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

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Abstract

Wildlife diseases pose a significant threat to public health, livestock, and biodiversity conservation. In this context, spatial epidemiology offers a robust framework for elucidating disease dynamics and informing policy-making and disease management. The workflow in spatial epidemiology involves three main steps: (1) descriptive analysis of spatial dynamics; (2) exploration of the observed dynamics; and (3) prediction of pathogen distribution and spread. Descriptive analysis, such as disease mapping or clustering analysis, focuses on identifying spatial patterns, enabling the formulation of hypotheses regarding potential risk factors driving disease dynamics. Subsequently, risk factor analysis associates the presence of the pathogen with ecological or anthropogenic factors to explain its spatiotemporal dynamics. Furthermore, the circulation and spread of endemic and emerging pathogens can be further understood at a finer resolution. System-based Susceptible-Infected-Recovered (SIR) models, such as diffusion and lattice-based, allow for the parametrization of pathogen spread at the population level, while individual or group-based SIR models, such as metapopulation or network approaches, consider the impact of host behavior and social structure in disease dynamics. Molecular epidemiology, through the identification of genetic variants of pathogens and the mapping of their phylogenetic relationships, aids in understanding outbreak origins and the epidemiological linkages among hosts. Finally, for emerging pathogens, the knowledge about disease dynamics should be implemented in predictive modeling for anticipating disease spread. In this regard, ecological niche models and species distribution models project potential pathogen distributions through their association with ecological or anthropogenic factors, whereas simulations parameterize the processes of pathogen spread to predict its expansion over time. System-based simulations focus on population-level dynamics, while agent-based simulations incorporate individual-level dynamics, offering detailed insights into disease spread and control measures. Consequently, the integration of descriptive analyses, exploratory procedures, and predictive models provides a robust framework for addressing the challenges posed by wildlife diseases and developing management and control measures. The interdisciplinary approach in spatial epidemiology is crucial for mitigating the impact of wildlife diseases on

public health and biodiversity, emphasizing the need for collaboration among ecologists, epidemiologists, statisticians, and policy-makers.

Keywords: agent-based models, cluster analysis, disease mapping, emerging diseases, endemic diseases, interdisciplinary approach, network analysis, risk factor.

1) Introduction

Wildlife diseases, understood as pathogens present in wildlife (WOAH 2015), have an impact on human and livestock health, leading to public health issues and substantial economic losses worldwide (Siembieda *et al.*, 2011; Wolfe, Dunavan & Diamond, 2007; Knight-Jones & Rushton, 2013). Moreover, these pathogens can threaten the conservation efforts of endangered species, as outbreaks can devastate already vulnerable populations (Pedersen *et al.*, 2007; Daszak, Cunningham & Hyatt, 2000). Spatial epidemiology offers a crucial framework for understanding and mitigating these challenges by elucidating spatial patterns of disease transmission, identifying high-risk areas and species, and informing targeted interventions across diverse ecosystems (Stevens & Pfeiffer, 2011).

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 (Gortázar *et al.*, 2007; Simpson, 2002). Furthermore, issues such as sample collection, data gathering on populations and individuals, and the consideration of animal behavior and social dynamics, present unique challenges when working with wild animals not previously found in human and domestic animal populations (Stallknecht, 2007; Morner *et al.*, 2002). 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 became a key tool for overcoming all the aforementioned difficulties, understanding pathogens in relation to the host and the environment under the perspective of the "disease ecology" (Tompkins & 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).

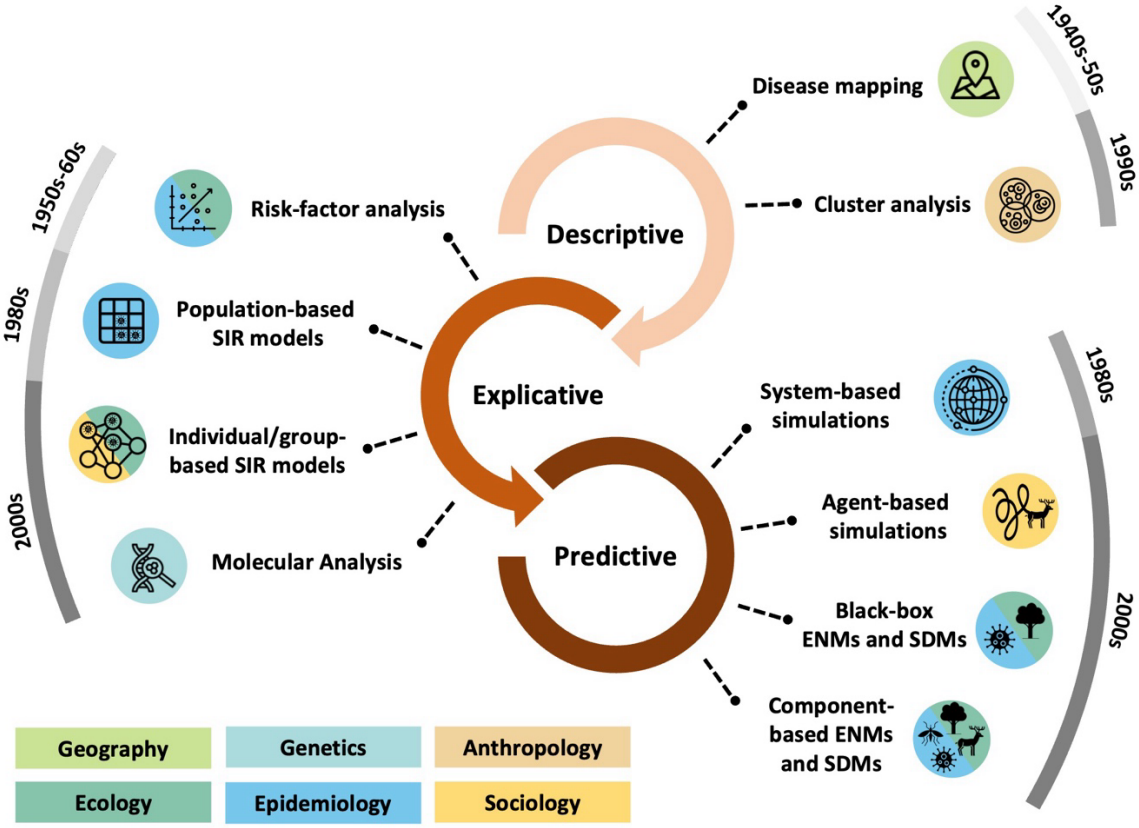

Figure 1. Historical framework for the interdisciplinary development of methodologies used in the spatial epidemiology of wildlife diseases. These methodologies are classified according to their purpose: (1) to describe spatial patterns, (2) to explain these patterns, and (3) to predict the evolution of these patterns. The green, blue and yellow colors indicate the 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.

The field of spatial epidemiology has experienced remarkable advancements driven by this interdisciplinary input, enabling researchers to gain deeper insights into the dynamics of pathogen transmission among wildlife populations and at the wildlife-livestock-human interface (Gortazar *et al.*, 2014). However, the in-depth development of monitoring and analytical tools has led to their isolation, lacking a broad framework for studying the spatial and temporal dynamics of pathogens in wildlife. This review presents a comprehensive overview of spatial analysis in wildlife epidemiology. The workflow in spatial epidemiology is based on three key steps: (1) identifying and describing spatial patterns, (2) exploring these patterns and identifying the factors that explain them, and (3) predicting the behavior of these patterns(Bailey & Gatrell 1995; Pfeiffer *et al.* 2008; Pfeiffer & Stevens 2015). We walked through the evolution of the field across these steps, from early descriptive analyses to state-of-the-art methodologies for understanding and predicting spatial pathogen dynamics (see

Figure 1). Furthermore, we explored the origin of these methodologies to understand their strengths, weaknesses, and potential applications (see Table 1), as well as in producing insights for their future development.

2) Descriptive epidemiology

The investigation of wildlife diseases gained momentum in the 1950s and the 1960s, with surveys of wild individuals to detect pathogens that could potentially pose a threat to public health or a risk of transmission to domestic animals (Keymer 1959; Mair 1973). Some studies included descriptive maps indicating the geographic locations of samples or positive cases (e.g., Anderson & Beaudoin 1966; Beauregard 1969). However, most studies only indicated the detected pathogens or their prevalence (Andrews 1969; Hedger 1972).

Cluster analysis is a type of descriptive analysis originally developed in anthropology and later applied to epidemiology. This analysis can identify spatial patterns in the distribution of pathogen occurrences (usually derived from cases) compared to an expected distribution (null hypothesis), which is typically random (Ward & Carpenter 2000). Pathogens naturally tend to form spatial clusters, meaning that occurrences are often concentrated in certain areas (Wartenberg 2001; Albery et al. 2021). 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 serves as a preliminary step in identifying spatial patterns (potential outbreaks) in pathogen distribution (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 formulation of directional hypotheses regarding potential risk factors driving the spatiotemporal dynamics of pathogens (Bailey & Gatrell 1995). Subsequently, these hypotheses must be tested through more refined and comprehensive statistical analyses, such as risk factor analysis (Artois et al. 2009; Hofstetter et al. 2014).

115 **Table 1.** Main analytical approaches employed in the study of pathogens in wildlife from a spatial and/or spatiotemporal perspective.

116

Approach	Input info	Output info	Potential	Strengths	Weaknesses	Key references
Disease mapping	Pathogen occurrence.	Spatial representation of the cases or infection rates.	Identification of areas for focusing surveillance and control measures.	Enables the formulation of hypotheses.	Further analysis is needed to explore the factors driving the observed patterns.	Pfeiffer *et al.* (2008)
Clustering analysis	Pathogen occurrence.	Spatial aggregation of the occurrences.	Identification of areas for focusing surveillance and control measures.	Enables the formulation of hypotheses.	Further analysis is needed to explore the factors driving the observed patterns.	Tango (2010); Ward and Carpenter (2000)
Risk factor analysis	Pathogen occurrence. Risk factors (e.g., environmental conditions, management, hosts presence, etc.).	Factors associated with pathogen risk.	Identification of factors on which efforts to control the pathogen can be focused.	Allows for research at any scale. Does not require individual data.	Difficult to obtain information from wildlife (requires active surveillance for quality data). Limited ability to assess how risk factor effects vary across space. Correlation does not imply causation.	Bender (2009); Blangiardo and Cameletti (2015); Lawson, Browne and Rodeiro (2003); Pfeiffer *et al.* (2008)
Population-based SIR models – Diffusion and lattice-based models	Pathogen occurrence.	Basic reproduction rate. Pathogen spreading velocities.	Quantification of pathogen transmission and spread parameters that can help identify at-risk areas. Influence of parameters (such as demographical or environmental) on pathogen dynamics.	Does not require individual data. Simple statistics and low computational requirements.	Does not capture the social and ecological complexity of the system, so only suitable for simple parameter inference.	Heesterbeek and Roberts (1995); Keeling and Rohani (2008); Maki and Hirose (2013); Riley (2007)

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. Identification of the influence of individual, social and environmental traits on contact patterns.	Provides highly detailed information about pathogen transmission dynamics.	Requires individual or group data (intensive field work). High computational requirements. Highly dependent on the assumptions about transmission and contact dynamics. Transmission is a complex process that is difficult to implement. Interaction does not imply transmission. Vector-borne diseases are beyond scope.	Craft (2015); Farine and Whitehead (2015); Silk *et al.* (2017a); Silk *et al.* (2017b)
Molecular analysis	Pathogen occurrence with genetic/genomic data.	Pathogen phylogeography.	Identification of reservoirs. Identification of entrance and spread pathways and velocities, even for endemic pathogens. Identification of the direction of transmission. Association between landscape and genetic flow.	Works with evidence of transmission at the individual level.	Economically and technically demanding.	Blanchong *et al.* (2016); Dellicour *et al.* (2021); Fountain-Jones *et al.* (2018)
Black-box ENMs and SDMs	Pathogen occurrence. Explanatory factors (e.g., environmental conditions, hosts presence, etc.).	Pathogen potential or realized niche.	Identification of potential risk areas for pathogen spread. Identification of areas where the pathogen is present but has not been detected.	Operates with passive surveillance and low-quality data. Does not require local scale information.	Neglects biotic interactions, so fine-scale results may be unreliable. Assumes pathogen occurrence captures pathogen-host-vector interactions.	Escobar (2020); Johnson, Escobar and Zambrana-Torrelio (2019); Lawson (2018); Peterson (2014)

Component-based ENMs and SDMs	Components occurrence (pathogen, host, vectors, etc.). Explanatory factors (e.g., environmental conditions, hosts presence, etc.).	Pathogen, host, and vector potential or realized niches.	Identification of potential risk areas for pathogen spread. Identification of areas where the pathogen is present but has not been detected.	Operates with passive surveillance and low-quality data. Does not require local scale information. Considers de the intersection of pathogen-host-vector niches.	Biotic interactions are difficult to implement, so the results at fine scale could be unreliable. Neglecting an involved component may lead to an underestimation of the risk.	Escobar (2020); Johnson, Escobar and Zambrana-Torrelio (2019); Lawson (2018); Peterson (2014)
System-based simulations (top-down approach)	System-level parameterization of the observed patterns in pathogen spread and host ecology.	Simulation of the current and/or future spatiotemporal spread of a pathogen.	Identification of potential risk areas and the time it could take for the pathogen to reach them. Identification of the influence of parameters on pathogen dynamics. Permits the testing of the efficacy of control measures.	Parametrized with observed patterns (overlooking some factors would be acceptable as long as their effect is captured by the observed patterns).	Extrapolating observed patterns to predict under different conditions may result in unreliable predictions. Not breaking down the processes that lead to the observed patterns makes it challenging to identify the factors that influence them.	Hartig *et al.* (2011); Lloyd-Smith *et al.* (2005); McCallum (2016)
Agent-based simulations (bottom-up approach)	Parameterization of ecological traits of the host and the pathogen transmission. Parameterization of the host-host, host-pathogen, host-environment, and pathogen-environment interactions consequences.	Simulation of the current and/or future spatiotemporal spread of a pathogen.	Identification of potential risk areas and the time it could take for the pathogen to reach them. Identification of the influence of parameters on pathogen dynamics. Permits the testing of the efficacy of control measures.	Enables simulations under novel conditions. Enables the effect of each factor on the patterns observed at the system scale to be disentangled.	Requires a large amount of detailed input information for parameterization. Neglecting factors influencing pathogen spread could lead to biased predictions. Computationally demanding.	Grimm *et al.* (2005); Lane-deGraaf *et al.* (2013); Railsback and Grimm (2011)

3) Understanding what's described: Risk factor analysis

Factor analysis has been used in epidemiology to identify factors associated with an increased risk of infection or disease, both at the individual and population levels, leading to the procedure known as risk factor analysis (Giroux 2011). From an early stage, it was postulated that factors such as habitat, climate, host abundance, and anthropogenic disturbance could play a role in the epidemiology of pathogens in wildlife (Elton 1931). However, most studies that addressed factors that might influence the presence or prevalence of pathogens in wildlife focused on host traits, such as species, age, or sex (e.g., Johnston & Beauregard 1969; Hensley 1976). Environmental factors, which had long been considered in ecology to explain the distribution of biodiversity (Guisan & Zimmermann 2000), were later incorporated to explain the patterns of pathogens distribution in wildlife (e.g., Anderson & Beaudoin 1966; Pence & Windberg 1984). With their incorporation, the location of the analyzed samples is considered to be associated with the risk, implementing environmental traits to explain the presence of the pathogen, leading the analysis of risk factors acquiring a spatial dimension. This type of analysis has allowed for large-scale associations between the emergence of infectious pathogens and the diversity of wild hosts (but see Schmidt & Ostfeld 2001 regarding the dilution effect debate; Keesing et al. 2006; Jones et al. 2008; Johnson & Thieltges 2010) and the 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).

The analytical approaches used to implement this spatial aspect in the risk factors analysis in wildlife diseases have undergone a methodological evolution. Early studies relied on a-spatial statistical models, mainly GLMs (primary 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. 2021). 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 (BYM and BYM2) family and Stochastic Partial Differential Equation

(SPDE) based spatial and spatio–temporal models, have become central tools in wildlife spatial epidemiology, as they allow 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. First, sampling and diagnostic testing in wild animal populations continue to pose major challenges, as access to biological samples is often limited and/or opportunistic, and most diagnostic tools have been designed for pathogens in domestic species rather than wildlife (Stallknecht 2007; Gilbert et al. 2013). As a result, disease monitoring and surveillance programs frequently generate spatially heterogeneous datasets with under-sampled areas (Boadella et al. 2011), which can produce strong spatial observational noise that masks the true relationship between covariates and pathogen occurrence. Hierarchical Bayesian frameworks 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 & Cameletti 2015).

Second, the association between risk factors and pathogen dynamics may differ by location, even when dealing with 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 allow in the modeling also allow to assess how the risk factors interact with the disease presence across the space (Mandujano Reyes et al. 2024). Variation partitioning procedures identify the percentage of variability explained by each factor included in the model (Legendre 2007), and its spatial representation could

also potentially enable the identification of 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 identifying a spatial structure, limiting its 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).

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, in order to infer cause-effect relationships, it is essential to have a clear understanding of the underlying processes. Therefore, the statistical outcomes of a risk factor analysis should be interpreted in light of expert knowledge of the pathogen and host ecology.

4) Deepening into the dynamics

a) Dynamic models and network analysis

Dynamic models in epidemiology emerged in the early 20th century with the goal of deepening in the understanding of the dynamics of pathogen transmission through mathematical parameterization (Ross & 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 the quantification of transmission parameters and the identification of determining factors in pathogen spread dynamics (Anderson & 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).

194 In contrast to risk factor analysis, compartmental models offer the advantage of not only revealing the
195 association between the spatial pattern of the pathogen occurrences and a factor, but also delving into the
196 processes through which that factor influences the spatial dynamics of the pathogen. For example, correlational
197 studies identified an association between supplementary feeding and disease risk, such as of bovine tuberculosis
198 in white-tailed deer (*Odocoileus virginianus*) or chronic respiratory disease in house finch (Miller et al. 2003;
199 Moyers et al. 2018), while Becker et al. (2018) utilized an SIR model to delve into the underlying processes
200 driving this correlations, revealing how the resource availability causes attraction of dispersing individuals and
201 increased aggregation.

202 The SIR models can also be non-spatial, lacking an association between the location of individuals and
203 the transmission of pathogens among them (e.g., Almberg et al. 2021). The initial methods for incorporating the
204 spatial component into SIR models were diffusion models (e.g., Murray et al. 1986), which treat space as a
205 continuum (Maki & Hirose 2013), and lattice-based models (e.g., Preston 1973), which discretize space into
206 patches (Riley 2007). These techniques enabled the parameterization of processes such as pathogen spread
207 velocities or the R_0 (Keeling & Rohani 2008; White et al. 2018). However, these methods operate at the
208 population level, thereby neglecting individual differences and thus limiting their capacity to identify the
209 mechanisms driving the pathogen spatial dynamics (Tompkins et al. 2011).

210 To overcome this limitation, the metapopulation approach considers separated populations or groups of
211 individuals that are able to interact between them (Hanski & Simberloff 1997; White et al. 2018). It operates as
212 an ensemble of SIR models (Figure 4), incorporating variability in pathogen dynamics (such as the R_0) for
213 different groups and between groups (Hess 1996; McCallum & Dobson 2002; Cross et al. 2007). This approach
214 has been even used for the study of vector-borne diseases, such as West Nile Virus, through the breakdown of
215 the modeling into host (e.g., birds), vector (e.g., mosquitoes), and host-vector relationships (Wonham et al.
216 2004).

217 Network analysis, implemented in epidemiology through its development in sociology (Newman 2002;
218 May 2006), delves one step further into this differentiation of dynamics. It treats individuals or groups as nodes
219 connected by edges, and allows the assignment of characteristics to both, so that the peculiarities of each

individual as well as the characteristics of their relationships can 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 & Whitehead 2015; Silk et al. 2017a; Silk et al. 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 Takahe (*Porphyrio hochstetteri*) populations through individual translocations (edges = translocations), highlighting epidemiological implications and 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 spatio-temporal environmental information can be linked to the locations and times where individuals and contacts occur (Altizer et al. 2006). These data streams have been incorporated into emerging modelling frameworks, including dynamic contact networks driven by movement trajectories (Dougherty et al. 2022a; Dekelaita et al. 2023; Yang et al. 2023), and hierarchical models combining 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 vs. non-foraging) in zebra (*Equus quagga*), and behavior-specific step-selection

functions to assess habitat selection during movement. This comprehensive framework revealed that zebras exhibited a markedly higher probability of selecting areas with elevated anthrax risk when they are foraging.

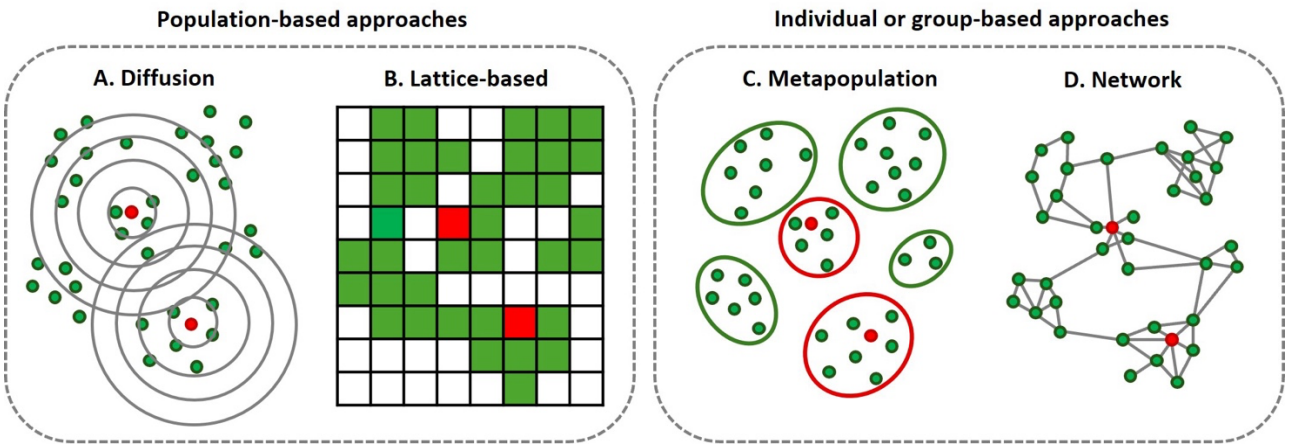

Figure 2. Spatial approaches for incorporating space into SIR models. Red color represents infected individuals or cells, while green color represents those which are susceptible to infection. In Diffusion (A), susceptible individuals in close proximity to infected individuals will be the first to become infected (Maki & 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).

Limitations and future directions

Network models require a significant amount of input data (Craft & Caillaud 2011; Silk et al. 2017b; White et al. 2017). However, 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).

Moreover, despite recent methodological advances that allow integrating host movement, behavioral states, and environmental context into network-based models, in many wildlife systems the necessary data to build such models are still limited. 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 could be far from reality, introducing potential biases (VanderWaal et al. 2016; Herraiz et al. 2024b).

Furthermore, the spatiotemporal variability of all parameters involved in transmission, along with the traits of the available data in wildlife, lead to uncertainty in modeling (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).

b) Molecular epidemiology

Molecular epidemiology emerged 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 & Wilson 1998; Benton et al. 2014). Molecular epidemiology not only identifies the genetic variants of the pathogen, but also its phylogeny, which is the degree of relatedness among these 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).

Phylogeography, which is the spatial representation of a pathogen's phylogeny, can assist in identifying potential causes of disease outbreaks or expansion routes, as well as detecting novel transmission events (Carnegie et al. 2023). Furthermore, the identification of the same pathogen variant in various individuals or species indicates an epidemiological linkage between them. For example, it has been widely applied to trace pathogen transmission between livestock and wildlife (Aranaz et al. 2004; Kamath et al. 2016). Molecular information can also be implemented in contact networks, with the advantage that the links or edges of the network refer to where pathogen transmission has already 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)

294 constructed a network at the livestock–wildlife interface in Kenya using subtyping of *Escherichia coli*, defining
295 edges as individuals sharing the same subtype. This approach identified Grant's gazelle (*Gazella granti*) as a
296 highly connected species within the network and zebras (*Equus burchelli*) as a key bridge for linking different
297 groups and species. If sample collection dates are available, it is even possible to determine the direction of
298 transmission, allowing the construction of "who infected whom" transmission trees and the identification of
299 transmission pathways across individuals and species (Kao et al. 2014; Rossi et al. 2021). For example, Kamath
300 et al. (2016) used spatio-temporal genetic data of *Brucella abortus* in Yellowstone to reconstruct transmission
301 sequences among elk (*Cervus canadensis*), bison (*Bison bison*) and livestock (cattle and domestic bison) over
302 48 years, revealing how outbreaks spread over time, and identifying elk as reservoir and source of livestock
303 infections. Molecular techniques have also been applied to the study of vector-borne diseases to identify the
304 species from which vectors feed and build networks based on potential transmission pathways (Blanchong et al.
305 2016). Moreover, the genetic characterization of hosts, vectors, and pathogens can be combined with
306 biogeographic and environmental variables to identify how the landscape shapes the genetic diversity of the
307 pathogen and its expansion flow (Dellicour et al. 2016), enabling the identification of biogeographic barriers
308 based on genetic differentiation, and testing hypotheses concerning the mode and tempo of pathogen dispersal
309 (Biek & Real 2010).

310 **Limitations and future directions**

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

316 Given the potential of genomic sequencing for parameterizing pathogen spread and identifying
317 determining factors (Dellicour et al. 2021), future developments are likely to focus on integrative frameworks
318 that can combine genetic datasets with quantitative models of disease risk, such as risk factor analyses, network
319 models, or agent-based models, helping reducing the 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).

5) Making predictions

a) Disease distribution modeling

Modeling species distributions in ecology can be traced back to the 20th century (Guisan & Zimmermann 2000). These models have been given various names, the most widely accepted being species distribution models (SDMs; Elith & 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 & 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 & Pfeiffer 2011; Peterson 2014). The rationale is analogous to that of risk factor analysis: to associate the occurrence of the pathogen with factors that can explain its distribution (see Figure 5; 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 usually seek a simpler fit, mainly based on environmental factors, that can predict the pathogen's distribution in response to the conditions, even in an independent system, rather than a perfect fit to identify all factors that may influence the presence of the pathogen (Escobar 2020).

Pathogen distribution modeling adds conceptual and analytical complexities compared to animal or plant modeling, as it involves the interactions between two or more 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 (e.g., 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 (e.g., Cuervo et al. 2022). This latter approach enables more accurate identification of potential pathogen transmission and expansion sites. However, it requires a thorough understanding of the pathogen, its transmission mechanisms, and the components involved, since neglecting a potentially involved species can result in 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 life-history shape co-occurrence patterns. This approach also improves the modeling for poorly documented species or diseases. For instance, Mowry et al. (2025) showed how JSDM can be used to overcome sparse data, by modeling multiple vectors species distributions leveraging data from associated human disease records (much more common) and from other vectors.

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 sanitary data at this scale is usually scarce (Gortazar et al. 2014; Escobar & Craft 2016), although modeling based on other components, such as vectors and hosts, can provide certain insights into the potential risk of pathogen presence (Peralbo-Moreno et al. 2022; Mowry et al. 2025). Furthermore, most of the literature on ENMs and SDMs applied to pathogens is based on abiotic factors, such as environmental variables, rather than biotic interactions, whose implementation in these models is less developed (Elith & Leathwick 2009; Johnson et al. 2019; Escobar 2020). However, the biotic component may be more relevant in epidemiological models than in ecological models, especially in the for directly transmitted pathogens, potentially leading to unreliable predictions when modeling pathogens (Peterson 2008, 2014; Carlson et al. 2020; Escobar 2020). Moreover, when using ENMs and SDMs, it is often assumed that a pathogen's response to environmental conditions is constant across time and space. Consequently, models are frequently projected to different regions or future climate scenarios that may be far removed from

the environmental conditions under which the model was originally calibrated, reducing its reliability (Owens et al. 2013).

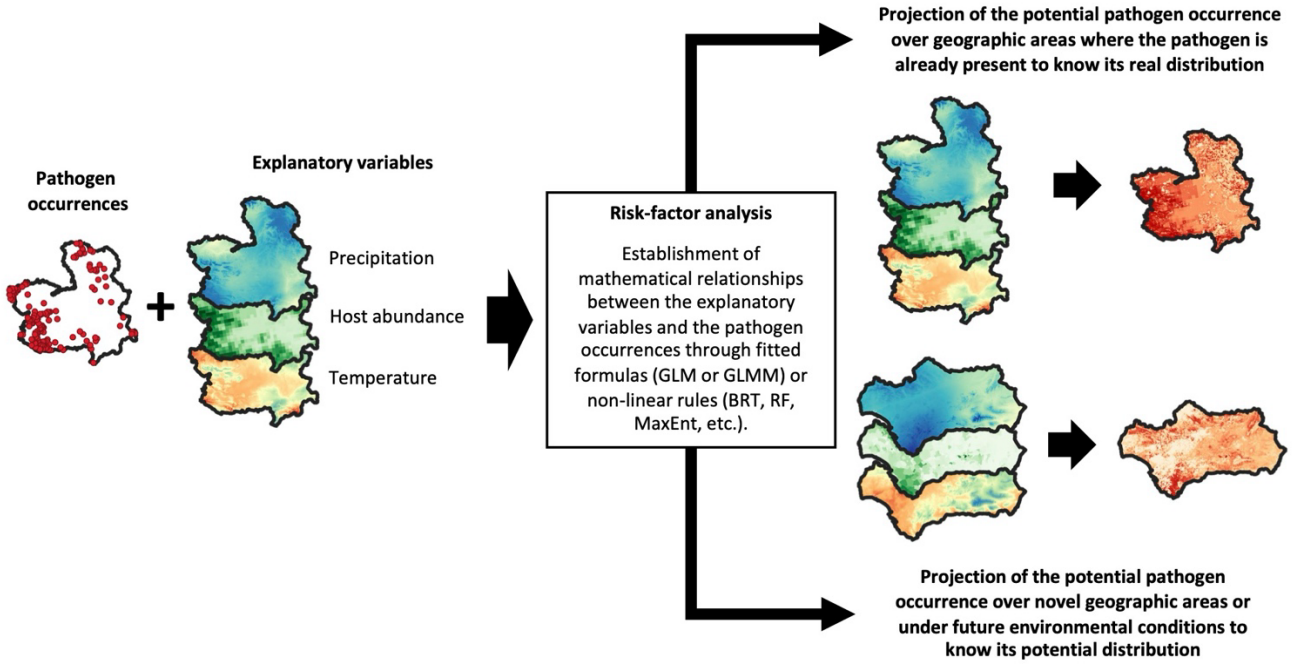

Figure 3. Workflow for identifying risk factors and projecting pathogen distribution. Pathogen occurrences can be related to explanatory variables (such as precipitation, host abundance or temperature) to identify risk factors (Lawson 2018). This association can be made using generalized linear models (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 & 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 & 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).

Recent advances in JSDM 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 JSDMs with other ecological and epidemiological information, such as movement patterns (Integrated Movement Movels; Miller & Holloway 2015; Buderman et al. 2024) or vector dynamics, to enhance predictive accuracy and better represent the mechanisms driving pathogen distribution.

It should be also noted that despite the advances in this approach, disease distribution modeling still often relies on approaches rooted in veterinary epidemiology, essentially projecting risk factor analyses spatially

391 rather than applying ENMs or SDMs. Some authors attribute this to a lack of collaboration between
392 epidemiologists and veterinarians on one side, and ecologists and biogeographers on the other (Johnson et al.
393 2019; Escobar 2020).

394 b) Simulations of disease dynamics

395 Simulations for studying pathogens in wildlife began with the development of the aforementioned
396 compartmental models (Barlow 1995). These models simulate the expansion of the pathogen through continuous
397 or discretized space based on the parameterization of the spatiotemporal dynamics of the pathogen occurrences
398 (Murray et al. 1986). Simple simulations can be constructed with just the spread velocities, but the inclusion of
399 additional parameters provides a more comprehensive understanding of the impact of each parameter on the
400 pathogen spread, improving the assessment of control measures (Barlow 1996).

401 Simulations of the spread of wildlife diseases began with a top-down approach, wherein system-level
402 parameters such as birth, death, or infection rates are parameterized, affecting the agents involved in the system
403 (Viboud et al. 2006). In contrast, agent-based models (ABMs), also known as individual-based models,
404 originally developed in sociology, follow a bottom-up approach, where the parameterization of the agents
405 comprising the system scales up into effects at the system level (Huston et al. 1988; Macy & Willer 2002).
406 ABMs comprise agents (e.g., individuals), the environment in which these agents exist (e.g., land use), and the
407 processes to which the agents (e.g., birth and death), the environment (e.g., degradation), and the interactions
408 between them (e.g., resource consumption) are subjected (Railsback & Grimm 2011). For instance, the rate of
409 new infections per unit of time can be directly incorporated into a top-down approach model (Kilpatrick et al.
410 2009; Almberg et al. 2021; Pepin et al. 2022). However, in ABM approach, the likelihood of each agent
411 contracting an infection can be broken down into the probability of coming into contact with an infected
412 individual and the probability of becoming infected upon contact (Halasa et al. 2019; Belsare & Stewart 2020).
413 These probabilities are simultaneously influenced by agent movement patterns, contact characteristics, or
414 environmental conditions (Manlove et al. 2022; Wilber et al. 2022). Therefore, ABMs enable the implementation
415 of the complexity of behavior, movement, and individual particularities in the simulation. Consequently,
416 although more complex, they permit the generation of results with greater resolution (Alexander et al. 2012).

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 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 approach under novel conditions for the presence of the pathogen, as parameterization based on a system with different conditions may lead to unreliable predictions (Winter & Escobar 2020). The bottom-up approach of ABMs allows overcoming this limitation, but similarly, imprecise parameterization at the agent-level can lead to inaccurate predictions at the system level (Macy & Willer 2002). 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). Therefore, ABMs require a thorough understanding of the biology of the species and the pathogen, as well as the environment in which they are found.

To improve the reliability of simulation results, experimental studies can be used to calibrate and validate both top-down and ABM approaches, ensuring that epidemiological and ecological processes and their interactions are realistically represented (Tompkins et al. 2011; Gortázar et al. 2016; Lange & 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).

6) Final remarks

Understanding the approaches' strengths and weaknesses is crucial in comprehending their potential for studying pathogens in wildlife, considering the pathogen characteristics (emergent, endemic, direct or indirect transmission, etc.) as well as the scale and objectives of the study (Tompkins et al. 2011; McCallum 2016). Several methods, including disease mapping, cluster analysis, risk factor analysis, and molecular analysis, can provide valuable information for all types of pathogens, regardless of scale. However, as complexity increases, the focus shifts to aspects whose epidemiological relevance may vary.

Population-level SIR analyses can be applied at different scales, but their usefulness in endemic pathogens may be limited as they primarily deal with pathogen spread (Heesterbeek & Roberts 1995; White et al. 2018). The sociological origin of network analysis leads this approach to be focused on the behavior and social structure of hosts (Craft 2015). This approach can be highly explanatory for understanding both the maintenance of an endemic pathogen and the spread of an emerging pathogen (Bohm et al. 2009; Springer et al. 2017). However, it has limited applicability to vector-borne diseases, where the interaction is determined by the vectors (although it can be improved by incorporating molecular data; see Nava-Doctor et al. 2021). Furthermore, although network analysis can be conducted on a large scale, its scope is primarily local, where social structure and behavior play a more significant role (Silk et al. 2017a).

On the other hand, ENMs and SDMs are effective at large scales, but they focus on the influence of the environment on transmission. This, along with the challenge of incorporating biotic interactions and social traits, limits their applicability for directly transmitted pathogens and at local scales (Peterson 2008; Escobar 2020). The development of these models for unveiling species distribution ranges highlights their potential for studying emerging pathogens (Sutherst 2013). However, they may not be as useful for studying endemic pathogens with a known distribution.

In contrast, ABMs show great potential the testing of control measures, being applicable to both emerging and endemic pathogens (McCallum 2016; Belsare et al. 2020b). However, ABM, which shares the sociological origin with network analysis, emphasizes the role of interactions in model dynamics, both among agents and between agents and their environment (Macy & Willer 2002; Marion et al. 2008). The interactions

with the environment can be challenging to be implemented (Grimm et al. 2005; Lane-deGraaf et al. 2013), and their potential impact on large-scale dynamics makes these models more suitable for local scales.

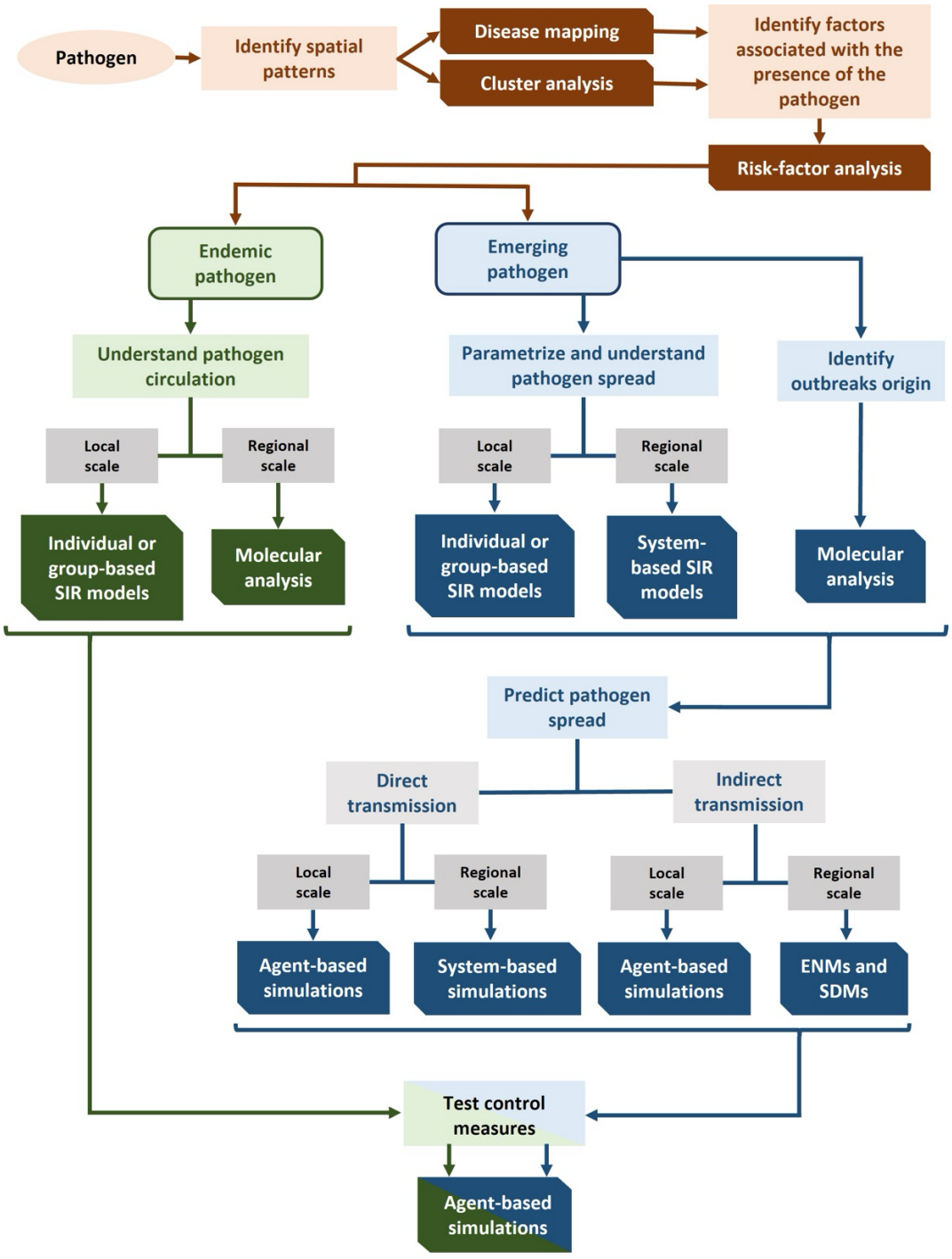

Figure 4. A workflow for studying emerging and endemic pathogens in wildlife from a spatial perspective to inform policy making. The initial steps require few input data and provide valuable information for designing measures and guiding subsequent steps. In the case of endemic pathogens, the following 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.

Consequently, it is essential to identify an appropriate approach for each case and study objective (Figure 6). In certain instances, the optimal solution may arise from the integration of diverse methodologies, for which it is crucial to develop interdisciplinary metamodels that facilitate a comprehensive statistical approach (Alexander et al. 2012; Lacy et al. 2013). This review highlights that spatial epidemiology in wildlife goes far beyond ecology and epidemiology, suggesting that ecologists and epidemiologists should not work in isolation, but rather seek collaboration with statisticians, sociologists, anthropologists, geneticists, and other relevant disciplines to identify and develop effective methodologies for studying wildlife diseases (Restif et al. 2012). However, as methodologies become more sophisticated, so do knowledge barriers between disciplines, increasing the risk of misapplication. Researchers must not only collaborate across fields but also understand the assumptions and language of each discipline. Training, funding, and publication practices should support this convergence. By recognizing the epistemological roots of each tool and integrating them thoughtfully, the field can build robust, realistic, and truly interdisciplinary frameworks to inform conservation, disease control, and policy.

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