18(1) 94-116, Spring 2026

Journal of Animal Environment

(E-ISSN 2717-1396, ISSN 2717-1388)

Journal homepage: http://www.aejournal.ir

Original Research Paper

Environmental Drivers of Zoonotic Disease Emergence in Rapidly Urbanizing Regions

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Key Words

Zoonotic spillover, Urbanization, Environmental drivers, Habitat fragmentation, Climatic stress, Spatial simulation.

Abstract

The increasing rate of urbanization has been noted as a context for the emergence of newly recognized zoonotic diseases, but the specific impacts of the urban growth on the development of the disease spillover potential are not sufficiently understood. This research provides an original, fully simulation-based, spatially defined evaluation of the risk of zoonotic spillover given the simultaneous influence of urban, ecological, climate, and atmospheric factors. Through a geospatial and environmental modeling integration, the analysis determines the risk potential of urbanization and habitat fragmentation, and how they reconfigure the interfaces between the human and non-human environments, and the role of climate variability and atmospheric pollution as secondary risk factor amplifiers. Spatial surfaces of spillover potential created by the simulator demonstrate the consistent presence of peri-urban regions with high- risk potential, and fragmentation and highly humanized interfaces with scattered reservoir species. The inter-modeling of coupled stressor interactions indicates that climate and atmospheric pollution effects are the greatest when they are overlaid on

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Received: 24 November 2025; Reviewed: 02 January 2026; Revised: 19 January 2026; Accepted: 30 March 2026
(DOI): 10.70102/AEJ.2026.18.1.9

fragmented ecosystems, which results in an increased and disproportionate spillover potential. Furthermore, analysis of the attributed risk factors demonstrates that interaction effects surpass the individual explanatory potential of the risk factors, indicating that the emergence of spillover is an integrated outcome of complex systems rather than an additive result. The findings outline the mechanisms and spatial contributors to the emergence of zoonotic diseases in the urbanizing peri-urban ecosystems and the necessity of integrated and holistic land planning, ecological system integrity, and quality of the environment to manage the risk of spillover potential.

Introduction

In the last twenty years, the emergence and re-emergence of zoonotic diseases as a result of large-scale and rapid environmental transformations have increasingly attracted attention in the biosciences. Among the many forms of environmental change, rapid and sustained urbanization is one of the strongest drivers of transformation in ecological systems, human–environment interactions, and pathogen transmission pathways. In the last three decades, the pace of urban land expansion, especially in low- and middle-income regions of the world, has greatly exceeded the rate of global population growth, resulting in profound changes in land use, climate conditions, biodiversity structure, and overall environmental quality (McDonald et al., 2020). These changes have created novel, and in many cases unstable, systems of interaction among wildlife reservoirs, domestic animals, and dense human populations, which are favorable for pathogen spillover and sustained transmission (Wang et al., 2024).

Evidence from many geographical settings shows that the emergence of zoonotic diseases occurs more often in areas experiencing rapid urban sprawl than in older urban centers or undisturbed natural environments (Anugwom & Anugwom, 2023). This implies that the process of urbanization itself creates the environmental conditions that facilitate spillover, rather than the simple presence or absence of urban centers. The land-use changes associated with urban expansion cause habitat fragmentation, wildlife population compression, increased edge effects where human populations meet reservoir species, and greater human–wildlife contact (Ingegnoli et al., 2022). These changes are further intensified by the rapid construction of transport infrastructure, the spread of informal settlements, and changes in patterns of resource use that increase human contact with potential zoonotic hosts (Saksena et al., 2022).

Along with land transformation, climatic modification adds further complexity to zoonotic risk. Urban heat islands, altered precipitation regimes, and increased climatic variability affect pathogen survival, vector ecology, and host susceptibility (Mora et al., 2017). Increased surface temperatures can prolong the survival of certain pathogens in the environment, while stress in wildlife populations can weaken immune function and increase pathogen shedding (Wilber et al., 2022). At the same time, extreme precipitation events and disrupted hydrological systems can redistribute pathogens across urban landscapes and increase human exposure (Zhang et al., 2024). These climatic factors do not act independently. They interact with land-use change and ecological disruption in ways that increase spillover potential far beyond what would be expected from any single factor.

The worsening state of the atmosphere is another serious and frequently neglected environmental aspect of zoonotic emergence in expanding urban areas. Increased concentrations of particulate and gaseous pollutants are associated with impairment of respiratory and immune functions, thereby increasing host vulnerability to infection (Franklin et al., 2015). At the population level, especially in highly populated urban areas, air pollution facilitates the establishment of infections as more host organisms come in contact with pathogens. The atmosphere can also influence pathogen survival, thereby modifying the potential for disease outbreaks in urban and peri-urban areas (Yang et al., 2024).

Loss of ecosystem diversity, together with habitat fragmentation, is also central to explaining why rapidly urbanizing areas are often identified as hotspots of zoonotic disease emergence. Where rapid habitat fragmentation occurs, the loss of biodiversity, traditionally viewed as a disease-dilution buffer, can shift the balance in favor of generalist host species with greater zoonotic competence (Civitello et al., 2015). Under urban sprawl conditions, such generalist species are often the ecological "winners," which increases host reservoir proximity to human populations (Gibb et al., 2020). These system-wide ecological shifts affect host movement, foraging behavior, and interspecies interactions, and they can alter transmission dynamics in ways that increase spillover risk.

Even with the increase of empirical and theoretical work on the various forms of environmental change, there is still a lack of explanation on the emergence of zoonotic diseases. Most of the studies that exist are retrospective correlation studies or studies on the effects of a single driver. While these studies are good, they still do not capture the overly complex and nonlinear interdependence of the processes causing zoonotic diseases to appear in urbanising areas (Plowright et al., 2017). More importantly, there remains a lack of integrated frameworks that simultaneously evaluate land-use change, climate stress, air quality, and ecological disruption within a single spatially explicit assessment of spillover risk. As a result, policy and planning interventions often lack the systems-level evidence needed to identify, compare, and prioritize high-risk locations before spillover occurs.

These limitations can be reduced through advances in geospatial analytics, environmental simulation, and computational modeling. Rather than relying only on descriptive mapping, integrated environmental risk surfaces generated from high-resolution satellite imagery, reanalysis climate datasets, and ecological modeling tools can provide a more mechanistic understanding of how multiple environmental stressors combine across space (Hasan et al., 2016). When coupled with machine-learning-assisted attribution techniques, such approaches can separate the relative and interacting contributions of urban growth, climatic stress, atmospheric degradation, and ecological fragmentation, while also revealing how different urbanization pathways shape future spillover patterns (Becker et al., 2019). These modeling frameworks are especially valuable in rapidly changing regions where conventional surveillance systems are often unable to keep pace with environmental transformation.

This study is positioned within this emerging analytical direction and seeks to quantify the environmental drivers of zoonotic disease emergence in the context of rapid urbanization. It is not intended as a narrative synthesis alone; rather, it develops an integrated simulation-based framework to examine spillover risk as the outcome of interacting urban, climatic, atmospheric, and ecological processes. The central argument of the study is that zoonotic emergence should be understood not as the product of a single environmental disturbance, but as a spatially structured systems response produced by the combined effects of multiple stressors. On this basis, the study aims to generate findings that support clearer environmental risk assessment and more targeted planning recommendations for rapidly urbanizing regions.

The growing recognition of the connections among urban planning, environmental governance, and public health further motivates this research. If managed properly, environmental factors associated with land-use zoning, infrastructure placement, pollution control, and ecosystem conservation may help reduce zoonotic risk (Brooks & Boeger, 2019). However, limited evidence on the spatial extent and interaction of these unmanaged factors continues to encourage reactive planning. To integrate disease control more effectively into sustainable urban ecosystem planning, the environmental dynamics of spillover must be understood more clearly.

The emergence of zoonotic diseases in fast-growing cities is a complex phenomenon involving multiple interacting environmental processes rather than a single isolated event. There is growing evidence linking urban growth, climate stress, air pollution, and biodiversity loss, but the interactions among these processes remain insufficiently understood. This study aims to address that gap by applying a fully integrated simulation-based methodology to identify and spatially allocate the environmental drivers of zoonotic disease emergence. In doing so, it seeks to improve both scientific understanding and environmental risk assessment in the context of rapid urbanization worldwide.

Methodology

The methodological framework of this study relies entirely on an integrated software-driven modeling system designed to generate spatially explicit outputs for zoonotic spillover risk. The approach does not rely on retrospective statistical fitting or case-count regression. Instead, spillover risk is treated as an emergent outcome of interacting environmental subsystems operating within heterogeneous urban landscapes. All framework components are computationally coupled, numerically resolved, and executed within a unified modeling environment to maintain consistency, reproducibility, and spatial comparability across simulation stages.

The integrated geospatial–environmental workflow used to simulate zoonotic spillover risk is shown in figure 1. The workflow includes data ingestion, preprocessing, spatial harmonization, solver coupling, and risk-surface generation. Although the workflow is modular in design, it is executed in a synchronized manner such that the spatial and temporal dynamics of land use, climatic stress fields, ecological agents, and human exposure are updated coherently at each simulation step. This architecture is intended to capture nonlinear emergent responses caused by changing environmental conditions during urbanization.

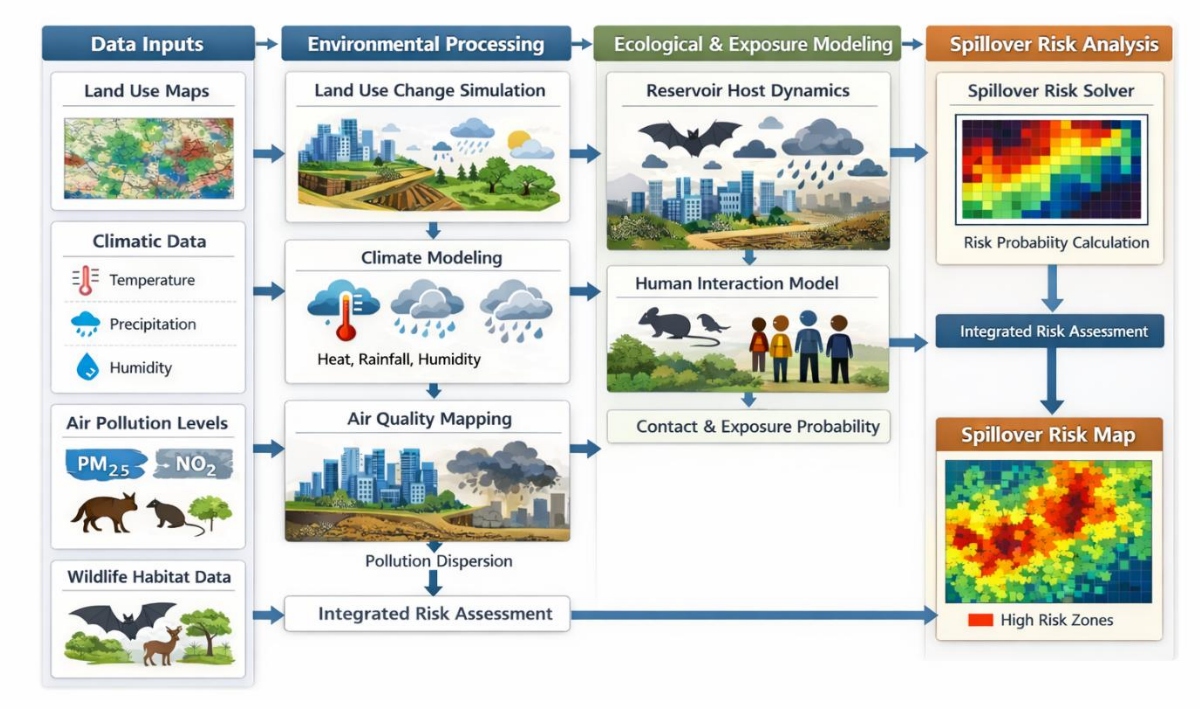

Figure 1: Integrated Geospatial–Environmental Modeling Workflow for Zoonotic Spillover Simulation

The overall spatial domain is represented using a grid-based geospatial framework, which serves as the common computational substrate for all simulations. Core land-use layers are derived from temporally segmented satellite classifications and mapped into major functional classes including urban core, peri-urban transition, agricultural mosaic, and residual natural habitat. All layers are resampled to a common resolution and transformed into a single coordinate reference system to enable pixel-level integration with climatic, atmospheric, and ecological variables. Spatial adjacency matrices are then constructed to represent neighborhood processes such as host movement, habitat-edge dynamics, and local diffusion of environmental stressors. If the study domain contains Nspatial cells, the adjacency matrix is defined as

$$A_{ij} = \begin{cases} 1, & \text{if cell } i \text{ shares a neighborhood boundary with cell } j \\ 0, & \text{otherwise} \end{cases}$$

where A_{ij} defines spatial connectivity between cells i and j. This matrix is used in movement updating, habitat continuity estimation, and local interaction propagation.

Temporal alignment is imposed across all environmental inputs so that land-use transitions, ecological shifts, and climatic variability are modeled within a common time window. Rather than treating the environmental layers as static, the framework applies sequential updating in which urban sprawl and habitat fragmentation at time t influence ecological and climatic response fields at time $t + 1$. This temporal coupling is important because spillover potential in urban systems is often shaped by cumulative degradation rather than by a single abrupt transition.

To ensure comparability among heterogeneous environmental variables, all continuous model inputs are normalized to dimensionless values in the interval $[0,1]$. For any environmental driver X_i at grid cell i, normalized intensity is computed as

$$X_i^* = \frac{X_i - X_{\min}}{X_{\max} - X_{\min}}$$

where $X_{\min}$ and $X_{\max}$ are the minimum and maximum values of that driver across the simulation domain. This normalization is applied to urban expansion intensity, fragmentation level, temperature anomaly, precipitation variability, humidity anomaly, particulate burden, habitat degradation and human exposure intensity. The normalization step prevents any one driver from dominating the solver only because of scale differences in the raw data.

Climate stress fields are incorporated as major spatiotemporal modulators of pathogen persistence, host stress, and environmental compatibility. Temperature anomaly (T_i^*), precipitation anomaly (P_i^*) and humidity anomaly (H_i^*) are represented as normalized fields relative to baseline climatology. A composite climatic stress index is then defined for each cell $i$as

$$C_i = \alpha_T T_i^* + \alpha_P P_i^* + \alpha_H H_i^*$$

where α_T, α_P and α_H are weighting coefficients satisfying

$$\alpha_T + \alpha_P + \alpha_H = 1$$

These terms represent the relative contribution of thermal anomaly, precipitation variability, and humidity anomaly to climate-related spillover amplification. This structure allows the solver to capture threshold-like responses such as heat-driven ecological stress or precipitation-mediated redistribution of environmental pathogen load.

Atmospheric stress is represented through pollutant concentration surfaces generated from $PM_{2.5}$ and NO_2 proxy layers associated with urbanization and industrialization. A normalized pollution burden Q_i is computed as

$$Q_i = \beta_1 PM_{2.5,i}^* + \beta_2 NO_{2,i}^*$$

where

$$\beta_1 + \beta_2 = 1$$

This layer is used to modify host susceptibility and environmental stress intensity. In the model, atmospheric burden is not treated as an isolated driver of spillover but as an amplifying stressor that increases effective vulnerability under fragmented and thermally stressed conditions.

The ecological subsystem is implemented through an agent-based reservoir host module embedded within the geospatial grid. Host initialization is governed by a habitat suitability index S_i, defined as

$$S_i = \gamma_1 V_i^* + \gamma_2 K_i^* + \gamma_3 L_i^* - \gamma_4 F_i^*$$

where V_i^* is normalized vegetation cover, K_i^* is habitat connectivity, L_i^* is local resource availability, and F_i^* is fragmentation intensity. The coefficients γ_1 to $\gamma_4$describe the ecological contribution of each factor. Host movement between neighboring cells is then governed by a transition probability

$$M_{ij} = \frac{A_{ij} S_j}{\sum_{k \in N(i)} A_{ik} S_k}$$

where $N(i)$ denotes the neighborhood of cell i. This formulation allows host redistribution to emerge from habitat structure rather than from arbitrary density functions. As fragmentation increases and habitat islands shrink, agents are forced into narrower corridors and higher-density edge zones, producing aggregated host presence in ecologically disturbed peri-urban regions.

Human interaction is represented through a spatial contact likelihood rather than through direct infection counts. Let U_i denote normalized human population intensity and D_i denote normalized reservoir host density in cell i. The environmental exposure field is defined as

$$E_i = U_i \times D_i \times \phi_i$$

where ϕ_i is an environmental filter term accounting for land use, flooding tendency, heat stress and local accessibility. This exposure field represents the environmental potential for host–human contact and spillover opportunity, without assuming that every contact event leads to infection.

The spillover risk solver integrates the outputs of the land-use, climate, atmosphere, ecology, and exposure modules into a single probabilistic surface. The normalized spillover risk at cell i, denoted Z_i, is computed as

$$Z_i = w_U U_i^* + w_F F_i^* + w_C C_i + w_Q Q_i + w_E E_i + \sum_{m<n} \lambda_{mn} \left(X_{m,i} X_{n,i} \right)$$

where w_U, w_F, w_C, w_Q and w_E are the primary weights assigned to urban expansion, fragmentation, climatic stress, pollution burden, and exposure intensity, respectively. The terms $X_{m,i} X_{n,i}$ represent pairwise interaction effects, and λ_{mn} denotes the corresponding interaction coefficient. The primary weights are constrained such that

$$w_U + w_F + w_C + w_Q + w_E = 1$$

Interaction terms are explicitly included to capture amplified risk under coupled environmental stress. In particular, the framework gives specific emphasis to urban expansion × fragmentation, fragmentation × climatic stress, and climatic stress × atmospheric pollution, because these combinations represent the most plausible amplification pathways under peri-urban environmental degradation.

To maintain risk values within interpretable bounds, the raw risk score is converted into a bounded spillover probability surface using a logistic transformation:

$$P_i = \frac{1}{1 + e^{-Z_i}}$$

where $P_i \in (0,1)$ denotes the final spillover potential at grid cell i. This transformation ensures that the final output remains spatially comparable across regions and scenarios and allows the model to generate normalized risk surfaces rather than unbounded composite scores.

The driver-attribution layer decomposes the final risk surface into the most influential environmental components. Attribution is performed by controlled perturbation of each driver while holding all other drivers constant. For driver d, the contribution score at cell i is defined as

$$A_{d,i} = \frac{| P_i - P_{i,-d} |}{\sum_{j=1}^{D} | P_i - P_{i,-j} |}$$

where $P_{i,-d}$ is the spillover probability recomputed after suppressing or perturbing driver d and D is the total number of drivers considered. In this formulation, $A_{d,i}$ measures the relative sensitivity of local spillover probability to each environmental driver. Dominant attribution maps are then generated by assigning each cell to the driver or interaction term with the highest contribution score. This allows the framework to distinguish whether a hotspot is primarily controlled by urban growth, habitat fragmentation, climatic stress, pollution burden, or a coupled interaction.

Model robustness is evaluated using internal consistency testing, perturbation stability analysis, and spatial pattern validation. First, sensitivity testing is performed by varying the primary weights and interaction coefficients across bounded ranges and measuring hotspot persistence. The stability of the risk surface is quantified using

$$\Delta P = \frac{1}{N} \sum_{i=1}^{N} | P_i^{(a)} - P_i^{(b)} |$$

where $P_i^{(a)}$ and $P_i^{(b)}$ are risk values generated under two neighboring parameter settings. Low ΔP values indicate solver stability under reasonable parameter variation. Second, hotspot consistency is evaluated by checking whether high-risk clusters persist in peri-urban and fragmented edge regions under repeated runs. Third, spatial realism is assessed by testing whether high-risk zones correspond to environmentally plausible transition regions such as fragmented habitat margins, peri-urban fringes, and mixed land-use interfaces. Although the framework is not designed to reproduce exact outbreak timelines, this spatial correspondence provides a defensible form of environmental validation for a fully simulation-based study.

Table 1 summarizes the environmental input datasets used in the framework together with their spatial resolution, temporal coverage, and parameterization ranges. The table supports transparency and reproducibility by documenting the representation scale of each environmental component used in the risk hotspot simulations. In combination with figure 1, table 1 provides a methodological reference for how heterogeneous environmental drivers are operationalized within a unified computational pipeline. This integration of datasets, model architecture, and numerical logic ensures that the resulting spillover risk surfaces are grounded in explicitly defined environmental mechanisms rather than in purely statistical associations.

Table 1: Environmental Input Datasets, Spatial Resolution, Temporal Coverage, And Model Parameterization

Environmental Component	Dataset Type / Source Category	Spatial Resolution	Temporal Coverage	Model Parameterization and Usage
Urban land-use structure	Multi-temporal satellite-derived land-cover rasters	30–100 m grid	2000–2022 (annual)	Classified into urban core, peri-urban, agricultural, and natural habitat classes; used to compute land-use transition probabilities and habitat fragmentation metrics
Urban expansion dynamics	Land-use transition probability surfaces	100 m grid	5-year transition windows	Drives spatial progression of urban growth and interface edge creation influencing host–human contact zones
Climatic stress fields	Gridded temperature, precipitation, and humidity anomaly fields	1 km grid (downscaled)	Monthly, 2000–2022	Modulates pathogen persistence, host physiological stress, and seasonal amplification of spillover risk
Atmospheric pollution burden	Particulate concentration fields ($PM_{2.5}$ and NO_2 proxies)	1 km grid	Annual mean, 2000–2022	Incorporated as host susceptibility modifiers affecting effective spillover probability under chronic exposure
Ecological habitat integrity	Vegetation cover and habitat connectivity rasters	100 m grid	Baseline with dynamic degradation	Used to compute habitat suitability indices governing reservoir host initialization and movement constraints
Reservoir host dynamics	Agent-based ecological population states	Grid-embedded agents	Dynamic simulation steps	Simulates host redistribution under fragmentation and environmental stress, producing spatially explicit contact gradients
Spillover risk integration	Multi-driver probabilistic fusion solver	100 m grid	Simulation end-state	Integrates urbanization, climatic, atmospheric, and ecological drivers into final spatial spillover risk surfaces

Urbanization–Environment Interaction Dynamics

The rapid expansion of cities involves tightly coupled processes that alter environmental structure and change the conditions under which zoonotic diseases may be transmitted from animals to humans. This section examines how urban expansion and environmental disruption interact, treating these mechanisms as the primary drivers of the quantitative risk framework developed later in the paper. Rather than considering urban growth and ecological disruption as independent processes, the analysis focuses on how their interaction creates spatially uneven environments that either amplify or reduce spillover risk depending on the pathway of urbanization.

Urbanization is represented here as a continuous spatial process extending from the urban core toward peri-urban and semi-natural areas. In the simulation of urbanization and environmental transformation shown in figure 2, the black, white, and grey areas represent semi-natural, urban, and peri-urban environments, respectively, while the red areas indicate predicted urban expansion derived from multi-year land-use transition modeling. The simulation generates geospatially explicit outputs at the cell level, where each cell reflects a specific combination of land-use state and fragmentation intensity. Urbanization is not spatially uniform. It follows different pathways shaped by transportation corridors, water systems, and economic activity. As a result, peri-urban zones emerge as areas of enhanced instability where human settlement, remaining vegetation, and wildlife increasingly intersect.

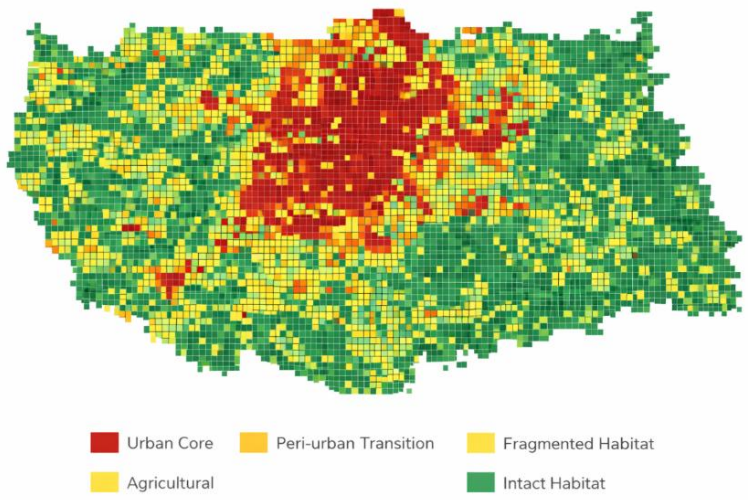

Figure 2: Simulated Urban Expansion and Habitat Fragmentation Field Derived from Multi-Year Land-Use Transition Modeling

When urban land cover replaces natural or semi-natural habitat, fragmentation becomes a major ecological consequence. It is not simply a matter of habitat loss; it also alters patch-size distribution, habitat connectivity, and edge-to-core relationships. The fragmentation field shown in figure 2 illustrates that urban expansion substantially increases edge density, particularly in areas of informal settlement and unregulated land conversion. These fragmented landscapes contain more small and isolated patches and fewer large contiguous habitats, thereby altering ecological movement patterns and resource distribution.

The relationship between urban expansion and fragmentation is nonlinear. During the early stages of urban expansion, fragmentation remains moderate because larger habitat patches are only partially disturbed. However, once a critical threshold of urban growth is reached, fragmentation increases rapidly, connectivity declines sharply, and reservoir hosts are displaced into new movement pathways. The simulation captures this threshold-like behavior by showing that relatively small increases in urban land expansion can produce disproportionately large increases in fragmentation. This finding helps explain why rapidly urbanizing rural and peri-urban areas often experience much stronger ecological disruption than would be expected from land conversion alone.

Fragmented peri-urban zones also differ from both intact ecosystems and dense urban cores in their microclimatic behavior. Within the simulation framework, these zones exhibit greater anthropogenic exposure, stronger thermal variability, and more unstable moisture conditions. Such microenvironmental stress influences both host behavior and pathogen persistence. In this way, fragmentation and microclimatic variability act together to shape spillover conditions, reinforcing the importance of considering environmental stress as an integrated ecological process rather than as a set of isolated disturbances.

Patterns of human activity further intensify these interactions. Mixed land use, high population turnover, and limited infrastructure, all common in peri-urban environments, increase the mobility of human populations within fragmented landscapes. The greater the spatial overlap between human activity and redistributed reservoir hosts, the greater the probability of contact, especially where the advancing urban fringe meets remnant forest and agricultural mosaic areas. By linking land-use change to exposure probability fields, the simulation indicates that contact probability is highest in structurally complex peri-urban zones rather than in the dense urban core.

Table 2 provides indices of urbanization and ecological disruption for the modeled regions and complements the spatial patterns shown in figure 2. These indices quantify major structural components of urban and habitat transformation, including urban expansion rate, fragmentation level, habitat connectivity loss, edge density, reservoir host redistribution, and composite disruption score. Although some regions have lower overall urban land coverage, peri-urban regions consistently show the highest fragmentation and interface intensity. This indicates that, for spillover-relevant environmental disruption, spatial configuration is more important than urban extent alone.

The contrasts in table 2 show that the degree of environmental disruption varies substantially with the urbanization pathway. Regions characterized by compact and planned growth exhibit lower fragmentation, lower interface density, and lower composite disruption scores. By contrast, regions undergoing extensive and unregulated expansion show the highest fragmentation values, the greatest connectivity loss, and the strongest interface density, creating conditions for repeated human–wildlife interaction. These differences indicate that urban form and growth pattern are critical determinants of spillover-relevant ecological change.

Table 2: Quantified Urbanization and Ecological Disruption Indices Across Modeled Regions

Modeled Region	**Urban Expansion Rate ($km^2 \cdot yr^{-1}$)**	**Habitat Fragmentation Index (0–1)**	**Habitat Connectivity Loss (%)**	**Peri-urban Interface Density ($km \cdot km^{-2}$)**	**Reservoir Host Redistribution Index (0–1)**	**Composite Environmental Disruption Score (0–1)**
Region A (Compact growth)	8.6	0.32	18.4	1.7	0.29	0.34
Region B (Planned expansion)	12.1	0.41	26.7	2.3	0.38	0.42
Region C (Mixed urban form)	18.9	0.58	41.2	3.6	0.55	0.61
Region D (Rapid peri-urban sprawl)	26.4	0.71	56.8	4.9	0.69	0.74
Region E (Unregulated expansion)	31.7	0.83	68.5	6.2	0.81	0.86
Region F (Infrastructure-led growth)	22.8	0.65	49.3	4.1	0.62	0.68

The simulation further suggests that ecological disruption participates in feedback loops associated with urban expansion. As habitats become increasingly fragmented, wildlife movement is redirected toward anthropogenic spaces such as urban waste zones, urban agriculture, and drainage corridors. This increases visibility and contact at the urban edge and further modifies boundary conditions in already fragmented landscapes. Such feedback intensifies fragmentation over time and concentrates interaction hotspots in specific spatial locations. Capturing this process is one of the strengths of the integrated simulation framework because it emphasizes the co-evolution of urban and ecological systems rather than treating them separately.

The temporal analysis also shows that the effects of fragmentation can persist even when the pace of urban growth slows. As time progresses, the density of interfaces and the persistence of ecological instability remain strongly influenced by the legacy of prior fragmentation. This temporal persistence helps explain why zoonotic spillover risk may remain high in urban settings where active land conversion has already stabilized. Once critical fragmentation thresholds are crossed, the resulting processes can maintain their own momentum even in the absence of continued rapid expansion.

Figure 2 also shows that high-fragmentation zones are spatially clustered and frequently associated with infrastructure such as highways, railways, and riverbanks. These clusters represent complex convergence zones where multiple fragmentation drivers operate simultaneously. Their importance lies in the fact that spillover-relevant ecological disruption is not evenly distributed, but concentrated in identifiable spatial hotspots. This makes such areas important targets for surveillance, monitoring, and spatial intervention in rapidly urbanizing regions.

Combined, the interaction dynamics demonstrated here show that the risk of zoonoses due to urbanization stems from the coupling of spatial expansion and ecological disruption, and not from either process in isolation. The alteration of the ecological means and the urban expansion means being constructed produces rural spaces where resonant human-reservoir interactions are spatially and environmentally amplified. For table 2, figure 2 provides a spatial and visual summary of these processes, while table 2 quantifies and structures the processes and relations in the mechanisms regionally for a spatial, measurable, and distinct attribution.

Results and Discussion

The integrated simulation framework shows that zoonotic spillover risk is a spatially structured outcome of interacting environmental drivers rather than a simple linear response to urbanization alone. The model-generated risk surfaces identify both the location and relative intensity of spillover amplification by combining urban expansion dynamics, ecological disruption, climatic stress, and atmospheric degradation within a unified solver. The resulting patterns indicate that risk is concentrated in transitional landscapes where multiple stressors overlap, rather than being uniformly distributed across the urban system.

Figure 3 maps the spatial spillover probability surface under combined urban growth and ecological disruption scenarios. The heatmap represents normalized spillover likelihood at the grid-cell level rather than observed case incidence. The most prominent pattern is the concentration of high-risk zones in peri-urban belts, whereas both dense urban cores and intact peripheral habitats remain comparatively lower-risk areas. This indicates that the highest spillover potential occurs in transitional landscapes where fragmented habitats, redistributed reservoir hosts, and human contact fields overlap spatially.

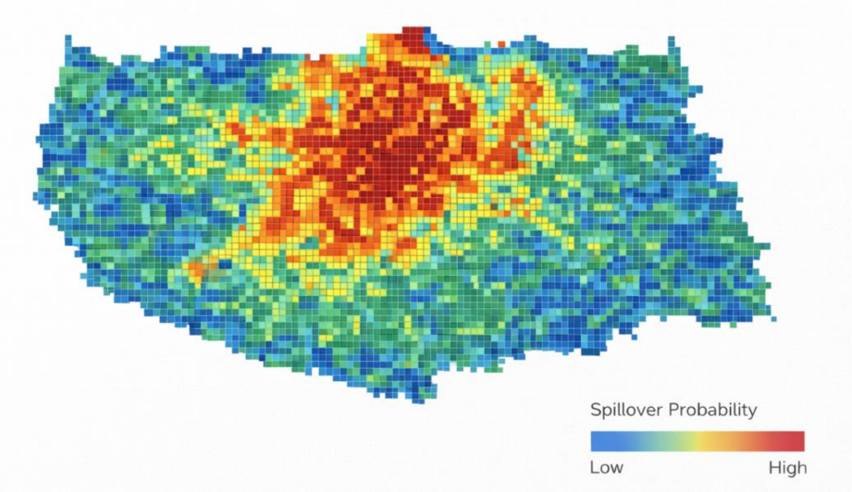

Figure 3: Spatial Spillover Probability Surface Under Combined Urban Growth and Ecological Disruption Scenarios

The spatial gradients further indicate that spillover probability increases sharply beyond a critical urban-fragmentation transition. In practical terms, risk does not rise uniformly with urban land fraction, but accelerates as fragmented land patches and human-modified edges begin to dominate the peri-urban landscape. High-risk cells are also aligned along transport and settlement fronts, suggesting that infrastructure-led expansion contributes to the redistribution of both environmental stress and host contact opportunity. This supports the interpretation that interface instability, rather than urban density alone, is the dominant structural condition associated with zoonotic emergence.

Figure 4 evaluates how ecological disruption interacts with climatic and atmospheric stressors to shape spillover intensity. The coupled response surface shows that spillover intensity increases nonlinearly when temperature anomaly, precipitation variability, and $PM_{2.5}$ burden act together. The highest modeled intensity occurs when warm thermal anomalies coincide with elevated particulate burden and moderate-to-high precipitation variability. Under low-fragmentation conditions, even strong climatic stress produces only limited spillover amplification, whereas under high-fragmentation conditions the same stressors generate much stronger responses

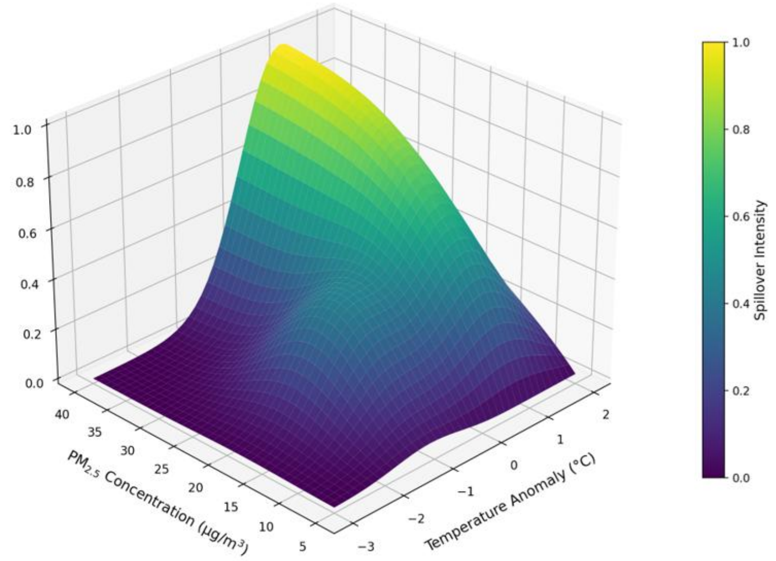

Figure 4: Climatic and Atmospheric Stressor Interaction Surface Influencing Spillover Intensity

The response surface also reveals asymmetry among the environmental drivers. Temperature anomaly has a stronger amplification effect when combined with pollution than when evaluated in isolation, implying that degraded air quality intensifies the ecological and physiological consequences of heat stress. Precipitation variability shows a nonlinear effect: moderate variability increases modeled spillover risk by enhancing environmental mixing and contact opportunity, whereas very high variability reduces local intensity in some cells because extreme hydrological disturbance disrupts host movement and spatial overlap. This helps explain why single-factor analyses often underestimate spillover complexity in rapidly changing urban environments.

Figure 5 presents the spatial attribution of dominant environmental drivers across the modeled urban gradient. Based on the attribution framework applied to the solver outputs, each grid cell is assigned to the driver or interaction term with the greatest control over local spillover probability. The attribution pattern shows a clear spatial transition. In early-stage peri-urban zones, urban expansion and habitat fragmentation dominate the risk signal, whereas in later-stage urban transition zones, climatic and atmospheric stressors become more influential. In relatively undisturbed peripheral areas, intact ecological structure remains the dominant constraining factor, limiting the amplification of spillover risk despite background climatic variability.

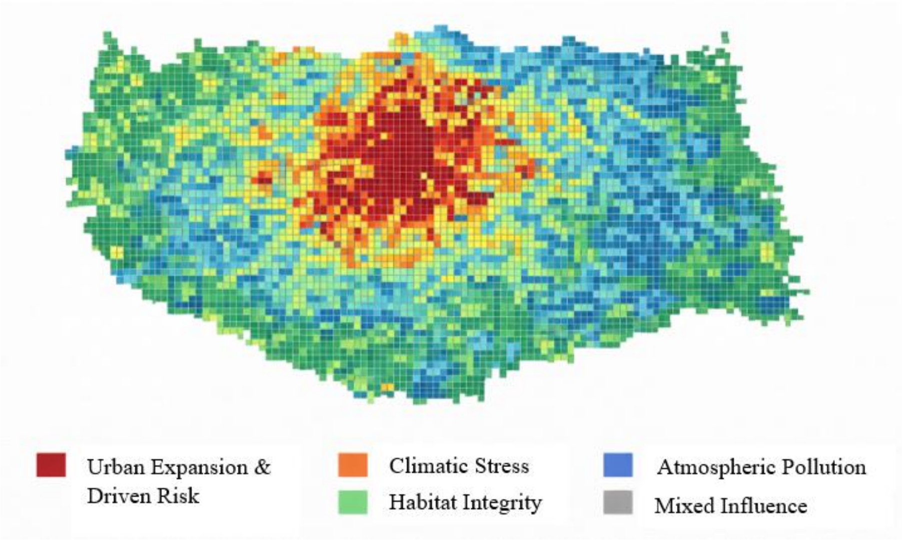

Figure 5: Environmental Driver Attribution Map Showing Dominant Spillover Determinants Across Urban Gradients

This spatial transition indicates that structural land transformation dominates the early formation of spillover hotspots, while secondary stressors become more important as urban landscapes become environmentally degraded and climatically stressed. The attribution map therefore suggests that spillover control strategies should not be spatially uniform. Newly urbanizing areas may require stronger land-use and fragmentation management, whereas mature urban margins may need stronger climatic and environmental quality interventions.

Figure 6 looks at the relationship between probability of spillover and the additional fragmentation along the pathways of urbanization. Instead of the former figures which deals primarily with attribution and spatial distribution, this one looks primarily at system behavior due to breaching fragmentation levels. The figure indicates that spillover probability is relatively flat at a primal level of fragmentation, but then shoots up once fragmentation surpasses some critical middle-range level. The most rapid and substantial levels of fragmentation are seen in peri-urban areas, where additional and minute levels of fragmentation result in a massive increase to the spillover risk.

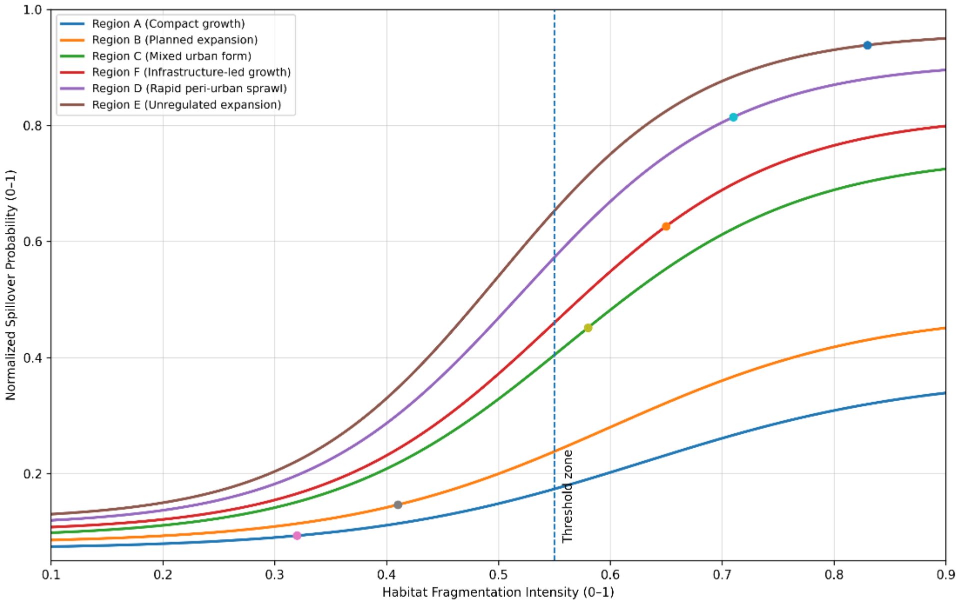

Figure 6: Threshold Response of Spillover Probability to Habitat Fragmentation Intensity Across Modeled Urbanization Pathways

The qualitative significance of figure 6 is that it demonstrates a clear regime shift in environmental risk. Regions of compact growth and planned expansion show risk levels that are smoother and lower and are exhibiting an ecological system that is relatively intact and unchanged in complexity despite the level of urbanization occurring there. On the other hand, areas of mixed growth, along with others like infrastructure led growth, and in particular areas of uncontrolled expansion show great steepness. This indicates that with adequate fragmentation levels, the system is less ecologically fortified. The figure captures the reality that relationship between land use change and spillover is complex. In an even larger context, the peri-urban regions are far more sensitive to habitat modification when the level of fragmentation, contact, and human presence reach a certain level.

Table 3 quantifies the contribution patterns underlying the spatial results shown in figures 3–5. Among the direct effects, habitat fragmentation is the strongest single contributor at 31.1%, followed by urban expansion at 27.4%, climatic stress at 14.6%, and atmospheric pollution at 9.2%. These values show that structural ecological disruption explains a much larger share of modeled spillover potential than pollution alone, with fragmentation contributing more than three times the direct effect of atmospheric burden.

Table 3: Model-Derived Spillover Risk Contributions and Interaction Effects of Environmental Drivers

Environmental Driver / Interaction Term	**Direct Contribution (%)**	**Interaction Contribution (%)**	**Total Explained Contribution (%)**	**Net Influence Score (0–1)**	**Dominant Spatial Context**
Urban expansion intensity	27.4	0.0	27.4	0.62	Urban core to peri-urban transition zones
Habitat fragmentation	31.1	0.0	31.1	0.68	Peri-urban and edge-dominated landscapes
Climatic stress (temperature + precipitation variability)	14.6	0.0	14.6	0.41	Thermally stressed peri-urban regions
Atmospheric pollution burden ($PM_{2.5}$)	9.2	0.0	9.2	0.33	High-density urban and industrial corridors
Urban expansion × habitat fragmentation	0.0	21.7	21.7	0.74	Rapidly expanding peri-urban belts
Fragmentation × climatic stress	0.0	17.9	17.9	0.69	Ecologically degraded, climate-sensitive zones
Climatic stress × atmospheric pollution	0.0	13.5	13.5	0.58	Heat-retaining, polluted urban margins
Multi-driver combined interaction	0.0	28.6	28.6	0.81	High-risk spillover amplification clusters

The interaction effects are equally important. The urban expansion × habitat fragmentation interaction contributes 21.7%, the fragmentation × climatic stress interaction contributes 17.9%, and the climatic stress × atmospheric pollution interaction contributes 13.5%. Most notably, the multi-driver combined interaction accounts for 28.6%, which is larger than the direct contribution of urban expansion alone and close to the direct contribution of habitat fragmentation. This means that the combined effect of interacting stressors is not secondary to the structural landscape variables; it is one of the dominant mechanisms through which high-risk spillover clusters are formed.

The net influence scores provide an additional ranking of environmental importance. The highest score belongs to the multi-driver interaction term at 0.81, followed by urban expansion × habitat fragmentation at 0.74, fragmentation × climatic stress at 0.69, habitat fragmentation at 0.68, and urban expansion alone at 0.62. By contrast, atmospheric pollution alone has the lowest score at 0.33. This ranking reinforces the conclusion that spillover emergence is best interpreted as a systems-level phenomenon. Individual drivers matter, but the strongest modeled effects appear when structural environmental change is reinforced by secondary climatic and atmospheric stress.

Figure 7 provides a regional comparison of direct and interaction-based spillover contributions under different urbanization pathways. The graph shows that compact and planned growth regions are dominated by lower direct-risk components and relatively weak interaction effects. As urbanization becomes less regulated and more spatially dispersed, the interaction-driven share of total spillover potential becomes progressively larger. In particular, the regions characterized by rapid peri-urban sprawl and unregulated expansion show the highest contributions from interaction terms, especially the combined urban expansion–fragmentation and multi-driver amplification components.

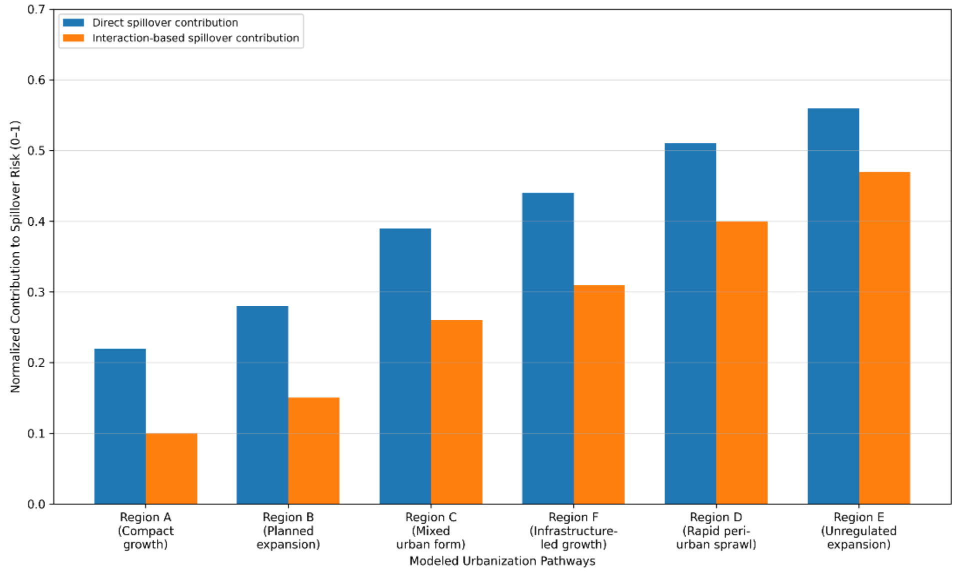

Figure 7: Regional Comparison of Direct and Interaction-Based Spillover Contributions Under Different Urbanization Pathways

The qualitative interpretation of figure 7 is especially important because it reveals that high spillover potential is not only a matter of stronger individual drivers, but also of stronger coupling among drivers. In planned systems, direct effects remain more visible because environmental controls reduce the opportunity for feedback amplification. In disrupted systems, however, interaction terms begin to dominate the risk structure, indicating that once ecological and climatic instability are embedded into the landscape, the overall system becomes much more sensitive to compound stress. This makes the high-risk regions qualitatively different from the low-risk ones. They are not simply more urbanized; they are more environmentally entangled, and this entanglement is what generates persistent spillover-prone conditions.

Taken together, the results expand current understanding of zoonotic emergence in rapidly urbanizing environments. The findings show that urbanization should not be treated as a uniform risk factor. Planned or compact expansion pathways are associated with lower fragmentation and therefore lower modeled spillover potential, even under background climate stress. In contrast, peri-urban sprawl and unregulated expansion generate persistent high-risk zones because they intensify interface density, ecological disruption, and multi-driver amplification. The practical implication is that the form of urban growth matters as much as the rate of urban growth.

A further implication is the temporal persistence of environmentally amplified spillover risk. Once fragmentation-driven hotspots are established, secondary drivers such as climate variability and pollution can continue to sustain elevated risk even when the pace of urban growth slows. This helps explain why spillover-prone landscapes may remain hazardous long after the initial land conversion phase. In this sense, the risk surface represents not only current environmental instability, but also latent spillover potential embedded in the spatial structure of the landscape.

The integrated framework also strengthens the practical relevance of the model. By distinguishing where particular drivers dominate, it provides a basis for spatially targeted intervention. Areas dominated by fragmentation may benefit most from habitat connectivity restoration and land-use control, whereas thermally stressed and polluted urban margins may require stronger environmental quality management. The results therefore contribute both scientific interpretation and planning value by showing not only where spillover risk is elevated, but also which environmental mechanisms are most responsible for that elevation.

Conclusion

The emergence of zoonotic diseases in rapidly urbanizing areas is not a diffuse or random process. Rather, it is shaped by distinct spatial patterns produced by interacting environmental processes. The results of this study show that, within a unified simulation framework, the combined effects of urban expansion, ecological disruption, climatic stress, and atmospheric degradation concentrate spillover risk in transitional landscapes rather than in dense urban cores or intact ecosystems. In particular, peri-urban zones characterized by fragmented habitats and dynamic human–environment interactions act as persistent amplification belts for zoonotic spillover.

The findings further indicate that spillover risk during urbanization is influenced more by the configuration and mode of urban growth than by the extent of urban growth alone. Compact and controlled expansion pathways are associated with lower ecological disruption and therefore lower spillover potential, even under climatic stress. In contrast, uncontrolled peri-urban sprawl produces sustained habitat fragmentation, ecological instability, and prolonged spillover risk. The threshold-based results further show that spillover probability increases nonlinearly once fragmentation crosses a critical mid-range level, indicating that ecological disruption can produce disproportionate increases in zoonotic risk rather than gradual change alone. These results emphasize the importance of land-use regulation and spatial planning in reducing the environmental conditions that favor emerging infectious diseases.

Climatic and atmospheric stressors function primarily as amplifying factors rather than independent drivers of spillover risk. Their influence becomes strongest when they are superimposed on already degraded ecosystems, where fragmented habitats and altered host movement patterns increase the sensitivity of the landscape to additional stress. The regional comparison of modeled pathways also shows that interaction-driven amplification remains relatively limited under compact and planned growth, but becomes much stronger in rapidly sprawling and unregulated urban forms. This helps explain why urban and peri-urban ecosystems undergoing both environmental degradation and climatic stress may show progressively higher spillover potential over time.

The study also demonstrates the value of spatially explicit, simulation-based modeling frameworks for zoonotic risk assessment. By integrating mechanistic environmental components with attribution analysis, threshold behavior, and regional pathway comparison, the framework moves beyond simple correlation and helps identify the dominant drivers and interaction effects across different urban gradients. In this way, the study provides a useful basis for anticipatory risk mapping and for adaptive management strategies that link public health, urban planning, and environmental governance.

In conclusion, zoonotic spillover in rapidly urbanizing regions should be understood as the outcome of interacting processes involving urban expansion, ecological disruption, and environmental stress. The results indicate that interaction effects are central to the formation of high-risk spillover zones and should therefore be considered in future risk assessment and management efforts. Reducing future zoonotic emergence will require coordinated strategies that address land-use structure, habitat integrity, and environmental quality together within urban and regional planning systems.

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