

Disease Regulation as a Missing Ecosystem Service in Restoration Planning

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Disease Regulation as a Missing Ecosystem Service in Restoration Planning

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Abstract

Ecosystem restoration is central to global biodiversity and sustainability commitments, yet its consequences for zoonotic disease risk remain poorly characterised. Assuming restoration uniformly reduces spillover risk overlooks how transmission ecology shapes the relationship between landscape recovery and disease outcomes. Here we use Monte Carlo Bayesian belief network simulations to model disease-hazard trajectories across four zoonotic transmission systems: mosquito-borne, tick-borne, rodent-borne, and direct-contact multi-host zoonoses, under increasing restoration intensity and realistic funding scenarios. Rodent-borne disease risk declines monotonically with restoration intensity, while vector-borne systems exhibit pronounced transitional risk amplification before declining under more complete restoration. Funding simulations reveal that delayed investment and mid-project funding collapse substantially erode health co-benefits, particularly in vector-borne systems where transitional risk windows are time-sensitive. Partial restoration consistently sustained transmission by increasing host connectivity, vector habitat suitability, or spillover interfaces before regulatory processes were established. These findings challenge the assumption that health co-benefits emerge proportionally with ecological recovery and provide a framework for integrating disease-specific surveillance into restoration policy, identifying disease regulation as a critical and undervalued ecosystem service.

Introduction

Landscape restoration, the process of assisting the recovery of an ecosystem that has been degraded, damaged, or destroyed in regaining ecological functionality and enhancing human well-being¹, is increasingly promoted as a critical strategy for biodiversity conservation, ecosystem resilience, climate change mitigation, and sustainable development^{2–5}. Alongside this, disease regulation is increasingly recognized as a critical ecosystem service provided by intact ecosystems⁶. The SARS-CoV-2 pandemic has renewed interest in understanding whether ecosystem restoration can recover disease-regulating functions and reduce the risk of future zoonotic emergence. Restoration interventions such as reforestation, wetland rehabilitation, and agroecological transitions can enhance ecosystem functioning, improve habitat connectivity, and support biodiversity recovery. However, restoration activities may also influence zoonotic disease dynamics, particularly during ecological transition phases when host communities, vector populations, habitat structure, and human-wildlife interactions undergo substantial reorganization^{7–9}. Given the scale of restoration commitments under the Bonn Challenge and Kunming-Montreal 30×30 target, there is an urgent need to understand how restored ecosystems transition to disease stable states, transforming restoration from a disease risk mitigation exercise into a strategy for restoring disease regulation as a core ecosystem service.

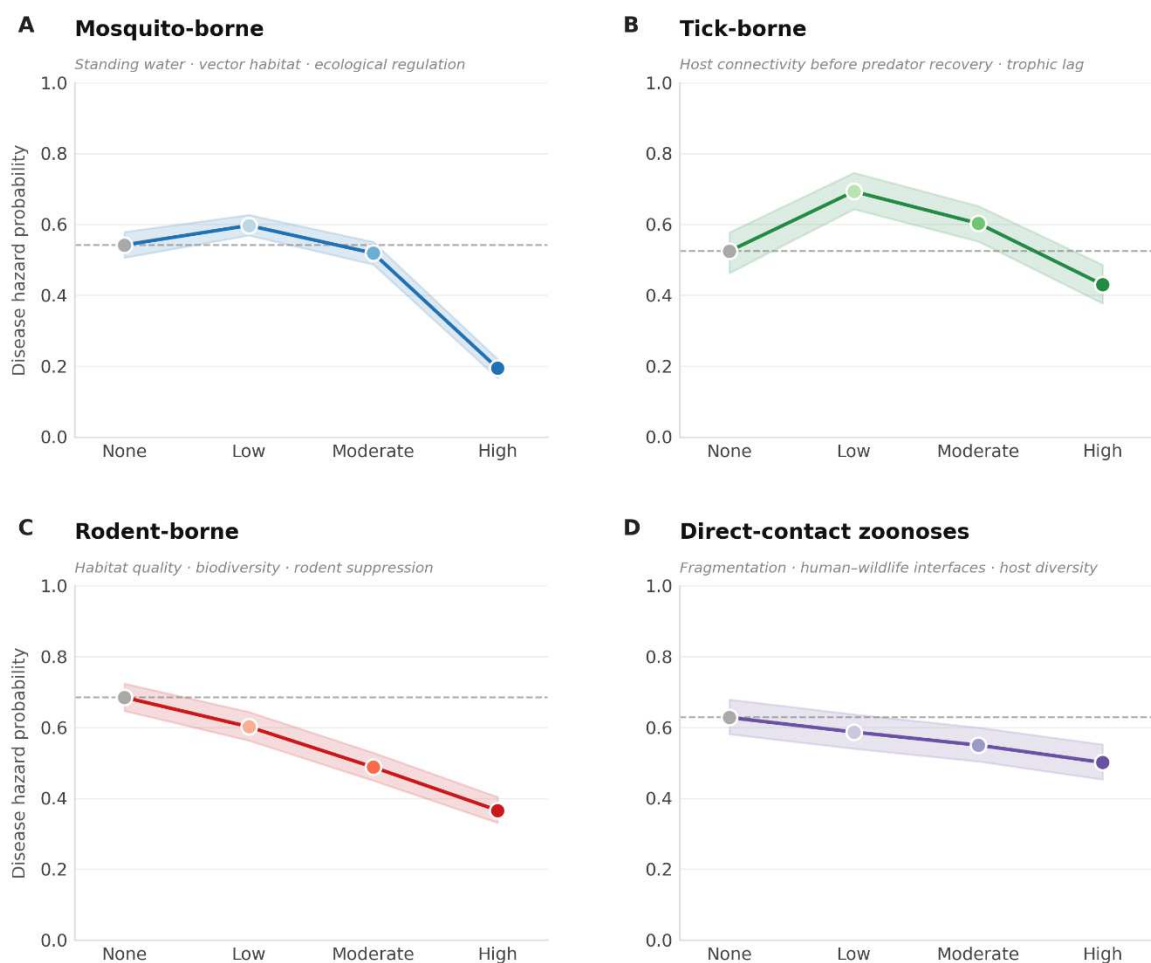
The relationship between restoration and zoonotic disease risk is highly complex and often nonlinear. Ecological restoration may reduce long-term disease risks by restoring trophic regulation, increasing biodiversity, and stabilizing ecosystem processes^{10,11}. Conversely, restoration may create temporary ecological conditions that favor pathogen transmission, including altered host abundance, increased vector habitat suitability, or increased human exposure during transitional recovery periods^{12,13}. These dynamics are likely to differ substantially among disease systems. For example, rodent-borne diseases are often associated with habitat simplification and altered host community composition and interactions, such that restoration may reduce disease risk by promoting biodiversity recovery and reducing reservoir dominance, while^{14,15} tick-borne diseases may respond more slowly than rodent borne diseases because restoration can initially increase host connectivity and opportunities for transmission before ecological regulation emerges¹⁶. The physical manifestation of the environment is also a key factor in driving changes; mosquito-borne diseases are strongly shaped by hydrological change, with restoration potentially creating temporary vector habitat before mature ecosystems suppress transmission⁹, while direct-contact zoonoses are influenced by disturbance-driven wildlife–human interfaces, which restoration may reduce through decreasing habitat fragmentation and contact rates between wildlife, domestic animals and humans¹².

Understanding these interactions is essential for designing restoration strategies that maximise ecological benefits while minimising unintended public health risks within a One Health framework. However, empirical evidence linking restoration trajectories and zoonotic disease emergence remains limited, highly context dependent, fragmented across ecosystem type and recovery stage, and characterized by substantial uncertainty¹⁷.

To provide greater clarity on this highly important and timely issue, we developed disease-specific Bayesian Belief Network models for rodent-, tick-, and mosquito-borne diseases to evaluate how restoration interventions influence zoonotic disease dynamics and long-term ecosystem stabilisation trajectories. The models integrated ecological recovery processes, biodiversity dynamics, host or vector responses, and regional risk contexts within a probabilistic Monte Carlo simulation framework. We use Monte Carlo sampling to propagate uncertainty through conditional probability tables and generate distributions of possible restoration outcomes rather than single deterministic predictions. Each simulation iteration represents a plausible ecological trajectory under a given restoration scenario, allowing uncertainty in ecological recovery and disease dynamics to be explicitly quantified. Specifically, we evaluated how restoration interventions alter disease-hazard trajectories across rodent-, tick-, and mosquito-borne systems and estimate the probability and timing of ecological stabilization under restoration versus no-restoration scenarios. We further quantified trade-offs among restoration intensity, zoonotic disease risk, ecological regulation, and long-term ecosystem stabilization under uncertainty. By integrating restoration ecology, disease ecology, and Bayesian scenario modelling, we identified the conditions under which restoration is most likely to reduce disease-hazard, reveal potential transitional risk-amplification phases during ecosystem recovery, and evaluate how restoration trajectories influence the emergence of disease-regulating ecosystem services.

Results

Throughout the results, disease risk represents transmission hazard, the probability that ecological conditions favour pathogen transmission under a given restoration scenario, rather than individual human disease incidence, which would additionally require site-specific exposure data not captured at this landscape-modelling scale. Monte Carlo Bayesian Belief Network simulations revealed that ecological restoration consistently increased regulation and long-term ecosystem stabilisation across all four zoonotic disease systems, but that the direction, magnitude, and trajectory of disease-hazard responses were strongly mediated by transmission ecology (Figure 1).



Shaded bands: 95% Monte Carlo credible intervals ($n = 1,000$ iterations). Dashed lines: no-restoration baseline hazard. All panels conditioned on high-risk region.

Figure 1: Restoration-mediated disease hazard trajectories across four disease systems. Each panel reflects system-specific (A. Mosquito-borne; B. Tick-borne; C. Rodent-borne; D. Direct-contact zoonoses) ecological mechanisms. Shaded bands represent 95% Monte Carlo credible intervals. Dotted line indicates the disease risk probability at the start point of simulations under a “no restoration” scenario

Restoration produced three distinct risk trajectories across disease systems. Rodent-borne diseases showed a monotonic and comparatively steep risk decline across all restoration levels, from a mean disease hazard probability of 0.69 (95% UI: 0.65–0.72) under no restoration to 0.37 (95% UI: 0.33–0.40) under high restoration, consistent with a direct pathway in which habitat quality improvements progressively suppress rodent reservoir dominance. Direct-contact multi-host zoonoses also declined monotonically, but far more gradually and with substantially wider uncertainty intervals (from 0.63 to 0.50), reflecting the diffuse influence of restoration on fragmentation, edge effects, and human–wildlife contact rates in systems where host diversity effects remain weak and unpredictable. In contrast, both vector-borne systems exhibited pronounced non-monotonic responses. Mosquito-borne disease hazard peaked at low restoration (mean 0.60; 95% UI: 0.57–0.63) before declining sharply to 0.19 (95% UI: 0.17–0.22) under high restoration, the largest absolute reduction observed across all systems and the only scenario in which hazard fell substantially below the no-restoration baseline. Tick-borne diseases followed a shallower version of the same pattern, with a hazard peak at low restoration (0.69; 95% UI: 0.64–0.75)

that persisted into moderate restoration (0.60) before declining to 0.43 under high restoration.

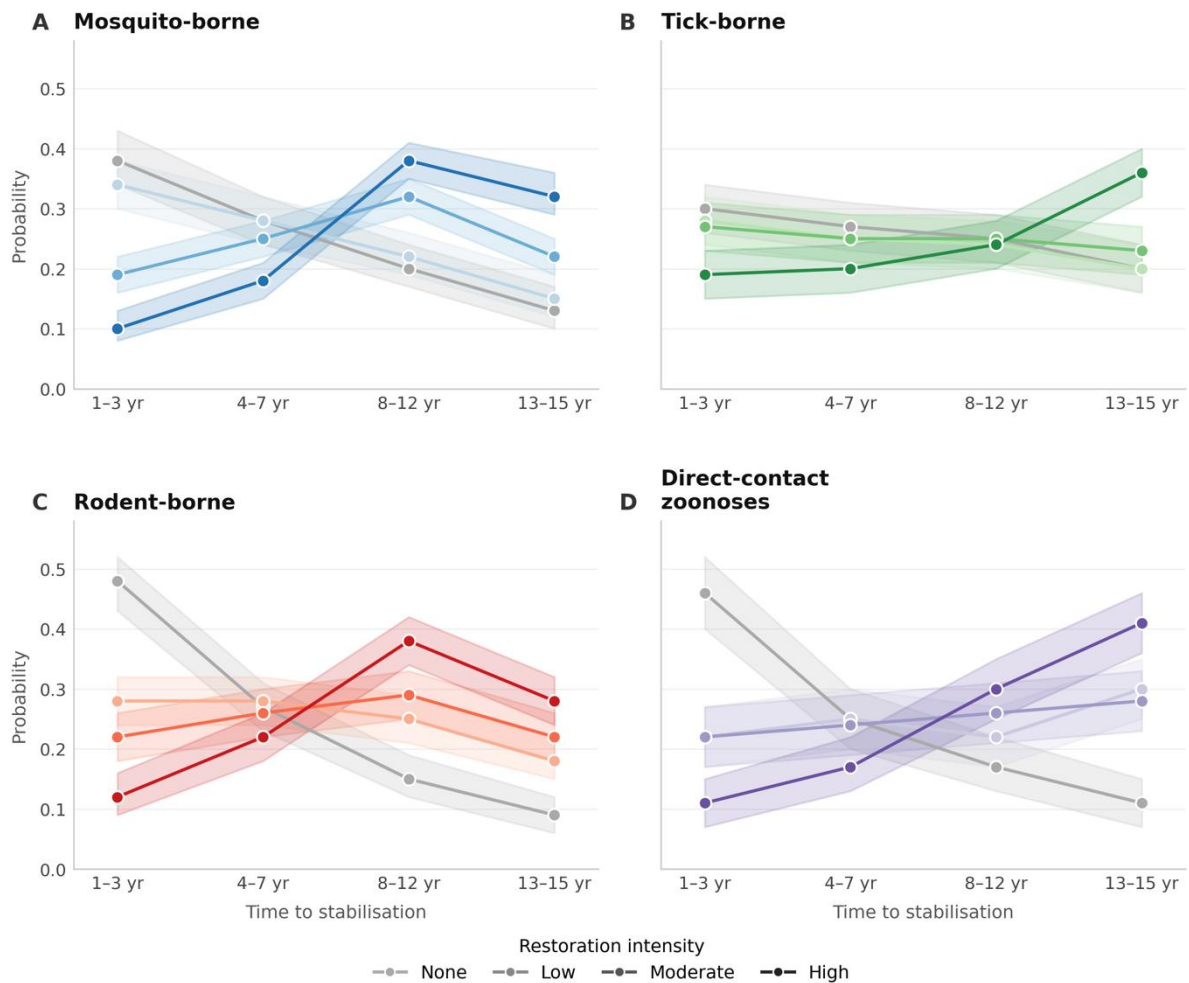
Transitional hazard amplification was greatest where ecological lag was longest. The hazard humps observed in mosquito- and tick-borne systems arose through pathways that are structurally analogous, partial restoration enhancing transmission before top-down regulation re-establishes, but ecologically distinct in the predator communities involved and the timescales over which they recover (Figure 1). In both cases the underlying mechanism is predator-mediated regulation, but the relevant predator guilds differ fundamentally. In mosquito systems, suppression of vectorial capacity depends on the recovery of aquatic and semi-aquatic predator assemblages, including larval-feeding invertebrates, amphibians, and insectivorous wetland birds, whose establishment tracks vegetation succession and hydrological maturation. Early-stage hydrological re-wetting creates abundant standing water and peak vector habitat before these communities have assembled, generating the hazard hump. Because the regulatory mechanism is tied to hydrological and vegetation dynamics that respond relatively quickly to restoration intervention, the hump is shorter in duration and smaller in amplitude. In tick systems, the relevant predator guild operates at a higher trophic level, medium and large mammalian predators whose return drives trophic cascades that suppress deer and rodent host populations and thereby reduce tick burden. The recovery of these predator communities lags vegetation and host recovery by years to decades, producing a more prolonged and pronounced transitional hazard window. Accordingly, the probability of strong ecological regulation, queried from the Ecological_Regulation node, remained at 0.05 under no restoration and 0.10 under low restoration in mosquito systems, rising to 0.75 only under high restoration (Table 1). In tick systems the equivalent probability remained low through both low (0.20) and moderate (0.27) restoration, reaching only 0.46 under high restoration, reflecting the slower return of mammalian predator guilds relative to aquatic and semi-aquatic assemblages. In both cases, the transitional hazard window corresponds precisely to the period before the relevant predator guild has recovered sufficiently to exert top-down control, and intermediate restoration stages therefore represent a period of elevated zoonotic hazard rather than a safe intermediate state.

High restoration consistently delivered the strongest ecological and epidemiological outcomes, but with system-specific delays. Probabilities of strong ecological regulation reached 0.72–0.75 under high restoration in rodent, mosquito, and direct-contact systems, and 0.46 in tick systems, where trophic recovery is inherently slower. Long-term stabilisation (8–15 years; Figure 2) increased two- to threefold across all systems under high restoration relative to no restoration. The temporal pathway to these outcomes differed substantially: rodent systems stabilised predominantly within the 4–12 year window, mosquito systems within 8–12 years following hydrological maturation, tick systems most strongly in the 13–15 year window reflecting the decadal timescales of trophic cascade establishment, and direct-contact zoonotic systems showed persistently broad stabilisation distributions consistent with weak ecological predictability.

Table 1: Uncertainty structure among the four disease systems and restoration intensities for disease hazard probability and long-term ecological stabilisation (cumulative probability of stabilisation within the 8–15 year window, representing the likelihood that the ecosystem has reached a self-sustaining configuration with established ecological regulation within this policy-relevant timeframe).

Scenario	Mosquito (95% uncertainty interval)		Tick (95% uncertainty interval)		Rodent (95% uncertainty interval)		Multi-host zoonoses (95% uncertainty interval)	
	Disease hazard	Ecological stabilisation	Disease hazard	Ecological stabilisation	Disease hazard	Ecological stabilisation	Disease hazard	Ecological stabilisation
No Restoration	0.54 (0.51-0.57)	0.29 (0.25-0.34)	0.52 (0.46-0.58)	0.44 (0.39-0.49)	0.69 (0.65-0.72)	0.24 (0.20-0.28)	0.63 (0.58-0.68)	0.23 (0.20-0.27)
Low Restoration	0.60 (0.57-0.63)	0.37 (0.33-0.41)	0.69 (0.64-0.75)	0.41 (0.37-0.46)	0.60 (0.56-0.64)	0.33 (0.28-0.38)	0.59 (0.54-0.64)	0.53 (0.48-0.58)
Moderate Restoration	0.52 (0.49-0.55)	0.54 (0.50-0.57)	0.60 (0.55-0.65)	0.47 (0.43-0.51)	0.49 (0.45-0.53)	0.46 (0.42-0.50)	0.55 (0.50-0.60)	0.53 (0.48-0.58)
High Restoration	0.19 (0.17-0.22)	0.70 (0.65-0.74)	0.43 (0.38-0.49)	0.61 (0.56-0.64)	0.37 (0.33-0.40)	0.67 (0.62-0.71)	0.50 (0.45-0.54)	0.72 (0.68-0.76)

These temporal signatures carry practical implications; ecosystems undergoing partial or recently initiated restoration may simultaneously exhibit heightened zoonotic hazard and measurable gains in ecological regulation, creating a period in which standard ecological recovery metrics do not reliably indicate disease-hazard status.



Lines = mean stabilisation probability (1,000 MC iterations). Shaded ribbons = 95% uncertainty interval.
All scenarios conditioned on high-risk region context. Shared y-axis scale across panels.

Figure 2: Ecological stabilisation trajectories across restoration intensities and disease systems. Probability of ecosystem stabilisation within each temporal window (1–3, 4–7, 8–12, and 13–15 years) under four restoration intensities (none, low, moderate, high), derived from the *Time_to_Stable_State* node of each Bayesian belief network model. Lines represent mean stabilisation probabilities across 1,000 Monte Carlo iterations; shaded ribbons represent 95% uncertainty intervals

Uncertainty was greatest in transitional and multi-host systems. The narrowest credible intervals were in rodent systems (Table 1), indicating relatively consistent reductions in disease hazard and increases in ecological regulation with restoration. Tick-borne and direct-contact zoonotic systems showed broader uncertainty ranges, particularly under low and moderate restoration intensities, reflecting greater sensitivity to host connectivity, wildlife movement, and contact dynamics. Mosquito-borne diseases displayed the strongest contrast between restoration states, with relatively narrow uncertainty intervals around high-restoration outcomes but greater variability during transitional phases, consistent with the influence of hydrological conditions and vector habitat dynamics. Across all systems, uncertainty generally decreased under high-intensity restoration, suggesting that more complete ecological recovery produces more predictable long-term outcomes than either degraded or partially restored landscapes.

High restoration consistently delivered the strongest ecological and epidemiological outcomes, but with system-specific delays. We resolved this joint relationship between disease-hazard reduction and long term ecological recovery by mapping individual simulation iterations within a trade-off probability space (Supplementary Figure 1). Across 1,000 Monte Carlo iterations, high-intensity restoration consistently clusters in the optimal upper-left quadrant (low hazard, high stabilization) across all four models, though the degree of cluster separation varies substantially by system. The rodent-borne system displays the clearest, most linear separation between restoration intensities, reflecting its direct habitat-to-reservoir suppression pathway. In contrast, vector-borne systems show distinct cluster structures reflecting their transitional hazard peaks at lower restoration intensities, while direct-contact zoonoses display horizontal overlap across the hazard axis, confirming that epidemiological hazard and ecological stabilisation are only weakly coupled in complex multi-host systems.

Funding scenario simulations revealed that transition timing critically determines health outcomes. Sequential restoration simulations revealed that the timing of funding transitions substantially influenced disease-hazard outcomes, with effects that were highly system-specific and asymmetric between ramp-up and funding collapse scenarios (Figure 3). Across vector-borne systems, early transitions to high-intensity restoration produced disproportionate benefits relative to late transitions. In mosquito-borne systems, transitioning from moderate to high restoration at Year 5 produced a mean disease hazard of 0.348 (95% UI: 0.325–0.371), compared with 0.500 (95% UI: 0.473–0.529) when the same transition was delayed to Year 10, a difference of 0.152 units approaching the magnitude of the difference between sustained low and high restoration, reflecting the hump mechanism whereby earlier intervention shortens the time spent in the peak-hazard transitional window. In tick-borne systems, the cost of delayed investment was even more pronounced: low-to-high restoration transitioning at Year 10 produced mean hazard of 0.674 (95% UI: 0.625–0.723), barely distinguishable from the sustained low-restoration baseline, consistent with the decadal timescales required for trophic cascade establishment.

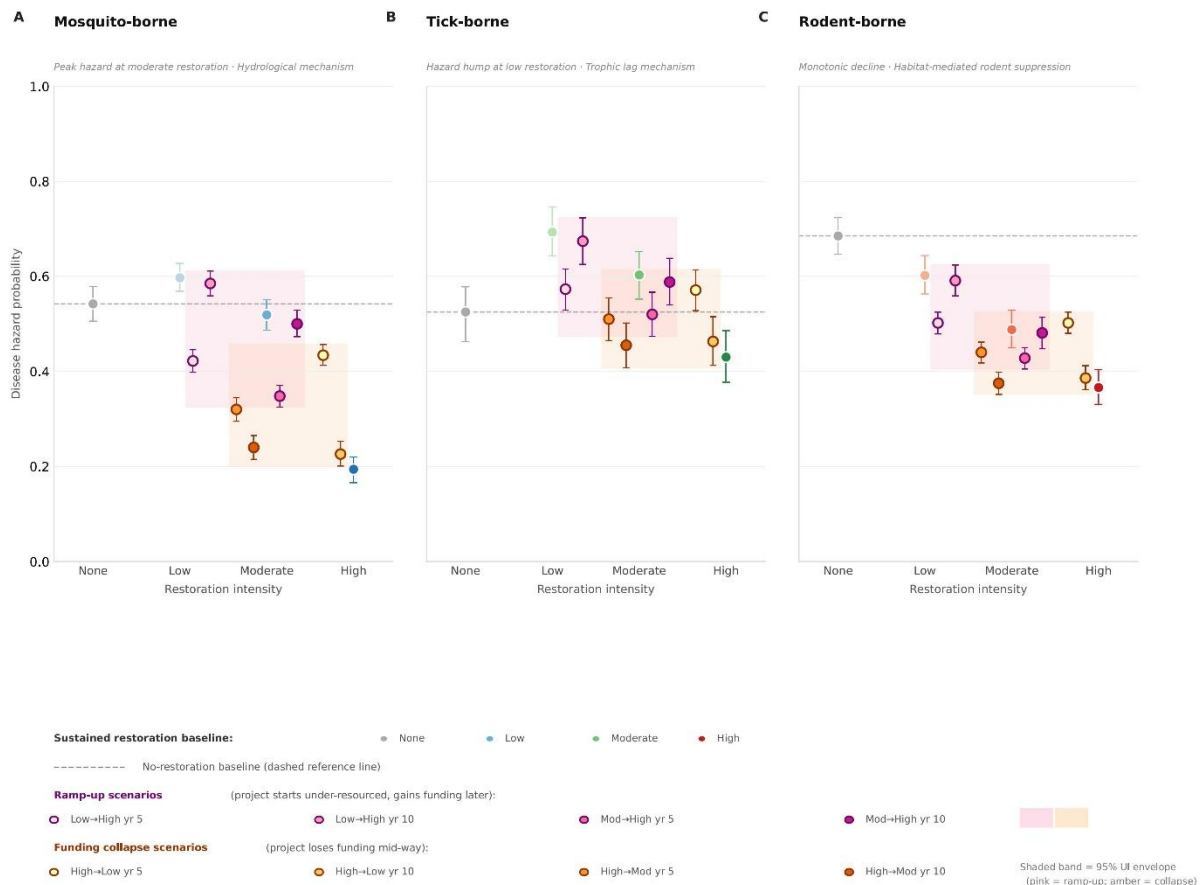


Figure 3 Each panel shows mean disease hazard probability (y-axis) across restoration intensity levels (x-axis: None, Low, Moderate, High) for mosquito-borne (A), tick-borne (B), and rodent-borne (C) disease systems. The dashed horizontal line in each panel marks the no-restoration baseline, the expected disease hazard in the absence of any intervention, against which all scenarios are compared. Points falling below this line indicate a net disease-hazard benefit relative to no restoration while points above indicate elevated hazard. Purple points show ramp-up scenarios: open circles represent transition from low to high-intensity restoration at Year 5 and Year 10; filled circles represent the transition from moderate to high intensity restoration at Year 5 (lighter) and Year 10 (darker). Yellow points show funding collapse scenarios: open circles = collapse from high to low funding scenarios at Year 5 (very pale) and Year 10 (pale); filled circles = collapse from high to moderate funding scenarios at Year 5 (lighter) and Year 10 (darker). Shaded rectangles show the 95% uncertainty interval envelope across all ramp-up (purple) and collapse (yellow) scenarios within each restoration intensity group. Results for direct-contact multi-host zoonoses are presented in Supplementary Figure S3

Funding collapse scenarios revealed marked asymmetries in ecosystem resilience across systems. In mosquito-borne diseases, collapse to low intensity at Year 5 (mean 0.434; 95% UI: 0.413–0.457) produced outcomes nearly as poor as sustained low restoration, whereas collapse at Year 10 (0.226; 95% UI: 0.201–0.253) remained close to the sustained high-restoration baseline (0.190; 95% UI: 0.164–0.216), suggesting a critical consolidation threshold between Years 5 and 10 after which ecological regulation becomes sufficiently self-sustaining to buffer against funding withdrawal. Rodent-borne systems showed greater resilience: high-to-low transition at Year 10 (0.386; 95% UI: 0.362–0.412) remained near the sustained high baseline (0.366), reflecting the relative persistence of habitat quality gains under reduced management intensity. Direct-contact zoonotic systems showed weak and partially counterintuitive responses across all funding transition scenarios (Supplementary Figure S2), consistent with the broad uncertainty and weak restoration sensitivity characteristic of multi-host spillover dynamics, in which human behavioural factors, host community composition, and stochastic contact dynamics exert influence comparable to or greater than landscape-scale restoration intensity. Results for this system should be treated

as indicative of the direction of effect rather than reliable quantitative predictions and are presented in full in Supplementary Figure S2.

Sensitivity analysis confirmed the robustness of cross-system comparisons. Model outputs were most strongly influenced by parameters governing the final transmission step, the probability of disease emergence given prevailing ecological conditions, rather than by upstream restoration or biodiversity drivers (Supplementary data Section 6, Figure S3). This pattern was consistent across all four systems, and parameters representing habitat quality, host diversity, hydrological recovery, and landscape connectivity contributed comparatively little variance to overall outcomes. The relative ordering of restoration trajectories is therefore robust to uncertainty in ecological assumptions and reducing predictive uncertainty in future restoration–health models will depend primarily on improving empirical understanding of transmission-interface dynamics.

In order to operationalise the framework for practitioners, we have developed an open-access decision-support tool. We implemented the Bayesian belief network simulations as the RESTOREID DST, an interactive browser-based decision-support tool accessible at <https://restoreid-eu.github.io/BBN/> or through the “Results” tab of the RESTOREID webpage (<https://restoreid.eu>). The tool allows users to select their disease transmission system of interest, restoration intensity scenario, and funding transition year, returning disease hazard trajectories and ecological stabilisation probabilities with 95% uncertainty intervals in real time. All four disease systems and the full suite of funding scenarios are accessible through the interface, enabling direct comparison of restoration pathways without requiring engagement with the underlying model code.

Discussion

Across all disease systems examined, restoration fundamentally reshaped the trajectory by which ecosystems approached stable states. Rather than accelerating stabilisation, restoration consistently delayed it while eventually producing higher ecological regulation and greater long-term stability than non-restored landscapes. This pattern likely reflects a fundamental property of ecological recovery: as biodiversity, trophic interactions, and connectivity increase, ecosystems become slower to return to equilibrium but more capable of sustaining ecological function over time. Classical complexity-stability theory predicts exactly this trade-off^{18,19}, and empirical work on trophic recovery and biodiversity-resilience relationships supports it^{20–22}. The delayed stabilisation observed here should therefore be interpreted as a signature of genuine ecological recovery rather than a management failure, a distinction with direct implications for how restoration success is evaluated in practice.

The effects of restoration on disease hazard across different disease types were more heterogeneous, and more ecologically instructive. Rodent-borne diseases declined monotonically with restoration intensity, consistent with progressive suppression of disturbance-adapted reservoir hosts through biodiversity recovery and trophic restructuring^{23,24}. Outcomes for vector-borne systems were more complex: Both tick- and mosquito-borne diseases exhibited transitional risk amplification at intermediate restoration stages, before declining substantially under high-intensity restoration. For ticks, increased host connectivity preceded predator recovery, while for mosquitoes, hydrological re-wetting

created peak vector habitat before mature ecological regulation emerged^{25,26}. These dynamics align with ecological succession theory, which predicts that recovering ecosystems pass through transitional states that differ substantially from both degraded and mature conditions^{18,27,28}. The practical implication is that disease outcomes can lag behind ecological recovery indicators, demonstrating that restoration sites may simultaneously score well on biodiversity metrics while remaining in elevated-risk transmission windows.

This finding points to a more general concern about partial restoration. Low and moderate restoration intensities consistently produced higher disease hazard than either extensive restoration or continued degradation, suggesting that incomplete interventions may sustain pathogen transmission while simultaneously increasing habitat suitability for vectors or spillover hosts²⁹. Landscapes trapped in transitional ecological states, where there is sufficient disturbance to maintain transmission interfaces but insufficient recovery to re-establish regulatory processes, may represent a previously underappreciated disease-hazard category. This systemic risk is visually striking in the joint hazard–stabilization probability space (Supplementary Figure 1). The trade-off trends illustrate that low and moderate restoration intensities risk trapping landscapes in high-hazard, low-stabilisation situations, particularly in vector-borne systems where partial intervention sustains vector habitat before regulatory predator guilds can recover. These distributions demonstrate that the degree of ecological recovery achieved matters as much as the area being restored; fragmented or low-intensity interventions that fail to transition ecosystems out of these intermediate, high-hazard states may yield negligible public health co-benefits compared to the robust, low-hazard equilibria achieved under high-intensity, landscape-scale restoration.

The funding scenario simulations suggest two policy-relevant conclusions. First, early investment in high-intensity restoration produces substantially greater disease-hazard reductions than equivalent investment delayed by five years, particularly in vector-borne systems where transitional risk amplification creates a time-sensitive window. Second, the resilience of achieved disease-hazard reductions to funding withdrawal is system-specific and time-dependent: in mosquito systems a consolidation threshold appears to exist around Years 5–10 after which moderate funding reductions are less damaging, while in tick systems the slow pace of trophic recovery means that even late-stage funding collapse substantially compromises outcomes within the 15-year horizon. These findings argue for front-loaded, sustained restoration investment as the most effective strategy for realising zoonotic disease co-benefits, and against staged or uncertain funding commitments that risk locking restoration projects into the transitional ecological states associated with elevated disease risk.

The development of RESTOREID DST (Figure 4) as an open-access, browser-based decision-support interface responds directly to a persistent gap in the translation of ecological modelling into restoration practice. Current restoration planning tools are predominantly focused on biodiversity, carbon, and ecosystem-service metrics, with One Health largely absent from decision frameworks despite growing recognition of zoonotic risk as a restoration-relevant outcome^{17,30}. By making the scenario outputs of the BBN models interactively explorable, we enable stakeholders to move beyond the aggregate patterns reported here and query system-specific outcomes relevant to their local context. Critically, the interface is designed to communicate uncertainty explicitly rather than presenting point

predictions, consistent with the hypothesis-generating rather than predictive scope of the underlying models and with established principles for risk communication in environmental decision-making. We suggest that tools such as this that embed probabilistic, mechanism-explicit disease-hazard outputs within accessible planning interfaces represent a practical pathway toward the integration of One Health principles into the operational workflows of the UN Decade on Ecosystem Restoration and the Kunming-Montreal Global Biodiversity Framework, where health co-benefit monitoring remains largely aspirational rather than operational.



Figure 4: RESTOREID Decision support tool landing page. The decision support tool allows users to start with the landscape context they are interested in, and then explore how restoration impacts hazard across different host-pathogen disease systems. The app is hosted in ShinyLive and runs a pre-loaded model simulation.

Collectively, these results reinforce calls to integrate health considerations into restoration planning from the outset. Adaptive monitoring that incorporates zoonotic risk indicators alongside ecological indicators, particularly during the transitional phases identified here, could help practitioners identify temporary risk-amplification windows and adjust management accordingly³¹. However, to do this effectively, clarification is needed on the relationship between transmission dynamics and ecosystem recovery, highlighted by this work as driving much of the uncertainty within the system. From this, markers of biodiversity recovery can be devised that can then be incorporated into monitoring within adaptive management programs. More broadly, the finding that restoration reshapes rather than simply reduces disease risk underscores the need for One Health frameworks that explicitly account for transmission ecology, temporal dynamics, and ecological mechanism rather than treating restoration as a uniformly protective intervention.

Our framework represents generalised transmission archetypes rather than individual pathogens, and therefore does not capture the full ecological heterogeneity within each disease system. Consequently, the model is most applicable to systems where the dominant transmission pathways align with the underlying BBN structure, as highlighted in the ecosystem map provided in the RESTOREID DST. Applying the framework to ecologically distinct systems, such as tropical forest malaria or environmentally mediated pathogens,

would require disease- and region-specific re-parameterisation using local empirical data (further details in the supplementary section S9). The uncertainty envelopes across several scenarios reflect genuine ecological complexity. Restoration outcomes vary with local species assemblages, pathogen biology, environmental context, and socioeconomic conditions. The Bayesian Belief Network framework captures this uncertainty explicitly, but the simulations remain hypothesis-generating rather than predictive. Empirical validation using long-term restoration projects with paired disease surveillance data is a clear priority. Coupling probabilistic models with spatially explicit ecological and epidemiological frameworks would further advance understanding of how restoration-health interactions vary across landscapes and under future climate and land-use trajectories.

However, we clearly demonstrate that restoration does not simply reduce or increase zoonotic disease risk. Rather, it reshapes the ecological pathways through which disease-hazard emerges. Delayed stabilisation, transient risk amplification, and system-specific transmission dynamics all point to the same conclusion that health outcomes depend not only on whether restoration occurs, but on its intensity, completeness, and the timescale of ecological recovery. Embedding these dynamics into restoration planning will be essential for realising the health co-benefits that One Health frameworks promise.

Methodology

We constructed a series of Bayesian Belief Networks (BBNs) to model probabilistic relationships between restoration processes and zoonotic disease dynamics for rodent-, tick-, mosquito-borne and direct-contact zoonotic disease systems. BBNs represent complex systems as networks of interconnected variables linked through conditional probabilities, allowing uncertainty and multiple sources of evidence to be incorporated within a single framework. Networks were implemented in R using the “bnlearn”³² and “gRain”³³ packages. Directed acyclic graphs (DAGs) were developed to represent causal relationships among ecological, environmental, epidemiological, and restoration-related variables³⁴, while conditional probability tables (CPTs) quantified the strength of these relationships. Disease-specific network structures were developed to capture the distinct ecological mechanisms underlying transmission in each disease system.

Disease-Specific Ecological Structures

Although all models shared a common restoration–health framework, disease-specific ecological pathways were incorporated to reflect differences in transmission ecology and expected responses to restoration. We selected four representative zoonotic disease systems that capture major transmission pathways relevant to restoration landscapes: rodent-borne diseases, tick-borne diseases, mosquito-borne diseases, and direct-contact multi-host zoonoses. Disease-specific BBN structures (Table 2) were developed to represent the dominant ecological mechanisms linking restoration processes to disease risk and ecosystem stabilization.

Table 2: Summary of the four zoonotic disease systems represented in the Bayesian Belief Networks (BBNs), including example pathogens, dominant transmission pathways, and the key ecological mechanisms through which restoration is hypothesized to influence disease risk, ecological regulation, and ecosystem stabilization.

Disease system	Example pathogens	Primary transmission pathway	Key restoration mechanisms represented
Rodent-borne diseases	Hantaviruses, Arenaviruses such as Lassa virus	Reservoir host transmission	Biodiversity recovery, reduced rodent dominance, habitat quality improvement, trophic regulation
Tick-borne diseases	Lyme disease, Tick-borne encephalitis virus, Crimean-Congo haemorrhagic fever virus	Vector-borne transmission	Host connectivity, habitat fragmentation, successional dynamics, delayed trophic regulation
Mosquito-borne diseases	West Nile virus, Rift Valley fever virus, Japanese encephalitis virus	Vector-borne transmission	Hydrological recovery, standing water availability, restoration intensity, ecological regulation
Direct-contact multi-host zoonoses	Nipah virus, henipaviruses, leptospirosis, multi-host wildlife–livestock spillover systems	Direct wildlife–human or wildlife–livestock transmission	Habitat disturbance, biodiversity recovery, human–wildlife contact rates, ecological regulation

Detailed descriptions of disease-specific network structures and associated ecological assumptions are provided in Supplementary Methods S1.

Data Collection and Expert Elicitation

We identified key environmental, ecological, and socioeconomic variables influencing zoonotic disease dynamics through a systematic literature review of peer-reviewed and grey literature¹⁷, expert elicitation surveys involving specialists in landscape ecology, epidemiology, conservation science, and One Health, and synthesis of restoration case studies and ecological recovery trajectories. We consulted experts through an online questionnaire (in supplementary material) to define causal relationships among variables, estimate the direction and magnitude of ecological effects, identify likely temporal stabilization trajectories and provide probabilistic estimates for restoration outcomes under varying ecological conditions.

Conditional Probability Tables and Uncertainty Representation

Conditional probability tables (CPTs) were parameterised using a hybrid approach combining literature synthesis and structured expert elicitation, following established guidelines for BBN development in ecological and environmental risk assessment^{35,36}. Where empirical data were insufficient to fully constrain parameters, expert elicitation was

used in conjunction with available evidence to define plausible probability ranges for each conditional scenario. Rather than assigning fixed point estimates, which would misrepresent the true state of ecological knowledge, we implemented a Monte Carlo sampling procedure in which CPT parameters were independently drawn from uniform distributions within their predefined ranges on each of 1,000 simulation iterations. Sampled values were floored at 0.01 to prevent zero-probability states and normalised to unity within each conditional probability vector prior to network fitting. This approach exploits a core strength of the BBN framework³⁷, the ability to combine uncertain knowledge from multiple sources while explicitly propagating that uncertainty through the network structure, and allows uncertainty-aware estimation of disease risk, restoration trajectories, ecological stabilisation times, and restoration-health trade-offs across all four disease systems.

For mosquito-borne, tick-borne, and rodent-borne systems, the disease outcome node represents transmission hazard, the conditional probability that ecological conditions are favourable for pathogen transmission given prevailing restoration and regulatory conditions, rather than individual human risk, which would additionally require specification of human exposure, population density, and behaviour. Human-wildlife contact is modelled explicitly only in the direct-contact system, where it constitutes the proximate transmission interface. This distinction follows the epidemiological convention of separating hazard characterisation from risk assessment³⁸ and reflects the appropriate scope of landscape-scale ecological modelling, which can characterise transmission-relevant ecological conditions but cannot replace site-specific exposure assessment. The framework is therefore most appropriately used to identify where restoration creates or suppresses transmission-favourable ecological conditions, informing targeted surveillance and exposure assessment, rather than to predict human disease incidence directly.

Ecological stabilisation was defined following the general recovery framework from a recent study³⁹, in which a disturbed ecological system is considered to have reached a stable state once its trajectory ceases substantial directional change, irrespective of whether that state resembles the pre-disturbance condition. In the present models, stabilisation is operationalised through the Time_to_Stable_State node, which estimates the probability that a given restoration trajectory has reached a self-sustaining ecological configuration, characterised by established ecological regulation, recovered trophic structure, and consistent disease-risk dynamics, within a given temporal window.

Monte Carlo Bayesian Simulations

We conducted Monte Carlo Bayesian simulations to generate probabilistic distributions of restoration outcomes across alternative scenarios. We performed 1000 simulation iterations for each disease system, with CPT values sampled at each iteration to account for parameter uncertainty and ecological variability. We incorporated region-specific priors to account for variation in baseline disease endemicity, landscape degradation, habitat fragmentation and governance capacity. These priors allowed simulation of differing ecological contexts and enabled comparison of restoration outcomes under low-, moderate-, and high-risk regional scenarios. Through scenario analyses, we compared restoration versus no-restoration trajectories, differing restoration intensities, regional risk contexts, and alternative ecological recovery pathways. Restoration intensity was treated as an ordered categorical

variable with four levels, none, low, moderate, and high, representing positions along the restorative continuum⁴⁰ and operationalised following the intervention continuum framework⁴¹ (further details under Supplementary Section 2). A temporal stabilization node was incorporated to estimate the probability of reaching a relatively stable ecological and disease-regulation state across four-time intervals: 1-3 years, 4-7 years, 8-12 years, and 13-15 years. These intervals were chosen to represent plausible management-relevant restoration horizons and to capture transitional recovery dynamics relevant to management and policy decision-making rather than complete ecosystem recovery. The attainment of complete ecosystem recovery may occur over several decades depending on ecosystem type. Long-term ecological stabilization, in this study, is defined as the combined probability of reaching this state within the 8–15 year window (the sum of the 8–12 year and 13–15 year probability windows), selected because this range captures the timescales over which trophic restructuring and biodiversity-mediated regulatory processes are expected to consolidate across the four disease systems modelled^{42,43}.

Trade-Off and Uncertainty Analyses

Trade-off curves were used to assess relationships between disease risk probabilities and long-term ecological stabilization under varying restoration scenarios. Uncertainty corridors summarized the distribution of Monte Carlo outcomes and highlighted the degree of variability associated with different restoration pathways.

Funding scenario simulations

To evaluate the disease-risk consequences of realistic funding constraints, we extended the four BBN models to simulate sequential restoration transitions within the 15-year modelling horizon. Two funding scenario types were specified: ramp-up scenarios, in which restoration begins at low or moderate intensity due to initial resource limitations and transitions to high intensity at Year 5 or Year 10 following funding consolidation, and funding collapse scenarios, in which restoration begins at high intensity and drops to low or moderate intensity at Year 5 or Year 10 following loss of resources. For each scenario, disease risk at the end of the 15-year horizon was estimated using an ecological legacy weighting approach. At the transition year, the ecosystem is not in a pristine state, it retains the ecological legacy of Phase 1 in proportion to how far its stabilisation trajectory had progressed under that restoration intensity. The legacy weight was defined as the cumulative probability of stabilisation by the transition year, derived from the Time_to_Stable_State posterior distribution under Phase 1 restoration. Final disease risk was computed as a weighted mixture of the Phase 1 and Phase 2 risk trajectories, scaled by the remaining years after the transition point. This formulation produces ecologically realistic outcomes: ecosystems that stabilised early under Phase 1 are harder to redirect under Phase 2, while those still in flux at the transition year respond more readily to the change in restoration intensity. All funding scenarios were run across 1,000 Monte Carlo iterations and queried under a high-risk regional context. Results are summarised as mean disease risk with 95% uncertainty intervals.

Sensitivity Analysis and Validation

One-at-a-time sensitivity analyses were conducted to identify variables exerting the strongest influence on disease risk and stabilization trajectories²⁶. Tornado plots were used

to assess node influence and uncertainty propagation across the networks (see supplementary information section 5, Supplementary Figure S2).

Model validation followed a qualitative pattern approach, in which the predicted disease-risk trajectories for each system were compared against published empirical evidence and ecological theory⁴⁴. Given the absence of long-term paired datasets linking restoration intensity to zoonotic disease risk across gradients, formal quantitative validation was not possible⁴⁴. Instead, predicted trajectories were assessed for consistency with the direction, shape, and mechanistic basis of patterns documented in the empirical literature for each transmission system³⁶. This approach is standard practice for expert-elicited Bayesian belief network models applied to complex ecological systems, where parameter uncertainty is substantial and system-specific mechanisms are established through theory and observational studies rather than controlled experiments^{35,36}. Full mapping of predicted trajectories to supporting empirical evidence is provided in Supplementary Section S7.

Decision-support tool

To facilitate practical application of the modelling framework, an interactive decision-support tool (RESTOREID DST) was developed in R Shiny and deployed as a browser-based application via ShinyLive, enabling user-side execution without server infrastructure (accessible at: <https://restoreid-eu.github.io/BBN/>). The tool allows users to select disease transmission system, regional risk context, restoration intensity, funding transition scenario and transition year, derived from the underlying Bayesian belief network models. RESTOREID DST is designed for structured scenario exploration and communication of uncertainty in restoration planning for policymakers rather than site-specific prediction, consistent with the hypothesis-generating scope of the BBN framework^{35,45}.

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