals, or microplastics in soil. The mechanistic pillar resolves how water limitation and rewetting alter pollutant bioavailability, microbial metabolism, repair, and resource allocation. The scaling pillar tests consequences for microbial abundance, community structure, decomposition, nutrient transformation, and functional recovery. Quantified dissimilarity separates stressors that converge on common pathways from those that perturb distinct processes, allowing synergy, antagonism, and buffering to be predicted rather than merely described [27]. In both examples, neither discipline alone supplies the complete causal chain; hormesis provides the dose-dependent logic that connects their complementary strengths.

The unification creates transformed goals and applications. Risk assessment does not need to replace protective thresholds with presumed beneficial doses (stimulation does not directly translate to benefit, which depends on context). Instead, ecophysiotoxicology expands assessment from an exclusive search for adverse-effect thresholds to characterization of the full biphasic response, including the conditions under which adaptive responses alter sensitivity, recovery, or vulnerability to a subsequent stressor. This is a more complete objective, not a relaxation of protection. In conservation and remediation, the framework also creates a basis for testing low-dose priming as a means of preparing organisms or populations for disease, heatwaves, or chemical pollution, and for testing hormesis-informed enhancement of phytoremediation. Such interventions require predefined target endpoints, dose limits, stopping rules, and evidence that short-term stimulation is not offset by later costs. By resolving general patterns and their context dependence, the unified discipline should improve predictions of ecosystem responses to interacting global-change stressors and advance next-generation risk assessment, evolutionary conservation biology, precision remediation, restoration, and ecosystem-resilience science.

3.4. Scope Conditions and Research Priorities

The following scope conditions strengthen the central role of hormesis. Evidence for hormetic adaptive responses is extensive at cellular and organismal levels, whereas population-, community- and ecosystem-level evidence is newer and scarce [53,63–67]. At higher levels of biological organization, a biphasic aggregate response may reflect not only hormetic adaptive responses within constituent organisms but also emergent ecological processes, including replacement of susceptible taxa by tolerant taxa, changes in competition or mutualism, and release from biotic constraints [57,68–71]. These mechanisms are not mutually exclusive: interacting components of plant-soil-microbe systems may either exhibit coordinated hormetic responses or jointly generate and modulate hormetic outcomes through changes in microbial abundance, community structure, metabolic activity, nutrient acquisition, or contaminant degradation. Evidence includes concurrent physiological and rhizosphere-microbial hormesis under crude-oil exposure, biphasic responses involving mycorrhizal activity or symbiotic nitrogen fixation, and microbiome-mediated low-dose plant stimulation under antibiotic or pesticide exposure [65,72–76]. Ecophysiotoxicology treats this translation as a main research problem: the mechanistic pillar identifies the origin of the response, while the scaling pillar tests whether it persists, is buffered, or changes sign across the hierarchy.

A second condition is that an adaptive response is not identical to an unconditional benefit. Induced repair, detoxification, or metabolic reorganization can increase resistance to a challenge while drawing resources from growth, reproduction, immunity, or future performance. These trade-offs are part of hormesis biology, not evidence against its unifying role [62,77]. Accordingly, studies should measure the response through time, include recovery or subsequent challenge, and distinguish the endpoint displaying hormesis from the protected ecological endpoint. Priming and resilience-enhancing interventions remain important applications, but their dose window and longer-term consequences must be established before field implementation.

4. Conclusions

The integration of ecophysiology and ecotoxicology into ecophysiotoxicology represents a paradigm shift driven by hormesis as a general biological principle. Hormesis reveals a shared dose-dependent logic by which natural and anthropogenic stressors can induce adaptive, compensatory, or protective responses at low doses and inhibition or harm at higher doses. This common foundation makes it possible to combine the explanatory power

of ecophysiology (its mechanistic resolution of molecular, metabolic, and physiological adjustments) with the dose-response and ecological-scaling tools of ecotoxicology. The resulting two-pillar field does not just place two established disciplines side by side but provides a basis for developing testable cross-hierarchy predictions for multiple natural and anthropogenic stressors within the same framework. Testing them would require a defined endpoint, biological level, exposure scenario, time window, scaling model, and independent validation with new, field, or mesocosm data. By preserving mechanistic depth while extending prediction across biological scales, ecophysiotoxicology can advance holistic risk assessment, conservation, and resilience-based intervention and strengthen the capacity of global change biology to address the interconnected biodiversity and environmental crises of the Anthropocene.

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Conflicts of Interest

None of the funders was involved in the study design, preparation, approval, or decision where to submit this work. The author declares no conflict of interest.

Use of AI and AI-Assisted Technologies

During the revision of the manuscript, the author used ChatGPT (GPT-5.6 Sol) in order to suggest improvements and enhance the text. After using this tool, the author reviewed and edited the content as needed and takes responsibility for the content of the publication.

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