Alignment dynamics: how environmental restoration reshapes disease risk

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Abstract

Ecological change can generate variable infectious disease outcomes even when it produces similar shifts in biodiversity, habitat structure, or pathogen hazard. Existing disease-ecology frameworks identify the components and pathways governing transmission, but less explicitly formalise how their relative timing and spatial organisation determine when transmission opportunities emerge. We argue that realised disease risk arises from the spatiotemporal alignment of hazard, exposure, and regulatory processes. Given that these processes respond asynchronously to environmental and socio-ecological change, disease risk is best understood as a dynamic state generated by the alignment of these processes rather than as a direct consequence of hazard alone. We introduce two complementary concepts, coupling regimes and alignment trajectories, to describe how transmission systems differ in the organisation of hazard, exposure, and regulation, and how these relationships change through time.

We characterise coupling regimes through response-time hierarchy, spatial coupling structure, exposure elasticity and regulation latency, and derive hypotheses concerning dynamic alignment, differential ecological and social clocks, and epidemiological decoupling.

Ecological restoration provides a powerful context for observing these dynamics because it simultaneously reorganises multiple ecological and social processes operating on different timescales. By formalising alignment dynamics, this perspective provides a comparative framework for understanding, predicting, and managing disease risk under ecological change.

Keywords: Infectious disease risk, ecological restoration, coupling regimes, alignment dynamics, social–ecological systems, landscape planning

1. Introduction

Anthropogenic landscape transformation restructures host–vector–pathogen interactions and can either amplify or reduce infectious disease risk. A large body of work has demonstrated that landscape influences disease emergence through interconnected effects on biodiversity, ecosystem structure, host competence, exposure pathways and contact among humans, domestic animals, wildlife, and vectors (Patz *et al.* 2004; Olival *et al.* 2017; Wells *et al.* 2018; Gibb *et al.* 2020; Manlove *et al.* 2022; Rulli *et al.* 2025). These advances have established that disease risk is not determined by any single ecological factor, but instead emerges from interactions among pathogen hazard, host exposure, susceptibility, environmental conditions, and management interventions (Keesing *et al.* 2010; Plowright *et al.* 2024).

Ecological restoration and rewilding have recently highlighted the complexity of these relationships. Biodiversity recovery, trophic reassembly, and habitat restoration can increase disease risk in some systems, reduce it in others, or generate transient responses that change through time (Carvalho *et al.* 2025; Ecke *et al.* 2025; Keesing 2025; Fell *et al.* 2026; Rafael *et al.* 2026). Similar restoration interventions can therefore produce contrasting epidemiological outcomes despite generating broadly comparable ecological changes. These observations increasingly suggest that disease emergence cannot be understood solely by asking whether restoration increases or decreases hazard, but by understanding how ecological and social processes reorganise following environmental change.

More broadly, this challenge reflects a central principle of complex systems. Across ecology, nonlinear dynamics and coupled human–natural systems, emergent behaviour is understood to arise from interactions among processes operating across multiple spatial and temporal scales rather than from the properties of individual components alone. Ecological patterns emerge through cross-scale interactions (Levin 1992); coupled dynamical systems undergo qualitative changes in behaviour as interactions among their components change (Strogatz 2001, Winfree, 2001 #66155); ecosystems reorganise through adaptive cycles operating across interacting temporal and spatial scales (Gunderson & Holling 2002); and coupled human–natural systems exhibit emergent dynamics generated through reciprocal ecological and societal feedbacks (Liu *et al.* 2007).

Disease ecology has developed complementary conceptual and mechanistic frameworks describing the ecological drivers of transmission, the pathways linking reservoirs, vectors and susceptible hosts, and the mechanisms through which environmental change modifies hazard, exposure, susceptibility and ecological regulation (Patz *et al.* 2004; Keesing *et al.* 2010; Manlove *et al.* 2022; Plowright *et al.* 2024). More recently, dynamic modelling approaches have begun to represent how disease risk evolves under environmental change. For example, (Vinson *et al.* 2022) showed

that land reversion can generate contrasting transient and long-term spillover trajectories depending on habitat-transition rates and the epidemiological properties of regenerating landscapes. These advances increasingly recognise that disease emergence is dynamic and context dependent. However, they generally describe the mechanisms or trajectories of particular transmission systems rather than providing a general conceptual framework for understanding how ecological and social processes become organised into the system-specific dynamics that generate realised disease risk over time under environmental change.

Here we develop such a conceptual framework by integrating established principles of complex systems into disease ecology. We conceptualise hazard generation, exposure pathways and regulatory processes as three interacting dynamical process classes that evolve on different ecological and social clocks. Environmental change rarely alters these processes synchronously; instead, it continuously reorganises their relative timing, spatial organisation and functional coupling. We propose that realised disease risk is therefore best understood as an emergent systems state arising from the temporary spatiotemporal alignment of hazard, exposure and regulation. To formalise this perspective, we introduce coupling regimes as transferable descriptors of transmission-system organisation and alignment trajectories as the emergent disease-risk dynamics generated by those regimes under environmental change. As transferable descriptors of transmission-system organisation, coupling regimes do not replace mechanistic explanations or mathematical models. Rather, they characterise the system-level organisation of hazard, exposure and regulatory processes in ways that can be compared across diverse disease systems. In doing so, they provide a common conceptual representation for integrating empirical observations, mechanistic understanding and quantitative models. Importantly, the objective is not to reduce disease emergence to four variables or to replace the mechanistic complexity of ecological systems. Rather, coupling regimes identify the organisational properties through which diverse ecological and social mechanisms become comparable across transmission systems. These properties may be represented through empirical observations, mechanistic model structure, combinations of model parameters, or inferred system trajectories. Although these representations differ analytically, we propose that they describe the common organisational properties governing how hazard, exposure and regulation interact across space and time, thereby providing a common conceptual foundation for comparing otherwise disparate transmission systems. Alignment trajectories therefore describe not only whether disease risk changes, but when transmission opportunities emerge, how long they persist, and how they disappear as ecological and social processes become reorganised.

Ecological restoration provides a particularly powerful context in which to observe these dynamics because it simultaneously reshapes hydrology, vegetation structure, host communities, landscape connectivity, management practices, and patterns of human activity. Importantly, these processes

rarely reorganise at the same rate. Hydrological conditions may change rapidly following peatland rewetting or floodplain reconnection, creating habitat for mosquito vectors within weeks or months, whereas tick-associated hazards may emerge more gradually as vegetation structure, host communities, and microclimatic conditions develop over years. Exposure pathways may be only weakly linked to ecological succession and instead reflect human decisions regarding public access, livestock management, recreation, infrastructure, or conservation policy (Ostrom 2009; Gottdenker *et al.* 2014; Zeimes *et al.* 2014). Regulatory processes, including trophic recovery, predator establishment, immunity, and adaptive management, frequently follow different trajectories again. Restoration therefore functions as a natural systems experiment that reveals how hazard, exposure, and regulation become coupled, decoupled and realigned through time.

Given that ecological and social clocks differ among transmission systems, restoration generates distinct alignment trajectories and disease outcomes even under similar environmental interventions. By formalising disease emergence as a problem of alignment dynamics, this Perspective proposes an organising conceptual framework for understanding how environmental change generates transient disease risk through the evolving spatial and temporal relationships among hazard, exposure and regulation. Coupling regimes describe the structural organisation of these interactions through response-time hierarchy, spatial coupling structure, exposure elasticity and regulation latency, while alignment trajectories describe the resulting disease dynamics. Together, these concepts complement rather than replace transmission-pathway frameworks, biodiversity-mediated mechanisms and mechanistic disease models by providing a transferable conceptual representation that can be expressed through empirical observations, mechanistic models or statistical inference, thereby supporting comparison, forecasting and intervention across heterogeneous disease systems.

2. A framework for alignment dynamics in disease risk

We propose a general framework in which realised infectious disease risk emerges from the spatiotemporal alignment of three classes of processes: hazard generation, exposure pathways, and regulatory processes. Hazard describes the distribution and intensity of infectious potential through pathogens, vectors, reservoirs, and competent hosts. Exposure describes how susceptible hosts encounter that hazard through movement, behaviour, resource use, land use, and ecological interfaces. Regulation comprises ecological and anthropogenic processes that constrain transmission, including predation, competition, immunity, veterinary interventions, vector control, and environmental management. These

processes can reduce the generation of infectious hazard, limit encounters between hazard and susceptible hosts, or reduce the probability that exposure results in transmission. In the framework, regulation is treated as a distinct process class because these transmission-constraining mechanisms can vary independently of the realised hazard and exposure state.

These components are individually well established within disease ecology. Our contribution is to identify their alignment as the co-occurrence in space and time of high hazard and exposure under conditions of insufficient regulatory constraint, which is a proximate mechanism through which transmission potential becomes realised disease risk (**Figure 1**). Hazard alone does not generate transmission, and exposure pathways do not necessarily produce infection when regulatory constraints are effective. Transmission opportunities emerge when hazard becomes embedded within active exposure pathways during periods in which regulation is absent, delayed, weakened, spatially disconnected, or otherwise insufficient to prevent transmission. Disease risk should therefore be viewed not as a property of hazard itself, but as a transient state arising from the alignment of hazard, exposure, and regulation.

A central feature of this framework is that hazard, exposure, and regulation are governed by different ecological and social clocks. Infectious hazard may respond rapidly or slowly to environmental change depending on pathogen, vector, reservoir, and host biology. Exposure pathways may be shaped by ecological processes, behaviour, infrastructure, management decisions, or social practices that operate on different timescales. Regulatory processes may emerge through trophic recovery, immune responses, recolonisation, management adaptation, or policy interventions, each characterised by distinct delays and spatial extents. Environmental change therefore rarely produces synchronous responses across all components; instead, it continuously alters the relative timing and spatial overlap among hazard, exposure, and regulation.

We define **coupling regimes** as the structural organisation of interactions among hazard generation, exposure pathways, and regulatory processes that determines how and when alignment becomes possible. Coupling regimes are not defined by the magnitude of hazard or exposure, but by four analytically distinguishable dimensions that independently constrain the emergence of transmission-favourable alignment.

(i) **Response-time hierarchy (temporal ordering of change)**. Response-time hierarchy describes the relative speed at which hazard, exposure, and regulation respond to environmental or management perturbation. It captures ordering in time, not delay magnitude or process strength. A system may exhibit fast hazard dynamics and slow regulation, or the reverse, independent of spatial structure or behavioural responsiveness. This dimension determines whether alignment tends to emerge early, late, or intermittently following environmental change.

(ii) **Spatial coupling structure (spatial interaction topology)**. Spatial coupling structure describes how hazard, exposure, and regulation are connected across space, independent of their temporal dynamics. It ranges from strict local co-location (high spatial coupling) to movement-mediated or networked interactions (intermediate coupling) to spatial decoupling where processes operate in distinct locations. This dimension determines whether alignment is spatially constrained, spatially diffuse, or dependent on connectivity pathways.

(iii) **Exposure elasticity (responsiveness of contact structure)**. Exposure elasticity describes the magnitude and speed with which exposure pathways reorganise in response to ecological, behavioural, institutional or management change. It includes changes in human movement, livestock distribution, access behaviour and contact-network structure. High elasticity indicates that relatively small changes in environmental or social conditions can rapidly reorganise exposure, whereas low elasticity indicates persistent contact structures that respond weakly or slowly. Exposure elasticity is distinct from predictability: social exposure pathways may be highly elastic yet either consistently responsive to known drivers or discontinuous and difficult to forecast.

(iv) **Regulation latency (activation delay of suppressive processes)**. Regulation latency describes the time delay between the emergence of hazard–exposure conditions and the effective activation of regulatory processes that reduce or interrupt transmission. Crucially, this dimension is distinct from overall regulation strength or speed of ecological recovery. A system may exhibit strong eventual regulation but still experience prolonged alignment windows due to delayed activation.

These four dimensions are defined to vary independently, although they may be empirically correlated in particular systems. Together, they define a four-dimensional coupling-regime space in which transmission systems are positioned. The interaction of these independent axes determines the likelihood, timing, and duration of spatiotemporal alignment and realised disease risk. Importantly, alignment is not an intrinsic property of any single dimension, but an emergent outcome of their joint configuration. Systems that differ in only one dimension may nevertheless generate fundamentally different alignment trajectories under identical environmental forcing. We focus on these four dimensions because each describes a distinct constraint on alignment that is not reducible to component magnitude: the ordering of process responses, the topology of their spatial interaction, the responsiveness of contact structure to forcing, and the delay before suppressive feedback becomes effective. Other system properties, such as stochasticity, seasonality, feedback strength and thresholds, may modify alignment trajectories and represent important extensions rather than defining dimensions of the proposed framework.

Environmental change and ecological succession dynamics generate alignment trajectories within these coupling regimes. Alignment trajectories describe how relationships among hazard, exposure, and regulation evolve through time as ecosystems, host communities, and human activities reorganise. Similar environmental changes can generate contrasting alignment trajectories because transmission systems differ in their underlying coupling regimes. Fast-responding vector populations combined with delayed predator recovery may generate transient pulses of alignment, whereas systems characterised by strong regulation or weak spatial overlap between hazard and exposure may exhibit delayed, diffuse, or absent transmission responses. Disease outcomes therefore reflect not only the magnitude of ecological change, but also the trajectory through which alignment emerges, persists, and collapses (**Figure 1**).

Different transmission systems should therefore comprise different coupling-regime configurations. Mosquito-borne, tick-borne, avian-vector, and grazing-associated transmission systems each exhibit characteristic coupling regimes and therefore generate distinct alignment trajectories under restoration despite experiencing some similarities in environmental interventions (**Figure 2**).

This framework generates explicit, testable predictions about how environmental change reorganises transmission systems. Existing disease-ecology frameworks identify the components, pathways and constraints governing transmission. The alignment perspective builds on these foundations by predicting how the relative timing, spatial organisation and interaction of those components determine when transmission opportunities emerge, persist and disappear under environmental change. Consequently, the framework shifts attention from the magnitude of individual risk factors towards the dynamic organisation of hazard, exposure and regulation through time and space. The specific hypotheses arising from this perspective are developed in Section 6.

3. Restoration as differential ecological and social forcing

Environmental change does not act uniformly on the processes that generate disease risk. Restoration, rewilding, and other forms of landscape reorganisation simultaneously modify hydrology, vegetation structure, host communities, landscape connectivity, management practices, and patterns of human activity (Haddad *et al.* 2015; Suding *et al.* 2015; Bullock *et al.* 2022). Their epidemiological effects therefore depend not only

on whether restoration increases or decreases hazard, exposure, or regulation, but on how it reorganises the relative timing and spatial coupling among these processes.

Across Europe and the United Kingdom, restoration increasingly occurs through large-scale interventions such as peatland recovery, wetland creation for biodiversity and flood-risk management, coastal realignment, river restoration, woodland expansion, urban green and blue space expansion, and transitions towards low-intervention grazing systems (Perino *et al.* 2019; Tickner *et al.* 2020; Zoderer *et al.* 2025). These interventions rarely produce a single ecological state change. Instead, they initiate trajectories of ecosystem reorganisation in which different ecosystem components and human-use processes respond at different rates.

Peatland restoration illustrates this response-time hierarchy. Hydrological interventions such as drainage blocking can raise water tables and create shallow aquatic habitats within months to years, facilitating early colonisation by aquatic and semi-aquatic invertebrates (Beadle *et al.* 2023). However, peat-forming vegetation, trophic complexity, and long-term hydrological functioning may require decades or longer to recover (Brown *et al.* 2016; Alderson *et al.* 2019; Rydgren *et al.* 2025). Forest restoration follows similarly extended trajectories, with tree growth, structural complexity, dispersal limitation, deadwood development, and trophic reassembly unfolding over decades to centuries (Fuentes-Montemayor *et al.* 2021; Waddell *et al.* 2024). These transitions are driven by successional and recolonisation processes that operate at different rates across taxa and functional groups, generating temporal lags in ecological regulation and community assembly (Watts *et al.* 2020). Consequently, habitat recovery is often strongly taxon- and system-specific, reflecting differences in dispersal, life-history traits, environmental filtering, and species interactions (Escobar *et al.* 2026). Early successional forest stages are frequently dominated by generalist species, whereas later stages increasingly support specialist taxa and greater structural and functional heterogeneity (Paillet *et al.* 2010). Because host community structure is a key determinant of pathogen transmission dynamics in multi-host systems, these successional changes can also reshape dilution and amplification processes in vector-borne disease systems such as Lyme borreliosis and West Nile virus, where shifts in host diversity and competence influence infection prevalence in vector populations (Kilpatrick *et al.* 2006; Ostfeld *et al.* 2006).

Restoration also alters spatial coupling structure. Hydrological restoration can redistribute surface water and create aquatic vector habitat, but increased vector abundance becomes realised risk only where it overlaps with susceptible hosts before regulatory processes, such as increased predator abundance or reduced surface areas for ovipositing mosquitoes in more permanent aquatic habitats, become effective (Dworrak *et al.*

2022). Vegetation recovery modifies microclimates, host movement, and contact rates, potentially favouring some vectors (for example ticks, that benefit from increased surface humidity on more dense vegetations) while reducing suitability for others (Medlock *et al.* 2013; Semenza & Suk 2018; De Frenne *et al.* 2021). Landscape connectivity can connect or dilute transmission pathways by redistributing hosts, vectors, and pathogens across space (Prist *et al.* 2023). Restoration therefore changes not only the magnitude of hazard, but where hazard is positioned relative to exposure pathways and regulatory processes.

Regulation may lag, strengthen, weaken, or shift scale during recovery. Predator establishment, trophic reassembly, host immunity, competitive interactions, veterinary management, deer control, or vector management may not track hazard generation. Host-associated and environmental microbiomes may respond rapidly to changes in diet, habitat, stress, and environmental conditions, potentially altering susceptibility or pathogen persistence on faster timescales than vertebrate community recovery (David *et al.* 2014; Rothschild *et al.* 2018; Caballero-Flores *et al.* 2023; Spragge *et al.* 2023; Burton *et al.* 2024). Regulatory capacity is therefore not simply absent or present; it changes with its own temporal and spatial dynamics.

Exposure pathways often follow different social clocks altogether. Human access, recreation, livestock movements, conservation policy, infrastructure, and land-management decisions may change rapidly and independently of ecological succession (Ostrom 2009; Gottdenker *et al.* 2014; Zeimes *et al.* 2014). Hydrological restoration may create mosquito habitat while public access is expanded through new footpaths or visitor infrastructure, or conversely while access is restricted by management. Grazing systems can shift rapidly through stocking density, veterinary treatment, or grazing agreements, even when ecological conditions change slowly. More generally, restoration outcomes often depend on whether management interventions operate on timescales commensurate with ecological recovery processes, highlighting the importance of temporal alignment between ecological and governance dynamics (Wells *et al.* 2026 in press).

Restoration therefore provides a powerful setting for studying alignment dynamics. It perturbs ecological and social processes simultaneously, but rarely synchronously. Disease outcomes depend on how restoration changes response-time hierarchy, spatial coupling structure, regulation latency, and exposure elasticity across transmission systems.

4. Transmission-specific coupling regimes

The epidemiological effects of restoration depend on the coupling regime governing each transmission system. Different systems vary in how hazard is generated, how exposure occurs, how spatially connected these processes are, and how rapidly regulation becomes effective. These differences produce distinct alignment trajectories across landscapes.

Mosquito-borne systems are often strongly structured by hydrological conditions. Short vector generation times allow mosquito populations to respond rapidly to environmental change (Dworrak *et al.* 2022; Meuti 2026). Exposure depends on local habitat use by humans, livestock, or wildlife, whereas regulatory processes such as aquatic predation and food-web recovery may lag behind (Chase & Knight 2003). This configuration produces fast, locally coupled regimes in which vector-mediated transmission pathways may be rapidly activated following restoration, but can remain transient if regulation strengthens or exposure is managed.

Tick-borne systems, including Lyme borreliosis, are structured by vegetation, microclimate, host density, and host competence, with slower dynamics in both hazard generation and regulation (Ostfeld *et al.* 2006; Ogden & Lindsay 2016). Exposure often arises through passive contact in shared habitats, resulting in transmission pathways that emerge gradually and persist over longer periods. These systems are therefore expected to generate delayed but potentially persistent alignment trajectories as habitat structure and host communities develop and shift exposure and regulation.

Bird–mosquito systems, including West Nile virus and avian haemosporidians, introduce a different coupling architecture because mobile reservoir hosts redistribute pathogens across broad spatial scales (Hellgren *et al.* 2007; Kirby *et al.* 2025). Hazard may therefore become partially decoupled from local environmental conditions if migratory or dispersive hosts dominate pathogen movement. Local restoration may alter vector habitat or host aggregation, but transmission dynamics may depend on connectivity beyond the restoration site.

Grazing-associated and helminth systems are more tightly linked to management structure. Transitions from intensively managed livestock systems towards semi-feral or low-intervention grazing can alter hazard, exposure, and regulation simultaneously by changing stocking density, pasture use, veterinary treatment, and environmental contamination (Kumar *et al.* 2013; Jenkins *et al.* 2020). Here, exposure elasticity and

regulation latency may be especially important because human decisions can rapidly strengthen or weaken coupling between hazard and susceptible hosts.

These examples are not isolated case studies, but archetypes of coupling-regime space. Mosquito-borne, tick-borne, avian-vector, and grazing-associated systems occupy different positions along response-time hierarchy, spatial coupling structure, regulation latency, and exposure elasticity. Restoration therefore does not impose a single epidemiological response; it generates system-specific alignment trajectories determined by the structural organisation of hazard, exposure, and regulation.

5. Designing restoration for epidemiological decoupling

If realised disease risk emerges from alignment, then restoration can be designed not only to reduce hazard, but to manipulate coupling. This reframes disease prevention from asking whether restoration increases or decreases disease risk to asking how restoration changes the timing and spatial organisation of transmission opportunity.

Designing for decoupling can operate through several mechanisms. First, restoration can reduce spatial coupling by separating hazard-generating habitats from exposure pathways, for example by locating wetland creation away from high-use visitor infrastructure, livestock congregation areas, or residential interfaces. Second, it can alter response-time hierarchy by delaying exposure until regulation has strengthened, or by accelerating regulatory recovery through habitat design that supports predators, competitors, or management access. Third, it can reduce regulation latency through adaptive surveillance, targeted vector control, deer management, grazing plans, or veterinary interventions during periods when hazard and exposure are likely to align. Fourth, it can modify exposure elasticity by managing how human or livestock behaviour responds to ecological change through footpath routing, public access timing, grazing agreements, signage, or seasonal restrictions.

This framing also clarifies a challenge for restoration governance and risk communication. Observable indicators of hazard, including mosquitoes, ticks, or recovering wildlife populations, are often interpreted as direct evidence of elevated disease risk. Yet realised risk depends on whether hazard becomes aligned with exposure under insufficient regulation. Restoration may therefore increase visible hazard while reducing realised risk if exposure pathways are decoupled or regulation strengthens. Conversely, modest hazards may generate substantial risk when they coincide with highly connected exposure pathways and delayed regulation.

Operationalising alignment-based disease ecology requires a shift from static measurement of individual components towards inference about their joint dynamics through time and space. In principle, field studies should integrate data on hazard, exposure, and regulation, including measures such as vector or pathogen abundance, host competence, human and livestock movement, predator presence, and management intensity. However, these variables are rarely observed at compatible temporal and spatial resolutions, and key regulatory and exposure processes often remain partially unobserved, particularly at wildlife–human interfaces where multiple ecological and social processes interact across scales (Roberts *et al.* 2021). For this reason, the coupling-regime framework should be viewed not as an immediately fully parameterisable model, but as a structured way of organising data collection, hypothesis generation, and comparative inference under ecological change (**Box 1**). Even when complete parameterisation is not feasible, identifying response-time hierarchies, spatial coupling patterns, regulatory delays, and exposure elasticity provides a consistent basis for comparing systems and restoration contexts.

Scenario-based and mechanistic modelling approaches offer one pathway towards partial operationalisation. These may combine species traits (e.g. generation time, dispersal capacity, host competence, habitat affinity, colonisation potential) with landscape descriptors of hydrology, vegetation, connectivity, land use, and management. Such approaches can be used to explore how alternative assumptions about coupling structure influence the emergence and timing of alignment, even when empirical data are incomplete. Emerging integrative modelling approaches, including digital-twin implementations where data streams are sufficient, may eventually allow continuous updating of system states under monitoring data. However, these remain aspirational for most ecological systems and should be interpreted as future extensions rather than prerequisites for applying the framework.

Crucially, the value of the framework does not depend on complete observability of all components, but on its ability to redirect attention from isolated drivers of risk towards the conditions under which hazard, exposure, and regulation become temporarily aligned. Even partial data can therefore be informative when interpreted through the lens of coupling regimes and alignment trajectories.

6. Research agenda: testing alignment dynamics across disease systems

The alignment framework shifts the central question in disease ecology from whether environmental change increases or decreases disease risk to how environmental change reorganises the ecological and social processes that generate transmission opportunities. Rather than viewing hazard,

exposure and regulation as independent determinants of disease emergence, the framework proposes that realised disease risk reflects their dynamic spatiotemporal organisation under environmental change. This perspective generates a comparative research programme centred on understanding how coupling regimes shape disease trajectories, why those trajectories differ among transmission systems, and how they can be modified through management. Three complementary hypotheses provide the foundation for this programme.

H1. Dynamic-alignment hypothesis

Environmental change generates disease-risk trajectories by reorganising the relative timing, spatial configuration and functional coupling of hazard generation, exposure pathways and regulatory processes. Disease outcomes should therefore depend not only on whether transmission-pathway components are individually permissive, but on how their relationships emerge, persist, recur and dissolve through time and space.

This hypothesis predicts that systems with comparable hazard, biodiversity recovery or restoration state may nevertheless exhibit contrasting epidemiological trajectories because hazard, exposure and regulation may become organised differently. Dynamic representations of lagged interactions, spatial overlap and regulatory constraint should therefore improve explanation and, where appropriate data exist, out-of-sample prediction of the timing, duration and recurrence of disease outcomes relative to hazard-centred, static-state or additive component models. Repeated environmental disturbances or management interventions are further predicted to generate recurrent alignment windows when they repeatedly reorganise coupling among hazard, exposure and regulation.

H2. Differential-clocks hypothesis

Contrasting disease trajectories emerge because hazard generation, exposure pathways and regulatory processes respond to environmental change on different ecological and social clocks. Taxon-specific colonisation, vegetation succession, food-web assembly, host-community reorganisation and immune development generate characteristic ecological response times. By contrast, exposure and management may respond to land-use decisions, infrastructure, policy, economic incentives and human behaviour on social timescales that are less tightly constrained by ecological succession and may change discontinuously.

Exposure elasticity and predictability are therefore distinct. Exposure may respond strongly to ecological or societal drivers while remaining difficult to forecast because those drivers themselves are variable or institutionally contingent. Consequently, disease trajectories should be highly

sensitive to temporal mismatches among ecological recovery, human activity and management interventions. Transmission opportunities emerge when hazard, exposure and regulation become synchronised in space and time, and may therefore increase, decline or recur depending on whether restoration, public access, grazing, veterinary intervention, vector control or conservation management reinforce or disrupt alignment. Differences in the ordering, magnitude and predictability of ecological and social responses should therefore determine whether alignment develops rapidly, gradually, recurrently, persistently or not at all.

Across restoration contexts, this hypothesis predicts relatively rapid hazard responses in mosquito-associated systems where vector populations closely track hydrological change, but slower and potentially more persistent trajectories in tick-associated systems governed by vegetation succession and host-community assembly. Systems involving migratory or widely dispersing hosts may generate recurrent or spatially displaced alignment, whereas grazing-associated systems may shift abruptly following changes in stocking density, livestock movement or veterinary intervention. These expectations are intended as heuristic predictions rather than deterministic classifications; the same transmission system may occupy different coupling regimes across landscapes, seasons and management contexts.

H3. Epidemiological-Decoupling Hypothesis

Disease risk can be reduced by disrupting the temporal or spatial alignment of hazard generation and exposure pathways, or by accelerating effective regulation, without necessarily reducing ecological hazard itself. Consequently, restoration design and adaptive management should increasingly focus on modifying coupling regimes rather than exclusively suppressing hazard.

Testing this hypothesis requires natural experiments and intervention studies that compare strategies targeting alignment dynamics with conventional hazard-reduction approaches. Potential interventions include altering the timing of restoration activities, modifying access or grazing regimes, redesigning spatial connectivity, accelerating ecological regulation or adaptive surveillance, and coordinating management across connected landscapes. The effectiveness of such interventions will depend not only on which processes most strongly influence alignment, but also on which components of a coupling regime can realistically be modified. Ecological and social processes differ markedly in the way they can be manipulated or engineered: human access, livestock movement, restoration scheduling and infrastructure may often be adjusted rapidly, whereas

species colonisation, food-web assembly and many ecological regulatory processes frequently respond over much longer timescales or remain only partially controllable. Effective epidemiological decoupling should therefore prioritise interventions that target the components and clocks most amenable to management within the relevant alignment trajectory. Importantly, epidemiological decoupling should be evaluated alongside biodiversity, ecosystem-service and social objectives because interventions that reduce disease risk may involve ecological or societal trade-offs.

Towards a comparative disease ecology

Together, these hypotheses establish a comparative research programme centred on understanding disease systems as evolving organisations rather than static collections of risk factors. Testing the framework requires longitudinal studies that quantify hazard, exposure and regulation simultaneously, comparative analyses evaluating whether similar coupling regimes produce comparable disease trajectories across different pathogens and landscapes, and quantitative models that explicitly represent response-time hierarchy, spatial coupling structure, exposure elasticity and regulation latency. Comparative support for the framework would arise if coupling-regime dimensions consistently improve explanation, forecasting or intervention design relative to approaches based solely on hazard, static environmental conditions or additive transmission components.

Equally important are the framework's boundary conditions. Alignment dynamics is expected to provide greatest explanatory value where hazard, exposure and regulation vary independently, respond asynchronously to environmental change or are modified by ecological and social feedbacks. Conversely, it should contribute relatively little where a single process overwhelmingly limits transmission, exposure remains effectively constant, or epidemiological outcomes closely track directly measurable hazard. Different ecological mechanisms may also generate similar alignment trajectories, and similar coupling regimes need not imply identical biological processes. Establishing where alignment dynamics succeeds, where it fails and why should therefore become a central objective of comparative disease ecology under environmental change.

7. Conclusion

Environmental change does not simply alter ecological hazard, but it reorganises the temporal and spatial relationships among hazard generation, exposure pathways and regulatory processes. We argue that these changing relationships constitute an overlooked systems property governing when, where and for how long infectious disease risk is realised.

We propose coupling regimes as transferable descriptors of transmission-system organisation and alignment trajectories as their dynamic expression under environmental change. Together, these concepts provide a conceptual representation that complements rather than replaces transmission-pathway frameworks, biodiversity-mediated mechanisms and mechanistic disease models by linking empirical observations, mechanistic understanding and quantitative modelling within a common systems perspective.

Viewing disease emergence through alignment dynamics reframes ecological restoration as a natural systems experiment in which ecological and social processes continuously reorganise transmission opportunities. More broadly, the framework generates a research programme centred on testing how coupling regimes shape disease trajectories, forecasting and intervention across diverse transmission systems. Although developed using restoration as a model context, we anticipate that alignment dynamics provides a general conceptual framework for understanding disease emergence under environmental change and a common representation for comparing, predicting and managing infectious disease risk across heterogeneous ecological systems.

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Conflicts of interests

The authors declare no competing interests

Use of Artificial Intelligence

For the parts of the manuscript, we used ChatGPT (OpenAI) to improve English grammar, refinement of text, and harmonise terminology; all supportive AI use was checked for accuracy, biases, and proper citation; use did not affect content, results or conclusions.

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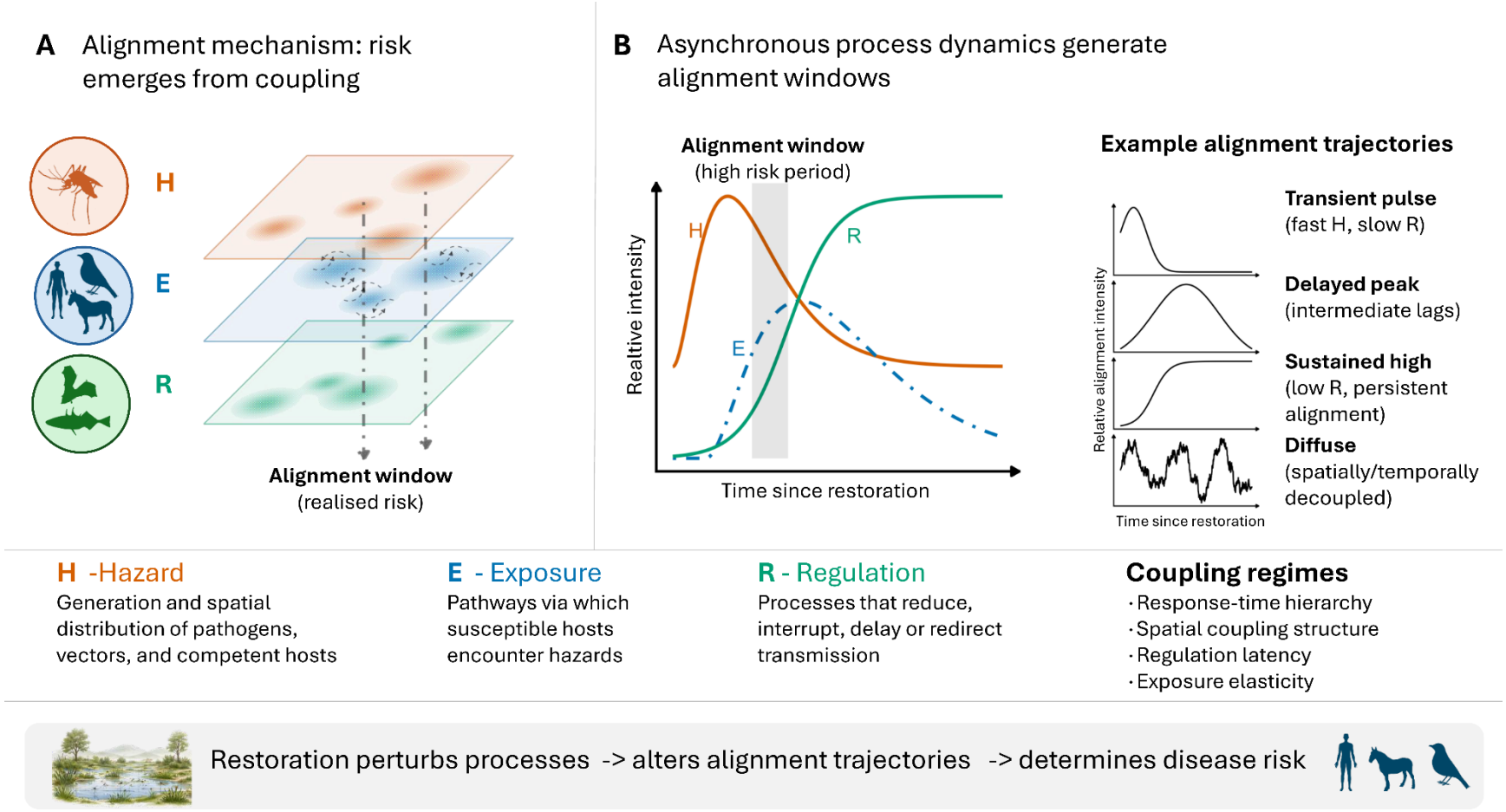


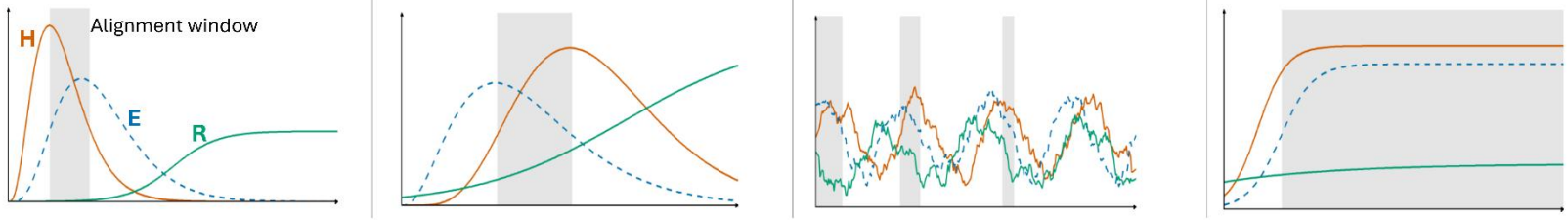
Figure 1 | Alignment dynamics govern realised disease risk under ecological change.

A: Conceptual representation of the alignment mechanism underlying infectious disease risk. Hazard (H), exposure (E), and regulation (R) are represented as spatially heterogeneous processes operating across landscapes. Realised disease risk emerges where infectious hazard overlaps with

active exposure pathways during periods or in locations where regulation is insufficient to prevent transmission. The alignment window (dashed projection) denotes this transmission-favourable configuration, not hazard alone. **B:** Environmental change alters the relative timing and spatial overlap of hazard, exposure, and regulation. Because these processes operate on different ecological and social clocks, they respond asynchronously to restoration and other forms of environmental change. The shaded region represents an alignment window, during which high hazard and active exposure coincide under insufficient regulation, generating elevated realised disease risk. Different coupling regimes produce distinct patterns in the timing, duration, and recurrence of alignment windows, termed **alignment trajectories**. Example alignment trajectories (right) illustrate conceptual patterns of relative alignment intensity through time, including transient pulses, delayed peaks, sustained alignment, and diffuse or intermittent alignment.

A System-specific coupling regimes

B Alignment trajectories



C Coupling regime attributes

H -Hazard E - Exposure - R – Regulation				
	Mosquito-borne systems	**Tick-borne systems**	**Bird-vector systems**	**Extensive grazer systems**
Response time hierarchy	H fast * E moderate * R slow	H moderate* E slow * R slow	H variable * E local * R weak	H high * E high * R absent
Spatial coupling structure	Local (hydrologic sites)	Co-located (host-habitat overlap)	Migration-mediated	Local (pasture/ host group)
Exposure elasticity	Moderate (human behaviour)	Low (passive contact)	Low- moderate (local)	High (intrinsic host exposure)
Regulation latency	High (predator colonisation lag)	High (community assembly lag)	Variable/ weak	Very low / absent control

Figure 2 | System-specific coupling regimes generate distinct alignment trajectories under restoration.

A. Conceptual schematics of four representative transmission-system archetypes illustrating how hazard (H), exposure (E), and regulation (R) are organised within different coupling regimes. Mosquito-borne systems are characterised by rapid hazard responses to hydrological change and delayed ecological regulation. Tick-borne systems exhibit slower hazard generation linked to host–habitat interactions and gradual regulatory

recovery. Bird–vector systems are structured by highly mobile reservoir hosts that redistribute pathogens across space, partially decoupling hazard from local environmental conditions. Extensive grazing systems are strongly influenced by management decisions that alter exposure pathways and regulatory constraints. **B.** Alignment trajectories generated by these coupling regimes following restoration. Coloured lines represent conceptual trajectories of hazard (orange), exposure (blue), and regulation (green). Grey shading denotes alignment windows, during which hazard and exposure coincide under insufficient regulation, creating opportunities for transmission. Differences in response-time hierarchy, spatial coupling structure, regulation latency, and exposure elasticity generate distinct temporal patterns of alignment, including transient pulses, delayed peaks, intermittent coupling, and sustained alignment. **C.** Positioning of transmission-system archetypes within coupling-regime space. Four dimensions characterise coupling regimes: response-time hierarchy, spatial coupling structure, exposure elasticity, and regulation latency. Together, these dimensions determine the likelihood, timing, duration, and spatial extent of alignment among hazard, exposure, and regulation. Differences in coupling-regime structure explain why similar restoration interventions can generate contrasting disease-risk trajectories across transmission systems.

Table 1 | Operationalising coupling regimes in alignment-based disease ecology. For each dimension defining coupling regimes (response-time hierarchy, spatial coupling structure, exposure elasticity, and regulation latency), the table provides its ecological interpretation, example measurable variables, and potential intervention levers through which restoration design or management may alter alignment dynamics and realised disease risk.

Dimension	Ecological meaning	Example measurable variables	Intervention leverage
Response-time hierarchy	Relative rates at which hazard, exposure, and regulation respond to environmental change or management interventions	Vector/pathogen generation time; host turnover rates; predator recolonisation speed; vegetation succession rates; timing of ecological change and/or management actions	Accelerate or delay system components to avoid overlap (e.g., timed vector control; staged restoration; synchronising management with regulatory recovery)
Spatial coupling structure	Degree of spatial co-location or connectivity among hazard, exposure, and regulation	Spatial co-occurrence of vectors/hosts and human use; landscape connectivity indices; movement corridors (hosts, vectors, people, livestock)	Reconfigure spatial layout to decouple risk (e.g., buffer zones; spatial zoning of restoration; relocation of access or grazing areas; fragmentation or connectivity management)
Exposure elasticity	Sensitivity of exposure pathways to ecological or social change	Visitor response to habitat change; livestock redistribution; seasonal recreation intensity; behavioural avoidance or attraction; policy-driven access changes	Modulate human/livestock behaviour (e.g., access design; grazing management; seasonal restrictions; behavioural interventions; infrastructure placement)

Regulation latency	Delay between emergence of hazard/exposure and effective suppressive or stabilising regulation	Time lag to predator establishment; delay in veterinary intervention; time to immune or community recovery; lag in management response or policy enforcement	Reduce delays in regulatory response (e.g., proactive predator reintroduction; early-warning surveillance; rapid adaptive management; pre-emptive interventions)

Box 1 | From coupling regimes to predictive alignment modelling.

A. Conceptual principle

Realised infectious disease risk emerges from the dynamic alignment of hazard generation, exposure pathways, and regulatory processes. Predicting when and where risk occurs therefore requires quantifying how these components co-vary through time and space under constraints imposed by coupling regimes. The objective is not to model hazard, exposure, or regulation in isolation, but to infer when their joint configuration permits transmission-favourable alignment

B. Operational workflow

Step 1: Characterise system components (H, E, R)

Quantify baseline state and dynamics of the three process classes.

Hazard: pathogen/vector/host presence, abundance, or environmental suitability

Exposure: contact processes driven by movement, behaviour, land use, or ecological interfaces

Regulation: processes that reduce transmission, including predation, immunity, management, and control

Step 2: Infer coupling-regime dimensions (analytically distinguishable system properties)

Assess four independent structural dimensions governing system dynamics:

Response-time hierarchy: ordering of temporal responses in H, E, and R

Spatial coupling structure: degree of spatial co-location versus movement-mediated connectivity among H, E, and R

Exposure elasticity: sensitivity of exposure pathways to ecological or socio-environmental change

Regulation latency: delay between emergence of H–E conditions and effective regulatory response

Step 3: Simulate spatiotemporal alignment dynamics

Model or infer how H, E, and R evolve under environmental or management change while preserving empirically derived coupling constraints.

Output: time–space trajectories of H, E, and R.

Step 4: Identify alignment windows

Derive periods and locations where:

- H is sufficiently high and
- E enables contact with susceptible hosts and
- R is delayed, weak, or spatially disconnected

These alignment windows represent conditions under which transmission becomes possible.

Step 5: Evaluate intervention scenarios

Assess how restoration or management intervention modifies coupling-regime structure and therefore alignment outcomes by altering:

- response-time hierarchy (e.g., accelerating or delaying ecological recovery)
- spatial coupling structure (e.g., changing habitat–use overlap)
- exposure elasticity (e.g., modifying behavioural or land-use responsiveness)
- regulation latency (e.g., improving speed of ecological or managerial response)

Output: comparative scenario evaluation of alignment risk.

C. Key implication

Coupling regimes define the structural constraints on system behaviour, while alignment windows and trajectories describe the emergent conditions under which transmission is realised. Predictive disease ecology therefore shifts from estimating risk factors alone to inferring when system dynamics permit alignment.

D. Implementation outlook

This workflow is compatible with multiple modelling and inference approaches, including:

- mechanistic and spatially explicit transmission models

- state-space and Bayesian dynamic systems
- agent-based models of host–vector–human interaction
- scenario-based simulation frameworks

Emerging “digital twin” approaches may integrate these components by continuously updating system states (H, E, R) from ecological and social data streams to evaluate alignment under alternative restoration or management scenarios.

Comparative support for the framework would arise if coupling-regime dimensions predict the timing, duration or spatial extent of disease outcomes better than hazard, exposure or vulnerability metrics alone.

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