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Review

From patterns to predictions: a framework for the spatial epidemiology of wildlife diseases

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Wildlife diseases pose a significant threat to biodiversity conservation, public health, and livestock. Spatial epidemiology provides a robust framework to understand disease dynamics and guide policy and management. However, the rapid proliferation of spatial methods has largely occurred in isolation across disciplines, leaving the field without a unified framework that connects these approaches and clarifies their strengths, limitations, and applications. The spatial epidemiology workflow includes three main steps: 1) descriptive analysis of spatial patterns; 2) exploration of observed patterns through explicative analyses; and 3) prediction of pathogen distribution and spread. Descriptive analysis, such as disease mapping and clustering analysis, identifies spatial patterns and informs hypotheses about potential risk factors. Subsequently, explicative analyses can link pathogen presence to ecological or anthropogenic drivers and explain spatiotemporal dynamics. Drivers of pathogen transmission can be further understood through mechanistic models, ranging from population-level Susceptible-Infected-Removed (SIR) frameworks to individual- or group-based SIR approaches such as metapopulation and network models that can incorporate host behavior and social structure. Molecular epidemiology, through genetic variant identification and phylogenetic mapping, can identify outbreak origins and host linkages. Lastly, predictions of pathogen distribution and spread are enabled by ecological niche models and species distribution models, which project potential pathogen distributions based on ecological, geographical, and/or anthropogenic factors, and by mechanistic simulations that parameterize pathogen spread processes to predict expansion over space and time. System-based simulations focus on population-level dynamics, while agent-based simulations incorporate individual-level dynamics, offering detailed insights into pathogen spread and control measures. Consequently, the integration of descriptive analyses, explicative procedures, and predictive models provides a robust framework for addressing the challenges posed by wildlife diseases and developing management and control measures.



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Introduction

Wildlife diseases, understood as pathogens present in wildlife (World Organisation for Animal Health 2015), have an impact on human and livestock health, leading to public health issues and substantial economic losses worldwide (Wolfe et al. 2007, Siembieda et al. 2011, Knight-Jones and Rushton 2013). Moreover, pathogens can threaten the conservation efforts of endangered species, as outbreaks can devastate already vulnerable populations (Daszak et al. 2000, Pedersen et al. 2007).

Epidemiological research has historically focused on humans and livestock, due to their direct impact on the economy and public health, resulting in a relative neglect of the role of wildlife in disease dynamics (Simpson 2002, Gortázar et al. 2007). Furthermore, practical challenges such as sample collection, data gathering on populations and individuals, and the characterization of animal behavior and social dynamics, are inherent to working with wild animals (Mörner et al. 2002, Stallknecht 2007). More recently, the One Health perspective recognized the role that the environment and wildlife populations play in emerging infectious diseases and encouraged their study (Coker et al. 2011). In this context, the interdisciplinary approach emerged as a key tool for addressing many of these challenges, understanding pathogens in relation to their hosts and the environment, under the perspective of the 'disease ecology' (Tompkins and Wilson 1998). The application of this concept has expanded in recent decades, with the ecological perspective of disease becoming increasingly common in wildlife management research (Joseph et al. 2013).

Spatial epidemiology offers a crucial framework to gain deeper insights into the dynamics of pathogen transmission among wildlife populations and at the wildlife–livestock–human interface, by elucidating spatial patterns of disease transmission, identifying high-risk areas and species, and informing targeted interventions across diverse ecosystems (Stevens and Pfeiffer 2011, Gortázar et al. 2014). The growing complexity of infectious disease systems research has motivated major methodological syntheses. These include reviews of mathematical models and their applicability across spatial, temporal, and organizational scales (Heesterbeek et al. 2015), multi-scale frameworks linking within-host dynamics to population-level transmission (Garabed et al. 2019, Doran et al. 2023), and ecosystem-level models embedding pathogens within ecological interaction networks to forecast emergence risk (Hassell et al. 2021). Despite these advances, their translation into wildlife spatial epidemiology has remained fragmented, as new monitoring and analytical tools have often developed in parallel across disciplines rather than within an integrated analytical framework. This fragmentation has left the field without a unifying synthesis that connects the available approaches and clarifies their

strengths, limitations, and potential applications for studying spatial dynamics of pathogens in wildlife. To address this gap, this review presents a comprehensive overview of spatial analysis in wildlife epidemiology through a workflow structured around three key steps: 1) identifying and describing spatial patterns, 2) exploring these patterns and identifying the factors that explain them, and 3) predicting the spatio-temporal behavior of these patterns (Bailey and Gatrell 1995, Pfeiffer et al. 2008, Pfeiffer and Stevens 2015). By tracing the methodological evolution of the field across these steps, from early descriptive analyses to state-of-the-art methodologies for understanding and predicting spatial pathogen dynamics (Fig. 1), this review examines the conceptual foundations, strengths, limitations, and practical applications of these approaches (Table 1), while highlighting priorities for future methodological development. Rather than providing an exhaustive catalogue of all available approaches, this work aims to provide a primer on key methods that can help researchers navigate the methodological landscape of wildlife spatial epidemiology.

Descriptive epidemiology

The investigation of wildlife diseases gained momentum in the 1950s and 1960s, with surveys of wild individuals aimed at detecting pathogens potentially threatening public health or posing a risk of transmission to domestic animals (Keymer 1958, Mair 1973). Some studies included descriptive maps indicating the geographic locations of samples and/or positive cases (Anderson and Beaudoin 1966, Beauregard 1969).

Initially, most studies only indicated the detected pathogens (occurrences) or their prevalence (Andrews 1969, Hedger 1972), without evaluating whether spatial patterns existed. However, pathogen occurrences naturally tend to be spatially aggregated (Wartenberg 2001, Albery et al. 2022). Cluster analysis is a type of descriptive analysis originally developed in anthropology (Kroeber and Driver 1932, Blashfield and Aldenderfer 1988) and later applied to epidemiology after its formalization as a quantitative method by Sokal and Sneath (1963). This approach can identify spatial patterns by comparing the observed spatial distribution of pathogen occurrences to that expected under a null hypothesis, typically random (Ward and Carpenter 2000). The identification of spatial clusters of pathogen occurrences has enabled the implementation of focused efforts in disease monitoring, surveillance, and management (Xu et al. 2012). Spatial cluster analysis can serve as a preliminary step in identifying spatial patterns in pathogen distribution, such as potential outbreak foci (Iglesias et al. 2010).

Although limited in their output, these descriptive spatial analyses are crucial for detecting epidemiological emergencies. Moreover, these studies represent the first step in the

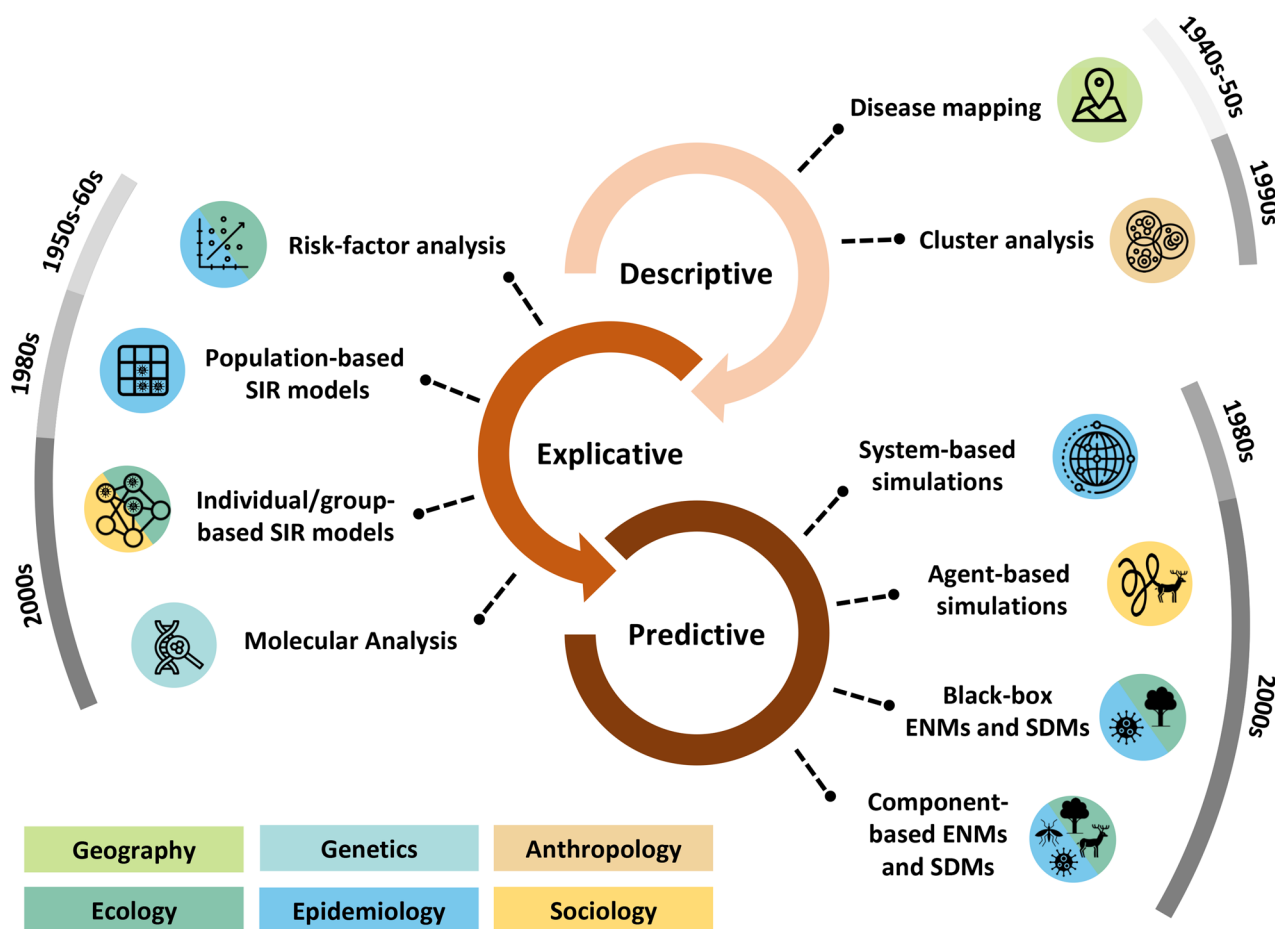


Figure 1. Historical framework for the interdisciplinary development of methodologies used in the spatial epidemiology of wildlife diseases. These methodologies are classified according to their purpose: 1) to describe spatial patterns, 2) to explain these patterns, and 3) to predict the evolution of these patterns. The green, blue, and yellow colors indicate different disciplines that have contributed to the development of methodologies for the study of wildlife diseases from a spatial perspective, whereas the outer gray indicates the decades in which these methodologies began to be implemented.

formulation of directional hypotheses regarding potential risk factors driving the spatial dynamics of pathogens (Bailey and Gatrell 1995). However, a fundamental but often implicit assumption underlying descriptive analyses – and indeed the broader spatial epidemiology framework – is that occurrence data are sufficiently complete and unbiased. In wildlife disease research, however, surveillance is frequently opportunistic and spatially heterogeneous, meaning that detected patterns may reflect variation in sampling effort or reporting capacity rather than true underlying disease risk (Stallknecht 2007, Boadella et al. 2011, Gilbert et al. 2013). This issue is well recognized in the broader infectious disease literature, where studies have shown that apparent hotspots of disease emergence correlate with proxies of surveillance intensity such as gross domestic product or publication effort (Jones et al. 2008, Allen et al. 2017, Stephens et al. 2021). Therefore, hypotheses derived from descriptive analyses should be subsequently tested using more refined statistical frameworks, such as risk factor analysis. These approaches allow spatial patterns to be linked to drivers and can explicitly incorporate

covariates that account for spatial bias in detection probability (Bailey et al. 2005, Artois et al. 2009, Hofstetter et al. 2014).

Explicative analysis

Risk factor analysis

In epidemiology, risk factor analysis has been used to identify factors associated with an increased risk of infection or disease at both the individual and population levels (Giroux 2011). Early on, it was suggested that factors such as habitat, climate, host abundance, and anthropogenic disturbance could influence the epidemiology of wildlife pathogens (Elton 1931). However, most studies addressing factors influencing pathogen occurrence in wildlife focused on host traits, such as species, age, or sex (Johnston and Beauregard 1969, Hensley 1976). Environmental factors, long used in ecology to explain the biodiversity's distribution (Guisan and Zimmermann 2000), were later incorporated to explain

Table 1. Main analytical approaches employed in the study of pathogens in wildlife from a spatial and/or spatiotemporal perspective. ENM, ecological niche model; SDM, species distribution model; SIR, Susceptible-Infected-Removed.

	Approach	Input info	Output info	Potential	Strengths	Weaknesses	Key references
Descriptive	Disease mapping and clustering analysis	Pathogen occurrence	Spatial representation of cases or infection rates and its aggregation	Detection of epidemiological emergencies. Identification of areas for targeted surveillance and control	Hypotheses-generating. No individual-level data required	Cannot identify drivers of observed patterns	Ward and Carpenter (2000), Pfeiffer et al. (2008), Tango (2010)
Explicative	Risk factor analysis	Pathogen occurrence. Risk factors (e.g. environmental conditions, management, host presence, etc.)	Factors associated with pathogen risk	Identification of factors on which efforts to control the pathogen can be focused	Applicable at any scale. No individual-level data required	Limited ability to assess spatial variability in covariate effects. Correlation does not imply causation	Lawson et al. (2003), Pfeiffer et al. (2008), Bender (2009), Blangiardo and Cameletti (2015)
	Path analysis and structural equation modeling (SEM)	Pathogen occurrence. Risk factors structured into hypothesized causal chains.	Direct and indirect effects of risk factors on pathogen dynamics.	Disentangling direct from indirect drivers. Testing mechanistic hypotheses about ecological causal chains.	Moves beyond correlation toward causal inference. Handles complex multi-variable systems.	Requires a priori specification of causal structure and larger sample sizes than standard regression.	Grace (2006), Eby et al. (2023), Sundaram et al. (2024)
	Population-based SIR models – diffusion and lattice-based models	Pathogen occurrence	Basic reproduction rate (R_0). Pathogen spread velocities	Quantification of transmission and spread parameters. Assessment of parameter influence on pathogen dynamics	No individual-level data required. Low computational requirements	Neglects individual variation and social complexity. Limited mechanistic inference	Heesterbeek and Roberts (1995), Riley (2007), Keeling and Rohani (2008), Maki and Hirose (2013)
	Individual/group-based SIR models – metapopulation and network analysis	Node interaction information (GPS locations, camera trap records, proximity logger contacts, etc.)	Interaction patterns between nodes	Identification of super-shedders or super-exposed individuals, groups, or species. Identification of potential transmission hotspots and corridors. Assessment of behavioral and social drivers of transmission	Provides detailed information on interaction dynamics. Accounts for individual and social heterogeneity	Requires individual- or group-level data. Interaction does not imply transmission. Vector-borne diseases are beyond scope. High computational requirements	Craft (2015), Farine and Whitehead (2015), Silk et al. (2017a, 2017b)
	Molecular analysis	Pathogen occurrence with genetic/genomic data	Pathogen phylogeography. Transmission trees	Identification of reservoirs, outbreak origins, and spread pathways and velocities, even for endemic pathogens. Identification of the direction of transmission. Linkage of landscape structure and genetic flow	Direct evidence of transmission events at the individual level	Technically and economically demanding. Integration with ecological models remains complex	Blanchong et al. (2016), Fountain-Jones et al. (2018), Dellicour et al. (2021)

Table 1. Continued

	Approach	Input info	Output info	Potential	Strengths	Weaknesses	Key references
Predictive	Black-box ENMs and SDMs	Pathogen occurrence. Explanatory factors (e.g. environmental conditions, host presence, etc.)	Potential or realized pathogen niche	Identification of risk areas for pathogen spread. Identification of areas where the pathogen could be present but remains undetected	Works with passive surveillance. No local-scale information required	Neglects biotic interactions. Assumes pathogen occurrence captures pathogen–host–vector dynamics	Peterson (2014), Lawson (2018), Johnson et al. (2019), Escobar (2020)
	Component-based ENMs and SDMs	Components occurrence (pathogen, host, vectors, etc.) Explanatory factors (e.g. environmental conditions, host presence, etc.)	Potential or realized pathogen, host, and vector niches	Identification of risk areas for pathogen spread. Identification of areas where the pathogen could be present but remains undetected	Works with passive surveillance. No local-scale information required. Accounts for pathogen–host–vector interactions	Neglecting an involved component may lead to an underestimation of the risk. Biotic interactions are difficult to implement mechanistically	Peterson (2014), Lawson (2018), Johnson et al. (2019), Escobar (2020)
	Joint species distribution models (JSDMs)	Components occurrence (pathogen, host, vectors, etc.) Explanatory factors (e.g. environmental conditions, host presence, etc.)	Co-occurrence patterns and inter-component correlations	Identification of risk areas for pathogen spread. Identification of areas where the pathogen could be present but remains undetected. Identification of biotic interactions driving co-occurrence	Leverages data from co-occurring species (improved predictions for data-poor pathogens). Accounts for shared environmental responses	Biotic interactions inferred statistically, not mechanistically. Computationally demanding with many components	Pollock et al. (2014), Mowry et al. (2025)
	System-based simulations (top-down approach)	System-level parameterization of pathogen spread and host ecology	Spatiotemporal simulation of pathogen spread under current or novel conditions	Identification of risk areas and expected pathogen spread timelines. Assessment of parameter influence on pathogen dynamics. Testing control measures	Parameterized from observed patterns (unaccounted factors are acceptable if their effect is captured by observed dynamics). Low data requirements relative to ABMs	Poor transferability to novel conditions. Does not resolve underlying transmission processes	Lloyd-Smith et al. (2005), Hartig et al. (2011), McCallum (2016)
	Agent-based simulations (ABMs; bottom-up approach)	Agent-level parameterization of host traits, transmission processes, and interactions (host–host, host–pathogen, host–environment and pathogen–environment)	Spatiotemporal simulation of pathogen spread under current or novel conditions with agent-level resolution	Identification of risk areas and expected pathogen spread timelines. Assessment of individual-level parameter influence on pathogen dynamics. Testing control measures	Applicable under novel conditions. Enables disentangling how individual-level parameters drive population-level patterns. Can incorporate both individual-level (bottom-up) and system-level (top-down) processes simultaneously	Computationally demanding. Requires highly detailed parameterization. Neglecting factors or processes influencing pathogen spread can bias predictions	Grimm et al. (2005), Railsback and Grimm (2011), Lane-deGraaf et al. (2013)

wildlife pathogen distribution patterns (Anderson and Beaudoine 1966, Pence and Windberg 1984). Their incorporation introduced a spatial dimension to risk factor analysis, as the geographic location of samples became explicitly linked to environmental conditions used to explain pathogen presence. This type of analysis has allowed for large-scale associations between pathogen emergence and diversity of wild hosts (Keesing et al. 2006, Jones et al. 2008, Johnson and Thielges 2010; but see Schmidt and Ostfeld 2001 regarding the dilution effect debate) and environmental conditions such as pollution and land use (Rohr et al. 2008), introducing the concept of landscape health and suggesting the development of environmental policies for disease management (Patz et al. 2004). At finer scales, risk factor analyses have also identified how host traits such as age, sex, or body condition, and local habitat characteristics, influence individual and population-level infection risk (Lovera et al. 2017; but see Albery et al. 2022), informing targeted surveillance and management at the local level.

The analytical approaches used to implement the spatial aspect in risk factors analysis in wildlife disease research have undergone a relevant evolution. Early studies relied on a-spatial statistical models, mainly GLMs (primarily logistic or Poisson regressions), which assume independence among observations (Cappelle et al. 2010, Recuenco et al. 2012). However, these models often violate the assumption of independence due to spatial autocorrelation in wildlife disease data (Albery et al. 2022). This led to the implementation of spatial regression models (e.g. SAR, CAR, and spatial error/lag structures) and geostatistical approaches that explicitly model spatial dependence (Lichstein et al. 2002, Wall 2004). More recently, Bayesian hierarchical models – including the Besag–York–Mollié family (BYM and BYM2) and stochastic partial differential equations (SPDE) based spatial and spatiotemporal models – have become central tools in wildlife spatial epidemiology, enabling partial pooling, uncertainty propagation, and flexible spatial random effects (Staubach et al. 2002, Osnas et al. 2009, Herraiz et al. 2023). In parallel, the increasing availability of remote-sensing data and high-resolution environmental covariates (Dlamini et al. 2019) has enabled the use of machine-learning (ML) and artificial intelligence (AI) approaches, such as random forests, boosted regression trees, GeoAI, and deep learning architectures, that capture non-linear associations and interactions between risk factors at multiple spatial scales (Fountain-Jones et al. 2019, Janowicz et al. 2019, Mandujano Reyes et al. 2025).

Limitations and future directions

Despite the methodological advances in incorporating the spatial aspect in risk factor analysis, several limitations, inherent to wildlife disease research, remain. As noted for descriptive analyses, surveillance data in wildlife are frequently opportunistic and spatially heterogeneous, which can obscure true associations between covariates and pathogen occurrence (Boadella et al. 2011). Risk factor analysis allows incorporating proxies of surveillance effort, such as sampling intensity or reporting capacity, as covariates, partially accounting for

spatial variation in detection probability (Jones et al. 2008, Allen et al. 2017, Stephens et al. 2021). Hierarchical Bayesian frameworks further help mitigate these issues by incorporating highly explanatory latent spatial random effects, which can absorb spatially structured noise, thereby improving the estimation of fixed effects (Blangiardo and Cameletti 2015).

Second, the association between risk factors and pathogen dynamics may vary spatially, even for the same pathogen and host species (Ryser-Degiorgis 2013). While Bayesian inference has enhanced the incorporation of spatial patterns into risk factor analysis models, the explicit assessment of spatial variability in covariate effects remains uncommon (Lawson 2018). The implementation of spatially varying coefficients also allows assessment of how risk factors interact with disease presence across space (Mandujano Reyes et al. 2024). Variation partitioning procedures quantify the proportion of variability explained by each factor (Legendre 2007), and their spatial representation could help identify the main factors associated with the pathogen dynamics in each region (Herraiz et al. 2023).

At the same time, ML and AI methods are increasingly used to capture complex, non-linear spatial patterns, even under strong sampling heterogeneity (Fountain-Jones et al. 2019, Peters et al. 2020). However, these methods identify spatial patterns without characterizing their spatial structure, limiting their interpretability and constraining mechanistic inference. The development of explanatory tools to identify the AI and ML spatial drivers, such as SHAP (SHapley Additive exPlanations) and hybrid strategies that integrate ML or AI derived spatial features into hierarchical frameworks, represent promising tools for spatial pattern recognition with biologically meaningful parameterization (Farooq et al. 2022, Li 2022, Temenos et al. 2022). For instance, Fountain-Jones et al. (2019) used SHAP to identify non-linear patterns and complex interactions, such as higher exposure of lions *Panthera leo* to canine distemper virus at a young age, but only in low-rainfall years. Similarly, Sundaram and Stephens (2025) used this approach to identify spatial variation in the impact of the factors predicting Orthoebolavirus spillover.

Finally, it is also crucial to bear in mind that correlation does not necessarily imply causation, and a given predictor may act as a proxy for multiple underlying processes. For example, rainfall has been associated with both increased and decreased risk of bovine tuberculosis, as moisture can favor the presence of the pathogen, while water scarcity can increase contacts between hosts (Broughan et al. 2016). Consequently, to infer cause–effect relationships, it is essential to have a clear understanding of the underlying processes. Path analysis and structural equation modeling (SEM) offer a promising framework to address this limitation, by explicitly modeling hypothesized causal pathways among variables and disentangling direct from indirect effects (Grace 2006). This is particularly valuable when drivers operate through causal chains of ecological processes. For instance, SEM revealed that climate oscillation drove food scarcity, which in turn increased aggregation of flying foxes *Pteropus* spp. feeding in agricultural areas, and thus Hendra virus spillover to horses

(Eby et al. 2023). Similarly, path analysis showed that fig tree habitat suitability and cercopithecoid primate diversity were the proximate factors most directly influencing Ebola-virus outbreak risk in humans, while frugivorous bats were more related to the virus's circulation in wildlife (Sundaram et al. 2024).

Models of transmission dynamics

Dynamic models in epidemiology emerged in the early 20th century with the goal of deepening the understanding of the dynamics of pathogen transmission through mathematical parameterization (Ross and Hudson 1917). This led to the development of compartmental models by Kermack and McKendrick (1927), referred to as SIR or SEIR models (Susceptible, Exposed, Infected, and Removed). This approach enables quantification of transmission parameters and identification of key factors driving pathogen spread dynamics (Anderson and May 1991), such as the basic reproduction number (R_0), which is of great utility in understanding the persistence of a pathogen in a host community (Swinton et al. 2002).

Although SIR models were initially formulated as non-spatial frameworks, lacking an association between the location of individuals and pathogen transmission (Almberg et al. 2021), the incorporation of space represented a key methodological advancement. In contrast to risk factor analysis, spatial compartmental models not only reveal associations between pathogen occurrence patterns and potential drivers, but also elucidate the processes through which those drivers influence pathogen spatial dynamics. For example, correlational studies identified an association between supplementary feeding and disease

risk, such as of bovine tuberculosis in white-tailed deer *Odocoileus virginianus* or chronic respiratory disease in house finch (Miller et al. 2003, Moyers et al. 2018), whereas Becker et al. (2018) utilized an SIR model to explore the underlying processes driving this correlation, revealing how resource availability attracts dispersing individuals and thereby increases local aggregation.

The initial methods for incorporating the spatial component into SIR models were diffusion models (Murray et al. 1986), which treat space as a continuum (Maki and Hirose 2013), and lattice-based models (Preston 1973), which discretize space into patches (Riley 2007). These techniques enabled the parameterization of processes such as pathogen spread velocities or the R_0 (Keeling and Rohani 2008, White et al. 2018). However, these methods operate at the population level, thereby neglecting individual differences and thus limiting their capacity to identify the mechanisms driving the pathogen spatial dynamics (Tompkins et al. 2011).

To overcome this limitation, the metapopulation approach divides the population into separate subpopulations or groups, each with its own transmission dynamics, while disease spread can also occur between groups through movement or contact (Hanski and Simberloff 1997, White et al. 2018). It operates as an ensemble of SIR models (Fig. 2), incorporating variability in pathogen dynamics (such as the R_0) within different groups and between groups (Hess 1996, McCallum and Dobson 2002, Cross et al. 2007). This approach has also been applied to vector-borne diseases, such as West Nile Virus, by coupling separate SIR models for pathogen dynamics within hosts (e.g. birds) and within vectors (e.g. mosquitoes), linked through a host-vector transmission model (Wonham et al. 2004).

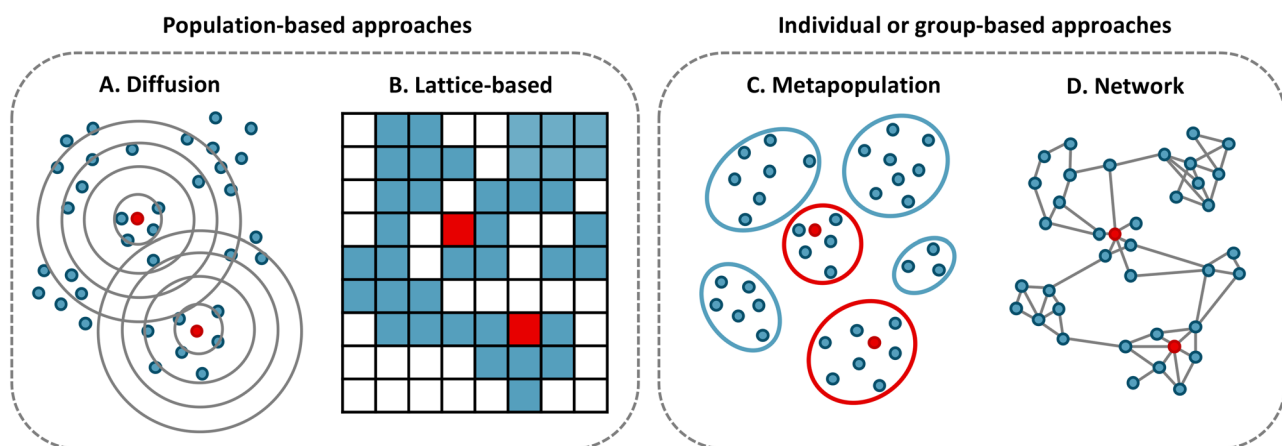


Figure 2. Spatial approaches for incorporating space into Susceptible-Infected-Removed (SIR) models. Red color represents infected individuals or cells, while blue color represents those that are susceptible to infection. In Diffusion (A), susceptible individuals in close proximity to infected individuals will be the first to become infected (Maki and Hirose 2013). In lattice-based models (B), infection is considered at the cell level rather than the individual level. Consequently, susceptible cells in close proximity to an infected cell will be the first to become infected (Riley 2007). The metapopulation model (C) operates as an ensemble of SIR models. An internal model is employed to determine the probability of infection for an individual within a group by an infected individual of the same group, while an external model is utilized to ascertain the probability of a susceptible group becoming infected by an infected group (Hess 1996). In the network model (D), transmission is facilitated by the connections (edges) between individuals (nodes), and it can incorporate both a metapopulation model and a lattice-based model (Craft 2015).

Network analysis, implemented in epidemiology after its development in sociology (Newman 2002, May 2006), goes one step further in capturing heterogeneity in transmission dynamics. It treats individuals or groups as nodes connected by edges and allows attributes to be assigned to both, thereby enabling individual traits and relationship properties to be considered as modifiers of the transmission dynamics (Craft 2015, White et al. 2017). Network analysis has been widely applied in wildlife disease research to investigate pathogen transmission. By capturing contact patterns and social structure in both intra- and inter-specific relationships, it helps identify key hosts, transmission pathways, and potential hotspots (Craft 2015, Farine and Whitehead 2015, Silk et al. 2017a, 2017b). For example, Triguero-Ocaña et al. (2020) applied network analysis at the individual level (nodes=individuals) using GPS collar data to study interactions (edges=interactions) between wildlife and domestic livestock in an arid region of Spain, identifying the formation of mixed groups of cattle and fallow deer *Dama dama* with potential implications for bovine tuberculosis transmission. In contrast, Grange et al. (2014) used this approach at the population level (nodes=populations) to evaluate connections between tsetse *Porphyrio hochstetteri* populations through individual translocations (edges=translocations), highlighting the risk of pathogen spread associated with translocating individuals between isolated populations and identifying key sites for disease surveillance.

Recent methodological developments have moved network analysis toward a more mechanistic representation of transmission by explicitly integrating host movement, behavioral states, and environmental context (Manlove et al. 2022, Wilber et al. 2022). High-resolution GPS, and sometimes even accelerometry data, analyzed through hidden Markov models (HMMs), now allow the inference of behavioral states during contacts (such as foraging, resting, or transit), enabling the construction of networks in which the probability of transmission varies according to the type of interaction (Dougherty et al. 2018, Manlove et al. 2018, Silk et al. 2019). At the same time, fine-scale spatiotemporal environmental information can be associated with the locations and times at which individuals are present and contacts occur (Altizer et al. 2006). These data streams have been incorporated into emerging modeling frameworks, including movement-driven dynamic contact networks (Dougherty et al. 2022a, Dekelaita et al. 2023, Yang et al. 2023), and hierarchical models integrating space use, behavior, and environmental covariates (Talukder et al. 2025). These models allow assigning a unique transmission risk or probability to each contact, according to the characteristics of the contact, the host, the environment, and the way these elements interact (Webber et al. 2023). For example, Dougherty et al. (2022b) integrated Maxent to model the environmental niche of the anthrax pathogen *Bacillus anthracis*, HMMs to infer behavioral states (foraging versus non-foraging) in zebra *Equus quagga*, and behavior-specific step-selection functions to assess habitat selection during movement. This framework

revealed that zebras were markedly more likely to select areas of elevated anthrax risk when foraging.

Limitations and future directions

Network models require substantial input data (Craft and Caillaud 2011, Silk et al. 2017b, White et al. 2017). The field methods available to collect the necessary information to build these models are usually expensive and time-consuming, and do not continuously track individuals spatially and temporally (Herraiz et al. 2024a; but see Yang et al. 2023). As a result, transmission is often simplified to a basic contact rate and a uniform probability of transmission per contact (McCallum et al. 2001, Craft 2015). Such simplification requires strong assumptions about the processes driving pathogen spread, which may be far from reality and introduce potential biases (VanderWaal et al. 2016, Herraiz et al. 2024b).

Furthermore, spatiotemporal variability in transmission parameters, combined with the sparse and heterogeneous nature of wildlife data, introduces substantial uncertainty into model outputs (Manlove et al. 2022). Emerging methodologies offer promising directions, integrating contact, behavioral, and environmental data, but future developments should focus on approaches that explicitly incorporate this uncertainty, ensuring that model outputs reflect both variability and potential biases (Han et al. 2020, Muff et al. 2020, Tredennick et al. 2021). Additionally, ML approaches, such as neural networks, could uncover complex, non-linear relationships within wildlife contact networks, detecting high-risk transmission pathways and key hosts, complementing mechanistic network models (Tonks et al. 2024, Mandujano Reyes et al. 2025).

Molecular epidemiology

Molecular epidemiology is based on the genetic typing and subtyping of pathogens (Tibayrenc 1998). Despite its wide application in human epidemiology, it was rarely implemented in wildlife diseases research prior to 2000 (Tompkins and Wilson 1998, Benton et al. 2014). Molecular epidemiology not only identifies genetic variants of a pathogen, but also its phylogeny – the degree of relatedness among those variants. In addition to its proven value in studying host–pathogen interactions and vaccine development, molecular epidemiology has great potential for studying the spatiotemporal dynamics of pathogens in wildlife (Benton et al. 2014, Blanchong et al. 2016). Crucially, the identification of the same pathogen variant in various individuals or species indicates an epidemiological linkage between them. For instance, it has been widely applied to trace pathogen transmission between livestock and wildlife (Aranaz et al. 2004, Kamath et al. 2016).

Phylogeography, which is the spatial representation of a pathogen's phylogeny in this context, can assist in identifying potential causes of disease outbreaks or expansion routes, as well as detecting novel transmission events (Carnegie et al. 2023). Molecular information can also provide direct evidence of transmission that complements contact-based

approaches. For instance, it can be implemented into contact networks, with the advantage that the links or edges of the network refer to where pathogen transmission has effectively occurred, rather than to contacts that do not necessarily imply transmission (Gilbertson et al. 2018, White et al. 2018). For example, VanderWaal et al. (2014) constructed a network at the livestock–wildlife interface in Kenya using subtyping of *Escherichia coli*, defining edges as individuals sharing the same subtype. This approach identified Grant's gazelle *Gazella granti* as a highly connected species within the network, and zebras *Equus burchelli* as a key bridge for linking different groups and species. If sample collection dates are available, it is even possible to determine the direction of transmission, allowing the construction of 'who infected whom' transmission trees and the identification of transmission pathways across individuals and species (Kao et al. 2014, Rossi et al. 2021). For example, Kamath et al. (2016) used spatiotemporal genetic data of *Brucella abortus* in Yellowstone to reconstruct transmission sequences among elk *Cervus canadensis*, bison *Bison bison*, and livestock (cattle and domestic bison) over 48 years, revealing how outbreaks spread over time, and identifying elk as reservoir and source of livestock infections.

Molecular techniques have also been applied to the study of vector-borne diseases to identify the species from which vectors feed and build networks based on potential transmission pathways (Blanchong et al. 2016). Moreover, the genetic characterization of hosts, vectors, and pathogens can be integrated with biogeographic and environmental variables to identify how landscape features shape pathogen genetic diversity and its expansion flow (Dellicour et al. 2016), enabling the identification of biogeographic barriers based on genetic differentiation, and testing hypotheses concerning the mode and tempo of pathogen dispersal (Biek and Real 2010).

Limitations and future directions

Despite the demonstrated utility of molecular epidemiology in wildlife disease research, its implementation has been hindered by technical challenges, time constraints, and high costs (Benton et al. 2014). Consequently, most studies on wildlife diseases still rely on serological testing rather than genetic analysis (Fountain-Jones et al. 2018). Moreover, while sequencing technologies have become faster and more cost-effective, integrating these data with epidemiological and ecological models remains methodologically complex.

Given the potential of genetic sequencing for parameterizing pathogen spread and identifying key drivers (Dellicour et al. 2021), future developments are likely to focus on integrative frameworks combining genetic datasets with quantitative models of disease risk – such as risk factor analyses, network models, or agent-based models – helping to reduce uncertainty in transmission inference. These approaches include Bayesian phylodynamic models, coalescent-based methods, and time-resolved phylogenetic reconstructions, which allow estimation of transmission rates, identification of sources and sinks, and reconstruction of spatiotemporal pathogen spread even with sparse sampling

(Guinat et al. 2021, Harvey et al. 2021, Pacioni et al. 2022, Dellicour et al. 2024).

Predictive analysis

Disease distribution modeling

Species distribution modeling in ecology can be traced back to the early 20th century (Guisan and Zimmermann 2000). These models have been given various names, the most widely accepted being species distribution models (SDMs; Elith and Leathwick 2009) and ecological niche models (ENMs; Peterson 2001). These terms are often used interchangeably, although there is a growing consensus that ENMs aim to model potential niche (i.e. where suitable conditions exist for the presence of a species), while SDMs aim to model the realized niche or actual distribution (Peterson and Soberón 2012). This approach has great potential to study emerging pathogens, enabling the identification of both their potential future spread and their present but unknown distribution (Stevens and Pfeiffer 2011, Peterson 2014). The rationale is analogous to that of risk factor analysis: associating pathogen occurrence with factors that can explain its distribution (Fig. 3; Peterson 2007). However, while risk factor analysis focuses on the associations between the risk and the factors, ENMs and SDMs serve primarily a predictive purpose. Therefore, they typically seek a simpler fit – primarily based on environmental factors – that can predict pathogen distribution across geographic regions or time periods beyond those used for calibration, rather than a perfect fit aiming to identify all factors influencing pathogen occurrence (Escobar 2020).

Pathogen distribution modeling is conceptually and analytically more complex than animal or plant modeling, as it involves interactions among multiple agents, such as host, pathogen, and vectors. Consequently, Johnson et al. (2019) identified two main approaches: 1) black-box, which models pathogen occurrence, assuming that it captures the underlying interactions (Baz-Flores et al. 2024), and 2) component-based, which models the involved components (pathogen, host, vector), treating the niche as the intersection between the niches of these components (Cuervo et al. 2022). This latter approach enables more accurate identification of potential transmission and expansion sites. However, it requires thorough knowledge of the pathogen, its transmission mechanisms, and all components involved, as neglecting a relevant species can lead to underestimation of risk (Johnson et al. 2019).

Joint Species Distribution Models (JSDMs) extend component-based approaches by modeling multiple interacting species simultaneously, accounting for shared environmental responses and correlations between species (Pollock et al. 2014). This allows better predictions and helps identify whether co-occurrence is due to environment or true biotic interactions. For example, Veitch et al. (2020) applied a JSDM to ectoparasites of deer mice *Peromyscus maniculatus*, revealing positive and negative associations between mites, botflies, and fleas, showing how host traits and parasite

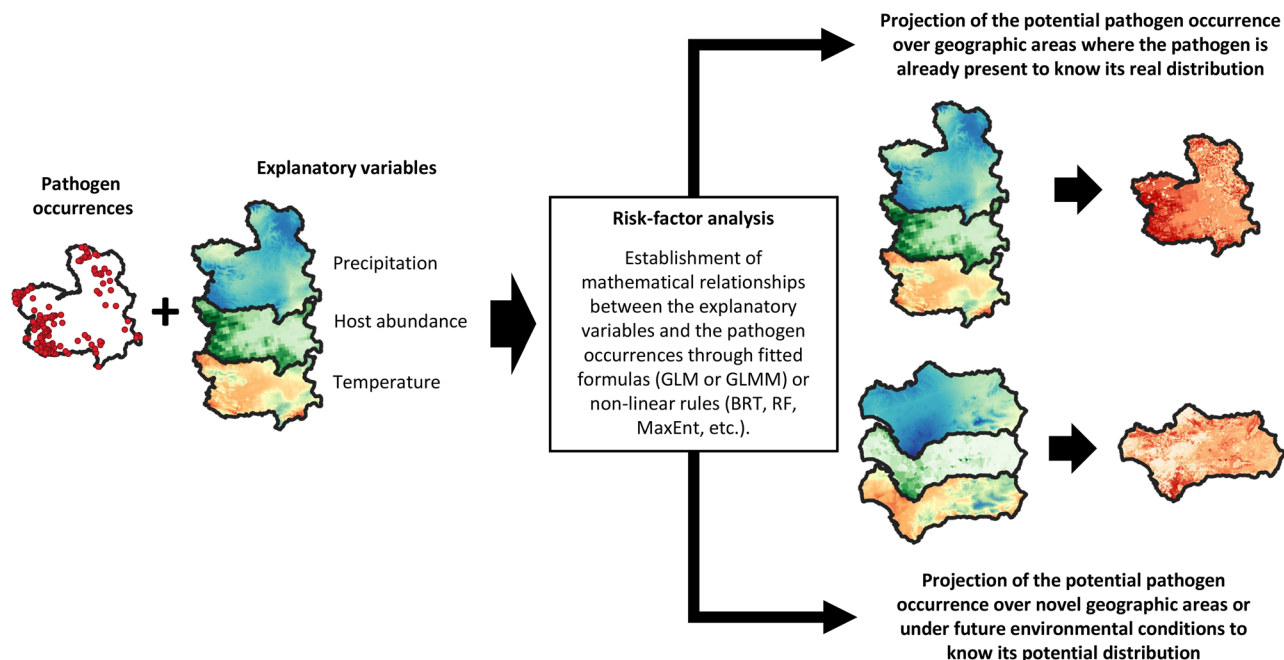


Figure 3. Workflow for identifying risk factors and projecting pathogen distribution. Pathogen occurrences can be related to explanatory variables (such as precipitation, host abundance or temperature) to identify risk factors (Lawson 2018). This association can be made using generalized linear models (GLM), which can be mixed models including random factors (GLMM), or by establishing non-linear rules, such as through boosted regression trees (BRT), Random Forests (RF), or maximum entropy algorithms (MaxEnt; see Elith et al. 2006 for further details, Stevens and Pfeiffer 2011). This relationship can subsequently be used to project the actual distribution of the pathogen in areas where it is present or to project the potential distribution in areas where it is not present (Peterson and Soberón 2012). Although risk-factor analysis, SDMs, and ENMs fall within the same methodological framework, the choice of explanatory variables and the method for establishing mathematical associations may vary depending on the purpose (Escobar 2020).

life-history shape co-occurrence patterns. This approach also improves outputs for poorly documented species or pathogens. For instance, Mowry et al. (2025) showed how JSMD can be used to overcome sparse data, by jointly modeling the distributions of multiple vector species, using human disease records – more widely available than direct vector occurrence data – as a proxy for vector presence and occurrence data from well-documented related vector species.

Beyond JSMDs, ensemble SDMs offer a complementary approach to improve predictive robustness by combining predictions from multiple algorithms, reducing dependence on any single method's assumptions and improving transferability across contexts (Elith et al. 2006, Acosta et al. 2024). Additionally, spatially explicit capture-recapture (SCR) models represent an underutilized but promising tool, simultaneously estimating population density, spatial disease prevalence, and transmission probabilities while explicitly accounting for imperfect detection (Royle and Dorazio 2008). For example, Ketwaroo et al. (2025) applied SCR to European badgers *Meles meles* infected with bovine tuberculosis to estimate spatially explicit prevalence and density, examine their relationship, and assess the effects of infection on survival and space use.

Limitations and future directions

Pathogen distribution modeling, particularly for wildlife, faces several key limitations. ENMs and SDMs are mainly applied

over extensive geographical areas, and wildlife health data at this scale are usually scarce (Gortazar et al. 2014, Escobar and Craft 2016), although modeling based on other components, such as vectors and hosts, can provide certain insights into the potential risk of pathogen occurrence (Peralbo-Moreno et al. 2022, Mowry et al. 2025). Furthermore, most ENMs and SDMs applied to pathogens rely on abiotic factors rather than biotic interactions, whose implementation in these models is less developed (Elith and Leathwick 2009, Johnson et al. 2019, Escobar 2020). However, the biotic component may be more relevant in epidemiological models than in ecological models, especially for directly transmitted pathogens, potentially leading to unreliable predictions (Peterson 2008, 2014, Carlson et al. 2020, Escobar 2020). Moreover, ENMs and SDMs often assume that a pathogen's response to environmental conditions is constant across time and space. Consequently, projecting models to regions or climate scenarios far removed from calibration conditions can substantially reduce their reliability (Owens et al. 2013).

Recent advances in JSMD offer promising avenues to address current limitations in pathogen distribution modeling, helping to capture biotic interactions that are typically overlooked in traditional ENMs and SDMs and improving predictions with sparse data (Veitch et al. 2020, Mowry et al. 2025). Future work should focus on integrating JSMD with other ecological and epidemiological information, such as movement patterns (Integrated Movement Models; Miller

and Holloway 2015, Buderman et al. 2024) or vector dynamics, to enhance predictive accuracy and better represent the mechanisms driving pathogen distribution.

It should also be noted that, despite the advances in this approach, distribution modeling of pathogens still often relies on approaches rooted in veterinary epidemiology, essentially projecting risk factor analyses spatially rather than applying ENMs or SDMs. Some authors attribute this to a lack of collaboration between epidemiologists and veterinarians on one side, and ecologists and biogeographers on the other (Johnson et al. 2019, Escobar 2020).

Simulations of disease dynamics

Simulations for studying pathogens in wildlife began with the development of the aforementioned compartmental models (Barlow 1995). These models simulate the expansion of the pathogen through continuous or discretized space based on the parameterization of the spatiotemporal dynamics of the pathogen occurrences (Murray et al. 1986). Simple simulations require only spread velocities, but incorporating additional parameters provides a more comprehensive understanding of their impact on pathogen spread, improving the assessment of control measures (Barlow 1996).

Early simulations of wildlife disease spread largely relied on a top-down approach, wherein system-level parameters such as birth, death, or infection rates are parameterized, affecting the agents involved in the system (Viboud et al. 2006). In contrast, agent-based models (ABMs), also known as individual-based models (IBMs), originally developed in sociology, follow a primarily bottom-up approach, where the parameterization of the agents comprising the system scales up into effects at the system level (Huston et al. 1988, Macy and Willer 2002). ABMs comprise agents (e.g. individuals), the environment in which they exist (e.g. land use), and the processes governing agents (e.g. birth and death), the environment (e.g. degradation), and their interactions (e.g. resource consumption) (Railsback and Grimm 2011). For instance, the rate of new infections per unit of time can be directly incorporated into a top-down approach model (Kilpatrick et al. 2009, Almberg et al. 2021, Pepin et al. 2022). However, in an ABM approach, the likelihood of each agent contracting an infection can be broken down into the probability of coming into contact with an infected individual and the probability of becoming infected upon contact (Halasa et al. 2019, Belsare and Stewart 2020). These probabilities are simultaneously influenced by agent movement patterns, contact characteristics, or environmental conditions (Manlove et al. 2022, Wilber et al. 2022). Therefore, ABMs enable the implementation of the complexity of behavior, movement, and individual particularities in the simulation. Consequently, although more complex and computationally demanding, they provide a finer-grained and more mechanistic representation of disease dynamics, enabling the analysis of spatial transmission patterns as emergent outcomes of individual-level processes (Alexander et al. 2012). Furthermore, ABMs are not restricted to a purely bottom-up logic; system-level parameters can also

be incorporated alongside individual-level rules within a single modeling framework.

An example of both modeling approaches can be seen in studies of chronic wasting disease (CWD) in white-tailed *Odocoileus virginianus* and mule deer *Odocoileus hemionus* in North America. Top-down models have been used to approximate the impact of the disease on population demographics and to test management interventions, such as culling (Almberg et al. 2011, Potapov et al. 2016). ABMs have incorporated host behavioral aspects, such as group dynamics, allowing researchers to better estimate the probability of detecting the disease through sampling of harvested individuals and to identify critical moments for implementing disease control measures (Belsare et al. 2020a, 2020b).

Limitations and future directions

Both top-down and bottom-up approaches are limited in that the results obtained are strongly linked to the assumptions made during parameterization (Wonham et al. 2006, Getz et al. 2019). This limits the applicability of the top-down models under novel conditions – different from those used for model calibration – as parameterization derived from a different system may yield unreliable predictions (Winter and Escobar 2020). The bottom-up nature of ABMs provides greater flexibility to overcome this constraint: by operating at finer mechanistic resolution and parameterizing processes at the individual level, ABMs can produce emergent patterns without requiring them to be parameterized a priori, which is a major advantage in novel or poorly understood systems. However, validation becomes substantially more challenging when emergent outcomes cannot be directly compared to observed data (Collins et al. 2024, Serdyuk 2025). More fundamentally, the primary limitation of ABMs is computational. A fully realistic representation of a complex wildlife system would require parameterizing an enormous number of processes and running many replicate simulations to calibrate the parameters, analyze sensitivity and characterize stochastic variability, rapidly becoming computationally intractable (Ten Broeke et al. 2016, Borgonovo et al. 2022, Chopra et al. 2025). In practice, ABMs must therefore be simplified and oriented toward specific objectives, which may mean omitting factors or mechanisms that influence the emergent patterns of interest, ultimately reducing predictive capacity and limiting the resolution at which transmission dynamics can be understood (Darwish and Leonenko 2026). Furthermore, imprecise parameterization at the agent level can propagate into inaccurate predictions at the system level (Macy and Willer 2002), making thorough knowledge of the species, pathogen, and environment especially critical. Therefore, factors such as whether the pathogen is multi-host or the changes it can induce in host movement patterns or contact structure must be considered (Craft 2015, Alderton et al. 2016, Dekelaita et al. 2023). Similarly, when evaluating control measures, it is important to consider their effect on agent behavior, such as dispersal or social disruption when culling is applied (McDonald et al. 2008).

To improve the reliability of simulation results, empirical studies can be used to calibrate and validate both top-down and bottom-up approaches, ensuring that epidemiological and ecological processes and their interactions are realistically represented (Tompkins et al. 2011, Gortázar et al. 2016, Lange and Thulke 2016). Furthermore, developing standardized frameworks for parameterization and sensitivity analysis can help quantify the uncertainty in model predictions, improving their reliability (Grimm et al. 2005, Borgonovo et al. 2022).

Concluding remarks

Selecting the appropriate analytical approach in spatial epidemiology requires balancing the pathogen's characteristics (emerging versus endemic, direct versus indirect or vector-borne transmission, etc.), the spatial and temporal scales of interest, and the specific aims of the study (Tompkins et al. 2011, McCallum 2016). No single approach is universally optimal; each provides a complementary perspective that must be understood within its conceptual and methodological scope (Alexander et al. 2012). Furthermore, combining approaches allows their strengths to be leveraged for a more complete understanding of pathogen dynamics across spatial scales (Fig. 4).

Descriptive methods such as disease mapping or cluster analysis remain essential across systems and scales, offering an initial view of pathogen patterns and generating early hypotheses about potential risk factors (Pfeiffer et al. 2008). Risk factor analyses then enable hypotheses testing and quantification of associations between pathogens and ecological and anthropogenic variables, though they are constrained by data availability and correlation-causation ambiguity (Hofstetter et al. 2014).

Mechanistic frameworks deepen our understanding of pathogen dynamics by explicitly parameterizing transmission processes. Depending on their structure, they represent transmission at different organizational levels. Population-level SIR models primarily quantify pathogen spread, but their aggregated structure limits their capacity to capture the mechanisms underlying endemic persistence (Heesterbeek and Roberts 1995, White et al. 2018). In contrast, network-based models emphasize behavioral and social interactions that can sustain endemic pathogens or facilitate the spread of emerging ones (Springer et al. 2017). However, their applicability to vector-borne diseases is limited, as transmission in such systems is driven by vector ecology rather than direct host contacts, though integration with molecular data can partially address this constraint (Nava-Doctor et al. 2021). Moreover, while network analysis can theoretically be scaled up, it is most effective at local or community levels, where social structure and behavior play a stronger role in transmission dynamics (Silk et al. 2017a).

Predictive approaches such as ENMs and SDMs are powerful at broad spatial scales, projecting potential or realized pathogen distributions under current or future environmental

conditions. However, their strong reliance on abiotic predictors and limited incorporation of biotic interactions reduce their reliability for directly transmitted pathogens or fine-scale epidemiological dynamics (Peterson 2008, Escobar et al. 2020). Although these models hold great potential for studying emerging and understudied pathogens, they may not be as useful for studying endemic pathogens with a known distribution (Sutherst 2013). Conversely, ABMs operate at the opposite end of the spatial scale, integrating individual behavior, environmental heterogeneity, and feedback mechanisms to simulate transmission dynamics (Marion et al. 2008, McCallum 2016). Their detailed parameterization enables a deeper understanding of disease dynamics and the evaluation of control strategies, but the potential impact of these parameters on large-scale disease dynamics and the computational constraints limit their feasibility at broad scales (Grimm et al. 2005, Lane-deGraaf et al. 2013).

A cross-cutting limitation of the framework presented here – and of spatial epidemiology more broadly – is the implicit assumption that occurrence data are sufficiently complete and unbiased. As discussed throughout this review, surveillance in wildlife is frequently opportunistic and spatially heterogeneous, meaning that analytical outputs may partly reflect variation in reporting capacity rather than true underlying disease risk. Researchers should therefore interpret results in light of this constraint, and where possible incorporate covariates or model structures that explicitly account for spatial variation in detection probability (Allen et al. 2017, Stephens et al. 2021).

Ultimately, progress in wildlife spatial epidemiology will likely depend less on the proliferation of new analytical tools than on how effectively existing ones are combined and interpreted. Rather than selecting methods in isolation, researchers should aim to integrate descriptive, exploratory, and predictive approaches into interoperable metamodels capable of linking processes across scales (Alexander et al. 2012, Lacy et al. 2013). Achieving this integration requires conceptual coherence in how epidemiological and ecological processes are defined (Hassell et al. 2021), technical interoperability allowing models and data to be combined across pathogens and spatial scales (Garabed et al. 2019, Doran et al. 2023), and validation strategies ensuring that statistical results are biologically and epidemiologically meaningful (Heesterbeek et al. 2015). As methods grow increasingly sophisticated, their correct application will rely on a deeper understanding of the assumptions and limitations underlying each discipline. By promoting conceptual transparency and true interdisciplinarity, spatial epidemiology can move from fragmented methodological advances toward a robust and unified framework that more effectively informs wildlife disease management, conservation, and policy.

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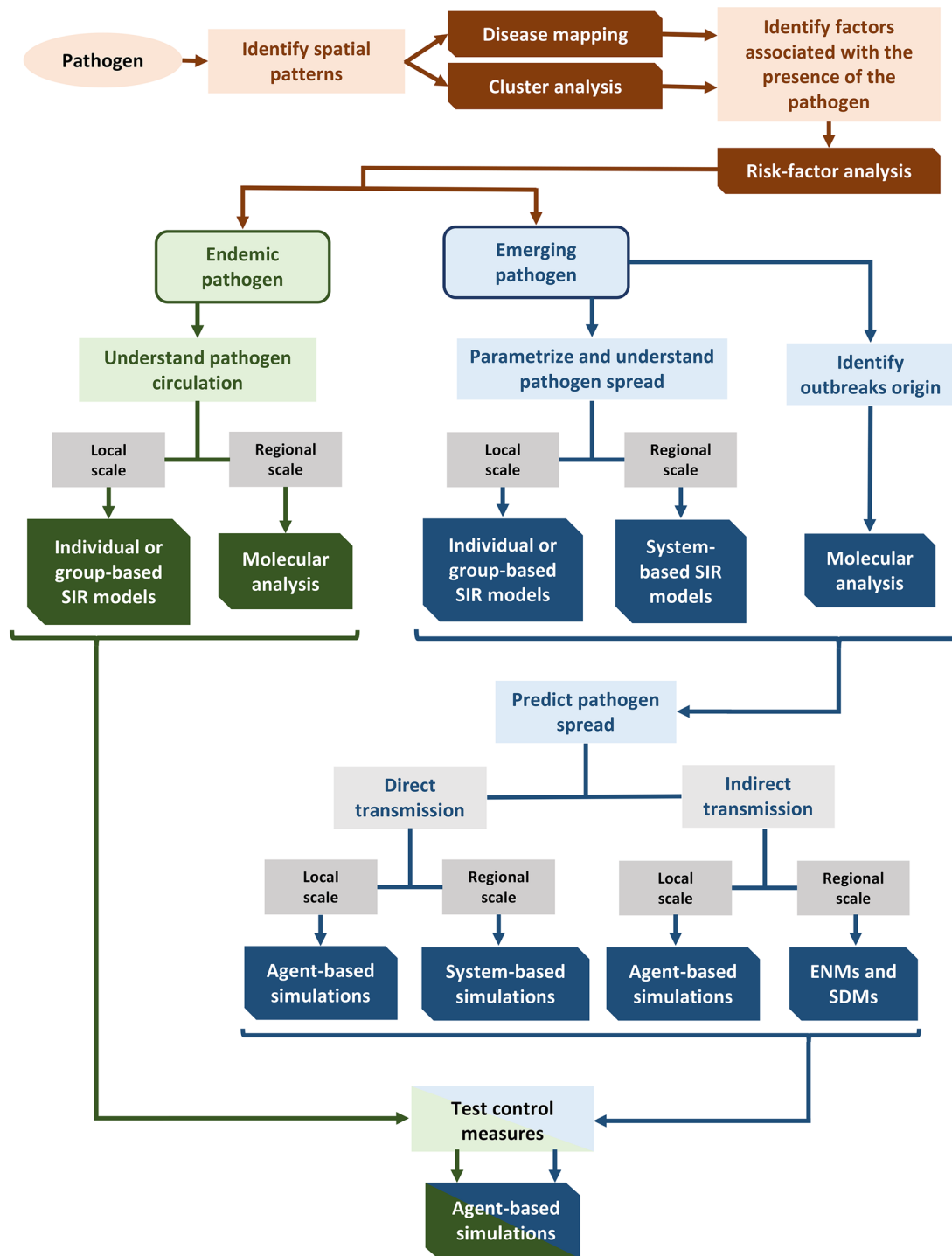


Figure 4. A workflow for studying emerging and endemic pathogens in wildlife from a spatial perspective to inform policy making. The initial steps require few input data and provide valuable information for designing measures and guiding subsequent steps. In the case of endemic pathogens, the following step is to delve into how the pathogen circulates. For emerging pathogens, it is necessary to identify the origin of the outbreaks and parameterize and simulate the pathogen spread. Finally, the designed control measures must be tested.

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Author contributions

Cesar Herraiz: Conceptualization (equal); Investigation (lead); Methodology (lead); Validation (lead); Writing – original draft (lead); Writing – review and editing (equal). **Pelayo Acevedo:** Conceptualization (equal); Methodology (supporting); Resources (lead); Writing – original draft (supporting); Writing – review and editing (equal).

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Data availability statement

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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