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PERSPECTIVE

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Defining an epidemiological landscape that connects movement ecology to pathogen transmission and pace-of-life

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Abstract

Pathogen transmission depends on host density, mobility and contact. These components emerge from host and pathogen movements that themselves arise through interactions with the surrounding environment. The environment, the emergent host and pathogen movements, and the subsequent patterns of density, mobility and contact form an 'epidemiological landscape' connecting the environment to specific locations where transmissions occur. Conventionally, the epidemiological landscape has been described in terms of the geographical coordinates where hosts or pathogens are located. We advocate for an alternative approach that relates those locations to attributes of the local environment. Environmental descriptions can strengthen epidemiological forecasts by allowing for predictions even when local geographical data are not available. Environmental predictions are more accessible than ever thanks to new tools from movement ecology, and we introduce a 'movement-pathogen pace of life' heuristic to help identify aspects of movement that have the most influence on spatial epidemiology. By linking pathogen transmission directly to the environment, the epidemiological landscape offers an efficient path for using environmental information to inform models describing when and where transmission will occur.

KEYWORDS
canonical activity mode, epidemiological landscape, host contact, host mobility, mining-modelling approach, movement mechanisms, movement-pathogen pace-of-life hypothesis, multipartite networks, spatial disease dynamics, transmission hotspot

INTRODUCTION

Environment—the spatially explicit biotic and abiotic context surrounding a host or pathogen at a particular point in time—shapes host movements and pathogen persistence, ultimately resulting in more transmission in some locations than others. The environment's role emerges through movements of the host and pathogen: wildlife pathogens are distributed according to environmental attributes that impede or attract the movements of their hosts (Biek et al., 2007; Hill et al., 2016; Merkle et al., 2018); livestock pathogens can jump between locations when hosts are transported from farm to feedlot (Kao et al., 2007; Mannelli et al., 2007) and human pathogens can follow spatial patterns tied to work or social engagements (Zhang et al., 2020). Environment and movement can also shape disease management (Manlove et al., 2019), especially if super-spreading events, invading epidemic 'waves', or local transmission rates are linked to particular environmental features (Cross et al., 2015; Grenfell et al., 2001; Lloyd-Smith et al., 2005; respectively).

Movement informs spatially explicit models of transmission at multiple scales. At a broad scale, resource limitations and productivity gradients can shape general patterns of host density and mobility (Bischof et al., 2012; Teitelbaum & Mueller, 2019). At a fine scale, spatial dependence among transmission events suggests that transmission aligns with high-resolution drivers of contact (Albery et al., 2022). Despite these connections, tools from movement ecology are rarely used to link environment and transmission mechanistically (Albery et al., 2021; Dougherty et al., 2018). This could be because (1) the tools' outputs do not directly match the parameters that govern pathogen transmission; and (2) potentially important processes like spatially explicit transmitting behaviours or variable pathogen decay rates are often overlooked. Leveraging movement information to understand how the environment influences spatial patterns of transmission requires a stronger integration of movement and disease ecology.

Movement and disease ecology have historically centered around fundamentally different relationships. Movement ecology has primarily investigated interactions between individual animals and their physical environments (Nathan et al., 2008), whereas disease ecology has emphasised the temporal dynamics of pathogen transmission across entire populations (Anderson & May, 1979; Kermack et al., 1927; May & Anderson, 1979). Scaling individual-environment interactions up to predict population- and landscape-scale transmission is a long-standing challenge symptomatic of a broader interdisciplinary divide: epidemiological models lack a systematic way to incorporate insights from movement ecology while retaining tractability and transferability beyond the focal environment. Although movement-disease links are being forged in silico (e.g. Faust et al., 2018; White et al., 2018), empirical integration remains limited, hindering our general understanding about how the environment shapes spatial patterns of pathogen transmission.

Spatial transmission dynamics depend on three central components: host density, which describes where hosts are located across the landscape (Box 1); mobility, which describes residency times and site-to-site movements (Box 1) and contact, which describes the frequency, duration and form of host–host and host–pathogen interactions and how those interactions relate to pathogen transmission rates (Box 1). Together, environmentally informed host densities, mobilities and contacts combine with pathogen life history to form an epidemiological landscape (Box 1) containing the set of paths by which a pathogen could travel across a landscape infecting hosts (Figure 1).

Density, mobility and contact are already included in most spatially explicit models of transmission (Box 2). For example in disease metapopulation models (Finkenstädt & Grenfell, 1998; Grenfell & Bolker, 1998; Sattenspiel & Dietz, 1995), the per capita infection rate (λ_i) can be written as: $\lambda_i = \left(\sum_j Y_j \left(\frac{1}{N_i^{\theta}}\right)\rho_{ij}\right)c$, where j indexes neighbourhood sites including the focal site i and Y_j is the number of infected individuals currently present at site j. Local host densities are contained in N_i, site-to-site connectivities and mobilities are subsumed into ρ_{ij}, transmission rates are related to local densities through θ and local rates of transmission-appropriate contacts are contained in c (parallel deconstructions of partial differential equations and other widely used spatially explicit disease models are outlined in Box 2). Modellers usually treat density, mobility and contact as constant user-specified inputs (e.g. Durrant et al., 2021; Ramiadantsoa et al., 2021; Swinton et al., 1998), but in reality, these entities update continuously according to environmental and social contexts. The epidemiological landscape view differs from conventional spatially explicit disease models by acknowledging the dynamism of real-world systems and linking spatial patterns of transmission directly to the environment through a lens of host and pathogen movements (Figure 1). A strong environmental grounding allows transmission predictions to shift when environmental or social conditions change.

Here, we describe how to specify and use the epidemiological landscape in practice. We: (1) dissect the epidemiological landscape to identify the pathways by which environmentally motivated movements shape spatially explicit disease dynamics (Figure 1); (2) propose the movement-pathogen pace-of-life hypothesis to help prioritise spatially explicit transmission analyses (Figure 2); (3) identify mechanisms that shape the epidemiological landscape (Figure 1; Table 1) and

BOX 1 Glossary

Contact: Ephemeral interaction events between hosts and hosts or hosts and pathogens that lead to transmission. These could vary in frequency, duration and form; and can have a variety of relationships with host density.

Environment-driven K-selected pathogens: Pathogens with prolonged environmental persistence but limited infection time within a host, that have slow first passage times in the pure-environment PCAM.

Environmental metric: Movement description relating spatial locations to features of the local environment.

Epidemiological landscape: The ensemble of environmentally informed host densities, mobilities and contacts that combine with pathogen life-history to form the set of paths by which a pathogen could travel across a landscape infecting hosts.

First passage time: The expected waiting time from arrival in one state (here, arrival of a pathogen in a new host or environmental reservoir) to arrival in another state. First passage time depends on both survival within the PCAM and transmission rate from the PCAM.

Geographical metric: Movement description based solely on locational information describing where an organism spends its time.

Host-as-environment PCAM: Pathogen movements in accordance with the movements of the primary host while infectious.

Host density: Concentration of hosts across the landscape.

Infection-and-environment-driven K-selected pathogens: Pathogens exhibiting long infections and prolonged environmental persistence that have slow first passage times in both the pure-environment and the host-as-environment PCAMs.

Infection-driven K-selected pathogens: Pathogens generating long infections, but with limited survival outside the host, that have slow first passage times in the host-as-environment PCAM.

Mobility: Residency times and probability or rate of engagement in site-to-site movements by hosts or pathogens across an environment.

Movement-pathogen pace-of-life hypothesis: Expectation that the epidemiological landscape component (density, mobility or contact) most important for shaping spatial patterns of pathogen transmission is determined by the pathogen's first passage time in both the pure-environment and in the host-as-environment PCAM.

Movement trajectory: The spatiotemporally explicit path an organism takes across a landscape, often described through step lengths and turning angles.

Pace-of-life syndrome hypothesis: Expectation that traits like growth rate, age of reproductive maturity and longevity should be correlated within a species, so that some species follow 'slower' *K*-selected lifestyles, while others follow *r*-selected 'live fast, die young' strategies.

Pathogen canonical activity mode (PCAM): Segments of pathogen movement trajectories corresponding to different facets of pathogen life history.

Pathogen fertility: The rate at which a pathogen produces new cases or colonies per unit time. In disease ecology fertility contributes, and is sometimes identical, to force of infection.

Pathogen reproductive window: The time over which the pathogen can generate cases before going locally extinct. In disease ecology, this is referred to as the pathogen's infectious period or the pathogen's environmental persistence period, depending on PCAM.

Pure-environment PCAM: Pathogen movements that occur in accordance with the external environment, vectors or intermediate hosts while outside the primary host.

r-selected pathogens: Pathogens engaging in a 'live fast, die young' strategy consisting of lower survival times, higher transition rates and higher first passage times among PCAMs.

Transferable models: Models that retain predictive accuracy when extrapolated to novel contexts.

match the epidemiological landscape's components and mechanisms to emerging tools from movement ecology (Table 2; Supplementary Text) and (4) outline three strategies for blending mechanistic insights with movement and transmission data to generate epidemiological forecasts that are transferable across landscapes and populations (Figure 3). We end by identifying a few open challenges.

A REVISED VIEW OF SPATIALLY EXPLICIT PATHOGEN TRANSMISSION

Transmission occurs at intersections of host and pathogen movement trajectories

Pathogen transmission only occurs if an uninfected host crosses paths with an infected host or pathogen

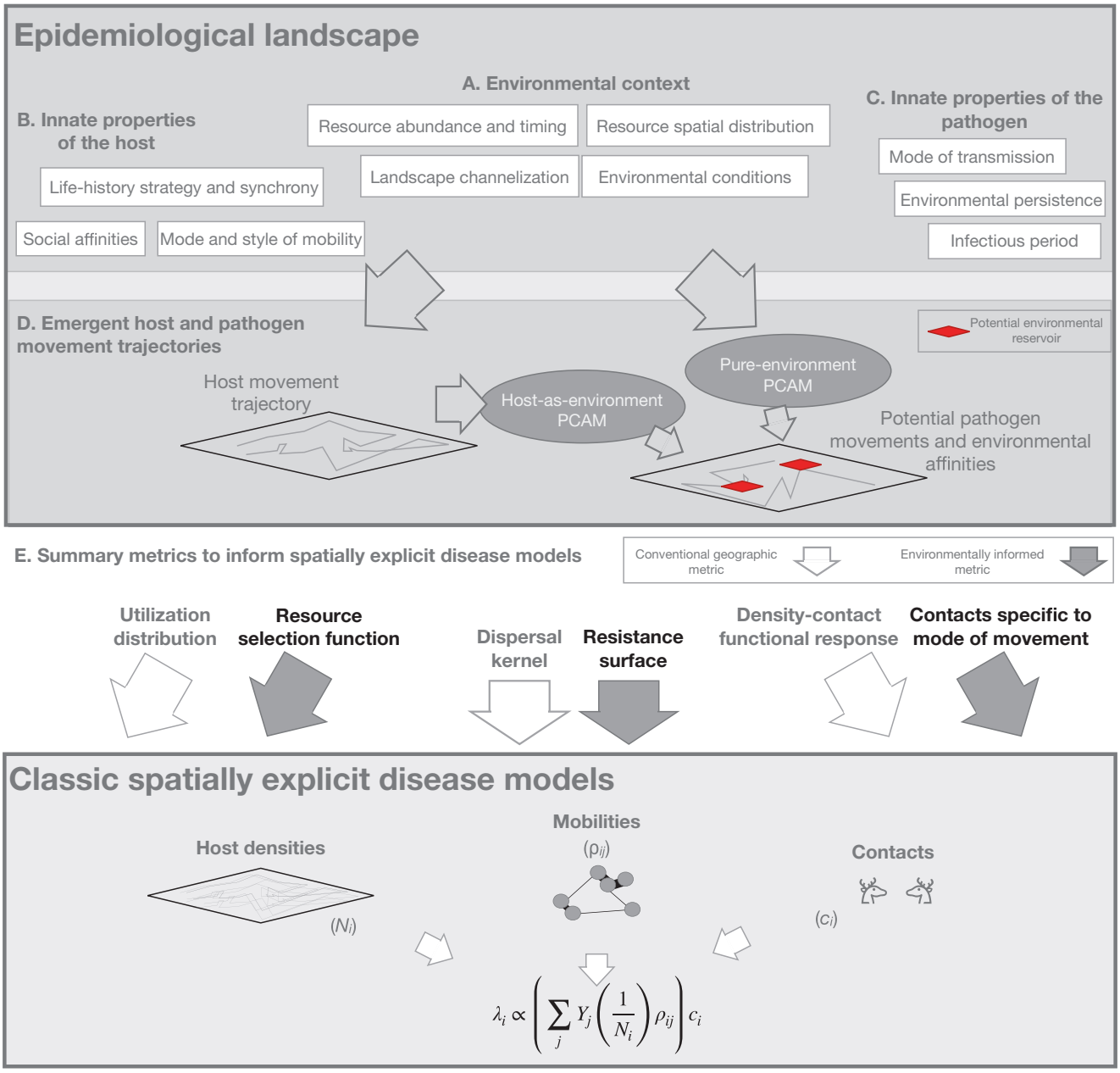

FIGURE 1 The epidemiological landscape can inform classic spatially explicit disease models. The epidemiological landscape (dark grey box; (a)–(d) with select mechanisms in white boxes defined and summarised in Table 1) consists of intrinsic attributes (a–c) and emergent interactions between the environment, hosts and pathogens that shape host and pathogen movements (decomposed into pathogen canonical activity modes, or PCAMs; (d) and locations of pathogen transmission. Information garnered from those movement trajectories can be used to inform inputs to classic spatially explicit disease models (light grey box). Conventionally, spatially explicit disease models relied on summary metrics that simply described host and pathogen locations, and did not link those locations to environmental attributes (white arrows in (e)). Movement ecologists are developing environmentally informed metrics (dark grey arrows in (e); Table 2) that could be used to adapt the classic modelling structures to changing environmental contexts, bringing the epidemiological landscape framework to full reality. λ_i is the per capita rate of infection at location i and Y_j is the density of infected hosts at location j.

(Figure 1d; Manlove et al., 2018; Wilber et al., 2022). At coarse scales, transmission rates should be proportional to the product of the susceptible host density and the pathogen intensity at the focal location (the 'S times I' term governing transmission in conventional susceptible-infected-recovered [SIR] disease models), appropriately rescaled by local host densities depending on whether transmission is frequency- or density-dependent. Host densities and pathogen intensities depend on the cumulative movements of all local hosts and pathogens, however, and movements are informed by an ensemble of environmental and social processes (Figure 1a–c). Clarifying how environment structures movement could inform models of host density, pathogen intensity and subsequent transmission.

BOX 2 Spatially explicit models of pathogen transmission

Partial Differential Equations (PDEs) describe the size of the infected class over continuous space and time. Their *host density* models are continuous, and often homogeneous (but see Garlick et al., 2011; Hefley et al., 2017). The *mobility* model[1] is a spatial diffusion rate and a corresponding functional form. *Outputs* include existence, structure and speed of travelling epidemic waves and spatially explicit times to epidemic peak. *Assumptions*: animals move according to the kernel, which is often isotropic and independent of environment; transmission occurs locally.

Semi-spatial and static network models allow pairwise interactions within local neighbourhoods. The *host density* model is implicit but relies on discrete units with corresponding disease states. The *mobility* model is defined through pairwise coupling coefficients between the 'locations', along with a specified 'neighbourhood' with which each location interacts. *Outputs* are usually derived from a master equation or simulation. *Assumptions*: known network structure and disease status; a priori definition of 'neighbourhood' (depending on analytical approach).

Metapopulation models track disease dynamics at physical locations coupled with one another across space. The *host density* and *mobility* models mirror those of semi-spatial models, but locations are spatially explicit, and mobility can include explicit functions of geographical distance. *Outputs* include spatial spreading rate, spatial synchrony among subunits and individual- and patch-level reproductive numbers. *Assumptions*: a priori knowledge of system connectivity.

IBMs allow movement and transmission to emerge organically from predefined rules applied to a set of actors. Inputs are individual-level attributes and parameters that govern them. The *host density* model can be continuous or discrete. The *mobility* model usually allows an individual's internal state and environment to interact through a set of movement rules. *Outputs* range from a simple wave front of disease spread to each individual's spatiotemporally explicit contribution to reproductive numbers. *Assumptions*: depend on model specifics.

Spatially embedded social networks describe disease dynamics across multipartite networks whose nodes correspond explicitly to locations in space. *Inputs* are bipartite networks linking individuals to different kinds of locations (households, peer groups, etc.), The *host density* model is a set of spatial centroids from each group, and *mobility* models can be distance-, gravity- or radiation-based. *Outputs* include estimates of R_0, total epidemic size and spatial and temporal patterns of transmission. *Assumptions*: constant connectivities; central-place space use patterns.

[1]The *contact process* is often subsumed into a constant transmission rate or absorbed into the *mobility model*.

An organism's movement trajectory is the temporally explicit route that it takes across a landscape (Box 1). Behavioural patterns within movement trajectories have been extensively studied in movement ecology (Abrahms et al., 2017; Edelhoff et al., 2016; Fleming et al., 2014; Getz & Saltz, 2008), where distinct movement motifs—for example foraging or resting—are referred to as 'canonical activity modes' or CAMs (Getz & Saltz, 2008). Pathogen movement trajectories have received less attention, but the CAM concept (which we refer to as the Pathogen's Canonical Activity Mode, or 'PCAM'; Box 1) still applies. Pathogens moving in accordance with the external environment while outside the primary host are in a 'pure-environment' PCAM (Box 1), and pathogens moving in accordance with the movements of the primary host while infectious are in a 'host-as-environment' PCAM (Box 1; Figure 1d). Vector-borne pathogens or pathogens with intermediate hosts may have additional PCAMs corresponding to each life-history phase, though for simplicity we emphasise the pure-environment and host-as-environment PCAMs here. The pathogen's movement and persistence are determined by the movement patterns, duration and ordering of its PCAMs, and could be described using the same hidden Markov modelling approaches as in behavioural and movement ecology; Edelhoff et al., 2016). PCAM duration and ordering themselves depend on two pathogen life-history attributes within each PCAM: (1) the pathogen's ability to produce new cases or colonies per unit time (its 'fertility'; Box 1); and (2) and the time over which the pathogen can generate cases before going locally extinct (its 'reproductive window'; Box 1).

A high-resolution movement trajectory view is not always necessary in spatial epidemiology, and simpler approaches can achieve many epidemiological aims (Figure 1e). For example Hendra virus spillover from flying foxes to horses is limited to locations where the virus and both hosts co-occur. Co-occurrence, and therefore spillover, is concentrated in horse paddocks with fruit trees where flying foxes roost (Plowright et al., 2015), so

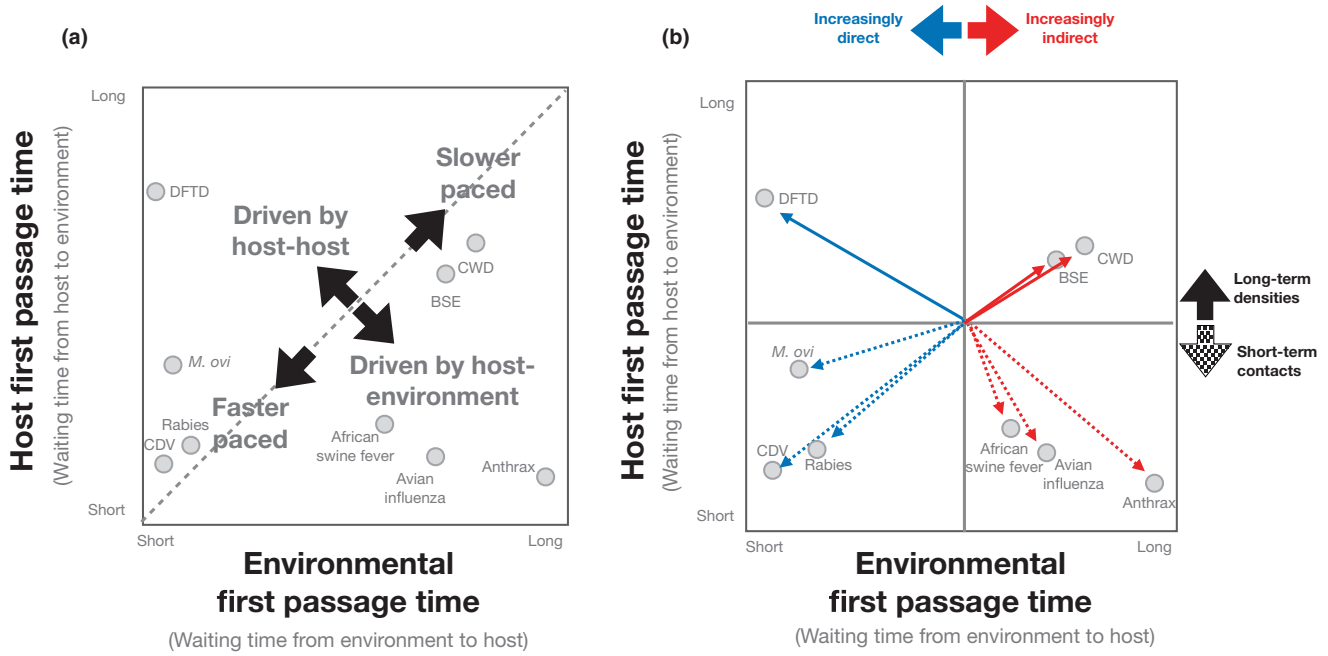

FIGURE 2 The movement-pathogen pace of life hypothesis and expectations about spatial patterns of transmission. (a) Which components of the epidemiological landscape dominate spatial patterns of pathogen transmission depends on the interface between movement and pathogen life history. Pathogen canonical activity movements (PCAMs) can be broken into pure-environment and host-as-environment modes, and the duration and ordering of these modes determines pathogen distribution across the landscape. Duration and ordering of PCAMs are in turn determined by two pathogen life-history traits: first passage times in the host and in the environment. Pathogen pace-of-life increases down the dashed diagonal line, with the fastest pathogens in the lower left-hand corner exhibiting rapid first passage times in both the host and the pathogen. Approximate locations of several pathogens are shown for orientation (*M. ovi* refers to *Mycoplasma ovipneumoniae*, an infectious pathogen of bighorn sheep). Spatial patterns of transmission for pathogens in the upper triangle will be dominated by the locations of host–host interactions, while spatial patterns of transmission for pathogens in the lower triangle will be dominated by interactions between the host and environmental reservoirs. (b) Pathogen life-histories can be summarised through vectors defined by host and environment first-passage times. The further vectors point to the left, the more transmission is driven by direct contacts; vectors extending further to the right are driven by indirect contacts. Pathogens whose vectors extend further to the bottom are expected to show transmission patterns driven by short-term contacts (mass aggregations; mass blooms), while pathogens whose vectors extend further towards the top will be driven by long-term patterns of host space use and density.

reasonable spatially explicit spillover predictions can be built from locations of paddocks with roost trees; individual-level movement analyses may have little to offer. By contrast, transmission of chronic wasting disease (CWD) in mule deer does not have a known environmental signature at coarse spatial scales (beyond higher risks associated with clay-heavy soils; Miller et al., 2004). However, there is a still a possibility that management could be applied at a finer scale if there are particular environmental signatures associated with precisely where mule deer shed prions, and how those shedding locations align with local soil types. Therefore, high-resolution movement analyses might be able to improve spatially explicit transmission forecasts in the CWD system (Box 1).

The Hendra virus and CWD examples highlight different spatial scales at which disease models can operate. At the individual movement trajectory level, host, environment and pathogen life-history mechanisms can inform when and where hosts and pathogens interact (as in the CWD example above). At the population level, densities, mobilities and contacts derived from all local pathogens and hosts contribute to an aggregate force of infection that can sometimes be linked to the environment directly using coarse scale data (as in the Hendra example). Both scales can be used to predict spatially explicit transmission patterns, and either may be reasonable depending on system dynamics.

An epidemiological landscape framework advances epidemiological modelling by mechanising the environment—movement—transmission relationship

Movements can be described in terms of either location or environment (referred to as 'geographic' and 'environmental space' in the movement ecology literature; Box 1 [Matthiopoulos et al., 2020; Moorcroft et al., 2006]). Metrics in geographical space are built from raw coordinates (e.g. whether an individual occupies a specific point on the landscape or moves a particular distance; Box 1), while metrics in environmental space relate those coordinates to local environmental features (e.g. whether an individual selects for cliffy habitats or moves rapidly near topographical bottlenecks; Box 1). Environmental metrics are mechanistic in that they capture how

environmental attributes alter patterns of movement. Metrics in both categories cover temporal scales from fine and behaviorally relevant to coarse and occupancy-relevant. Classic spatially explicit transmission models (Box 2) often rely on metrics in geographical space (e.g. subpopulations defined through overlapping home ranges [Craft et al., 2011; O'Brien et al., 2014]; Euclidean distance- or gravity-based descriptions of mobility or connectivity [Viboud et al., 2006]), and assume that the functional responses linking host density and per capita transmission rates are constant across environments.

Although some geographical metrics can be moved to new spatial domains (e.g. dispersal kernels), geographical metrics contain no information about the environment where the coordinates arose, so they cannot account for specific attributes of new environments. Therefore, applying geographical metrics in novel environments requires extrapolation in both geographical and environmental space. Environmental metrics are sometimes less spatially resolved than geographical metrics, but applying environmental metrics in novel settings only requires extrapolation in geographical space.

Transferable models are models that perform well when extrapolated to novel contexts (Box 1; Barbosa et al., 2009; Matthiopoulos et al., 2019). Transferable models are especially important for epidemiological systems where: (1) spillover could occur across a huge geographical range, making boundary controls infeasible (e.g. avian influenza spillovers from migratory waterfowl could occur at many points along a flyway; Hill et al., 2016); (2) management actions shift depending on the local environment (e.g. epizootic hemorrhagic disease virus [EHDV] management might prioritise water point sources for vector control when water is scarce, and host vaccinations when water is plentiful; Noronha et al., 2021); or (3) research is concentrated around pseudo-model systems but findings need to extend to a wider set of host-pathogen interactions (e.g. fine-scale but limited data describing life-history movement trade-offs in flying foxes could be used to generate broader-scale spatial predictions; Hayman et al., 2018). Models that describe density, mobility and contact in terms of the local environment are transferable to new locations, while models based on geographical metrics are not. Therefore, the epidemiological landscape approach relies on environmental metrics wherever possible.

Epidemiological landscape components can be prioritised according to the movement-pathogen pace-of-life hypothesis

Most methods for generating spatially explicit epidemiological forecasts cover only one component of the epidemiological landscape (densities or mobilities or contacts, but rarely all three). Which component has the most influence on spatial patterns of pathogen transmission depends on the interface between movement and pathogen life-history traits.

The pace-of-life syndrome hypothesis (Ricklefs & Wikelski, 2002; Box 1) proposes that traits like growth rate, age of reproductive maturity, and longevity should be correlated within a species, leading some species to follow 'slower' *K*-selected lifestyles, while others follow *r*-selected 'live fast, die young' strategies. For pathogens, pace-of-life reflects a trade-off between ability to colonise new hosts (i.e. to infect) and ability to survive. Traits associated with *r*- and *K*-selection have been extensively studied (e.g. Oli & Dobson, 2003; Thrall & Burdon, 1997), particularly in plant pathogens and sometimes with an explicit eye towards spatial spread (Eshelman et al., 2010; Susi & Laine, 2013; van Dijk et al., 2022). Assigning 'pace-of-life' to pathogens is complicated, however, because pathogens spend their lives switching among environments (the pure-environment while outside the host and the host-as-environment while infecting; i.e. the PCAMs from Section 'Transmission occurs at intersections of host and pathogen movement trajectories'). Pathogen longevity (here considered at the scale of an infecting colony) is determined by survival within each environment, along with transition rates among environments.

r-selected pathogens have high fertility rates (they are able to produce many new cases per unit time; Box 1) and short reproductive windows (they have limited time to produce new cases before dying out in the local host or environment; Box 1). These two attributes produce short first passage times (i.e. expected waiting time from arrival in one state to arrival in the next; Box 1) through both hosts and environments: *r*-selected pathogens move quickly from host-as-environment to pure-environment and back.

K-selected pathogens have low fertility rates (they are limited in how many cases they can generate per unit time) and long reproductive windows (they have more time over which to generate those cases). Long reproductive windows and lower fertilities correspond to longer first passage times, which can occur in the pure environment, the host-as-environment or both. Which environment has the longest first passage time has implications for how the pathogen distributes over space. Therefore, for clarity, we partition *K*-selected pathogens into three groups: infection-driven *K*-selected pathogens (Box 1); environment-driven *K*-selected pathogens (Box 1) and infection-and-environment-driven *K*-selected pathogens (Box 1). Four example systems—canine distemper virus, devil facial-tumour disease, chronic wasting disease and *Bacillus anthracis*—illustrate how these life-history strategies interact with movement to generate pathogen intensity patterns over space.

Canine distemper virus (CDV; a close relative of measles that infects carnivores; Terio & Craft, 2013), is an *r*-selected pathogen with high reproductive

TABLE 1 Mechanisms linking movement to the epidemiological landscape's central processes. 'Group' indicates whether the mechanism is derived through the pathogen ('P'), the environment ('E') or the host ('H')

Group	Mechanism	Description of effect on epidemiological landscape	Central component(s)	Examples of altered spatial transmission properties	Example reference
P	Mode of transmission	How a pathogen moves from host to host (e.g. whether transmission occurs through respiratory, faecal-oral, sexual or vertical routes). Connects local host density to the rate of potentially transmitting contacts. In general, pathogens whose acquisition and deposition processes are decoupled from host density should exhibit relatively constant prevalence rates across space, while pathogens with acquisition and deposition processes that vary with density should display more explosive dynamics and be more prone to super-spreading events or locations	Contact	Propensity for superspreading events[b], potential for persistence	Diallo et al., 2016; Borremans et al., 2019
P	Environmental persistence time outside the host	Duration of time a pathogen can survive and remain infectious outside a host's body. Transmission of pathogens that can survive for long periods of time outside the host should be more uniform over space, with enhanced potential for persistence. If all transmission is indirect, then pathogens with long environmental survival may also be prone towards slower growth rates. These factors should stabilise epidemic dynamics, leading to consistent trends in transmission rates across space and time (perhaps with seasonality induced through the host's or pathogen's life histories)	Contact	Rate of spread, pathogen persistence	Rohani et al., 2009
P	Behavioural or physiological manipulation of the host	Pathogen-induced alterations to host movement and contact patterns. Behavioural manipulation of the host often leads to elevated contact rates which in turn accelerate transmission and overall disease dynamics. Physiological manipulation might be expected to slow transmission by reducing local crowding around infected hosts through sickness behaviour, but this depends on whether and how the manipulation alters rates of potentially transmitting contacts. Physiological manipulation may also lead infected hosts to concentrate at specific resources, shifting the environments where transmission occurs	Contact, mobility	Rate of spatial spread, potential for pathogen persistence and location and timing of transmission hotspots	Berdoy et al., 2000
P	Infectious period	Duration of time during which the pathogen inhabits and is shed by the host	Density, contact	Rate of spatial spread, potential for persistence	Plowright et al., 2017
E	Resource abundance and timing	Density and timing of resources available to the host within its range. Resource availability should generally increase host densities, improving pathogen persistence as host numbers exceed the critical community size	Density	Rate of spatial spread, spatial synchrony and potential for seasonal cycling	Plowright et al., 2011
E	Resource spatial distribution	Configuration of resources through space within a host's range. Overdispersed resource distributions (where resources are concentrated at just a few sites) facilitate host aggregation, may concentrate movement pathways (i.e. create corridors), and are expected to accelerate transmission when transmission is density-dependent	Density, mobility[a]	Propensity for super-spreading events, location of endemic transmission hotspots	Park et al., 2002; Becker et al., 2015
E	Landscape channelisation and natural air or water currents	Extent to which landscape limits routes hosts can use to move over space. Landscape channelisation or natural currents should facilitate aggregation of hosts and pathogens at particular sites and steer them away from others. Depending on the relative rates of host population turnover, movement and infection, this may either facilitate or impede persistence	Contact, mobility, density[a]	Spread rate, spatial synchrony and locations of endemic hotspots	Bertuzzo et al., 2010
E	Environmental conditions	Aspects of the environment like temperature, precipitation or soil features that alter pathogen survival or movement outside the host. Variation in pathogen survival across abiotic and biotic conditions (e.g. water temperature is in avian influenza; soil type as in chronic wasting disease) can stabilise pathogen persistence through the establishment of reservoir locations with reservoir disruption expediting fade-out	Contact	Expected persistence time, location of endemic transmission hotspots and potential for spatial trapping	Weiss & Dishon, 1971

TABLE 1 (Continued)

Group	Mechanism	Description of effect on epidemiological landscape	Central component(s)	Examples of altered spatial transmission properties	Example reference
H	Life-history strategy and synchrony	Extent to which the host's migratory, mating or reproductive seasons are concentrated in particular parts of the year. Synchronous life-history events (e.g. pulsed births or synchronous patterns of host immune function) can synchronise disease dynamics across space. If synchrony is strong, this can increase fade-out rates by forcing a temporal 'trough' that the pathogen must survive between pulses of new, or newly susceptible, hosts	Density	Magnitude of temporal fluctuations and potential for stochastic fade-out	Peel et al., 2014; Cassirer et al., 2013; Mariën et al., 2020
H	Social affinity: Propensity to form mass gatherings	Extent to which the host forms large groups due to life history or social mediation. Mass gatherings increase local densities and potential for transmitting contacts, increasing potential for super-spreading events that can accelerate epidemic growth and spread	Contact, mobility, density affected if gatherings persist	Potential for super-spreading events[b]	Cross et al., 2005; Lloyd-Smith et al., 2005
H	Social affinity: Propensity for stable social bonds and host sociality	How stable and longstanding social interactions are between dyads and groups within a population. Can range from high stability (i.e. a strong propensity for stable social ties) to fission-fusion dynamics where groups are constantly forming and reforming	Contact, mobility, density	Rate of spread, potential for social trapping	Sah et al., 2017; Webber & Willis, 2020
H	Systematically varying engagement in transmitting behaviours	Do hosts selectively engage in pathogen-transmitting behaviours in certain environments but not in others? Also, does engagement in transmitting behaviour vary systematically among hosts according to intrinsic attributes like age, sex or dominance rank?	Contact	Location and timing of hotspots	Sih et al., 2018
H	Style of mobility: Time invested in random searches	Extent to which the host engages in directed as opposed to randomly searching movements. Non-random movement patterns re-enforce connectivities among particular sites and lead animals to concentrate their time at particular locations. However, whether non-random movements concentrate or decentralise spatial transmission risk depends on whether non-random routes are shared or partitioned across hosts	Mobility	Potential for spatial trapping, spatial synchrony, rate of spatial spread	Weiss & Dishon, 1971 ; Martin-Löf, 1998; Bhamidi et al., 2014
H	Migratory propensity	Whether host engages in a range resident, nomadic or migratory lifestyle, along with the extent of that lifestyle within its population (e.g. is the population completely or partially migratory)	Density, mobility	Potential for migratory release, spatial synchrony	Hall et al., 2014; Altizer et al., 2015; Teitelbaum et al., 2018
H	Cognizance and response to disease risks	Extent to which the host is aware of or adapts its behaviours to the presence of infectious disease. The ability to perceive disease as a threat likely varies across host-pathogen pairings. Information transfer in humans greatly facilitates disease threat responses in some cases, leading to collective shifts (usually decreases) in transmission-related movements and subsequent transmission rates. Responsiveness to disease also extends to a number of non-human animal systems	Contact, mobility	Potential for super-spreading events[b], impact for endemic transmission hotspots, spatial synchrony	Townsend et al., 2020; Stockmaier et al., 2021
H	Mode of mobility: capacity to avoid environmental risks and barriers	Extent to which the environment a host experiences while moving reflects the pathogen risks that the broader environment holds. Motility of some hosts (flight, transit infrastructure) allows them to avoid environmental risks and barriers	Contact, mobility	Persistence time, spatial synchrony, rate of spatial spread	Plowright et al., 2011; Altizer et al., 2015

[a]These processes are mediated by effects on another process; for example density-dependent effects on mobility, or animals piling up at sites due to channelisation during mobility.

[b]Super-spreading events reliably alter the rate of spatial spread.

TABLE 2 Using methods from movement ecology to directly infer central processes of the epidemiological landscape. Select methods for mechanistically or phenomenologically describing the constituent processes. Tactics flagged as Geographic operate primarily in geographical space, whereas Environmental tactics incorporate aspects of the environment measured in environmental space

Component	Tactic	Method	Strengths	Weaknesses	Methods example	Disease modelling example
Host density	Descriptive/ Geographic	Census or mapped spatial data	High spatial resolution; can be treated as discrete instead of continuous	Sensitive to sampling	(n/a)	Riley & Ferguson, 2006; Haw et al., 2020
Host density	Descriptive/ Geographic	Home-range estimates and utilisation distributions	Reasonably sensitive and specific if data coverage are good	Sensitive to sampling; assume good coverage and stationarity. Different estimators have different coverage and area properties. May not be transportable	Numerous; reviewed in Laver & Kelly, 2008; Noonan et al., 2019	O'Brien et al., 2014
Host density	Mechanistic/ Environmental	Habitat selection models	Can be used to infer space use beyond the extent of the instrumented animals	Do not account for appropriate but unoccupied habitat	Numerous, recently reviewed in Fieberg et al., 2021	Scherer et al., 2020
Host density	Mining/ Environmental	Random Forests or other ensemble-learning approaches	Accommodate nonlinear fits; limit requisite a priori specification of covariate structures. Allow for rankings of variable importance	'Black box' method. Fitting is constrained to the domain of the sampled covariates, which can limit transportability	Cutler et al., 2007; Shoemaker et al., 2018	Reynolds et al., 2017
Host density or mobility	Mining	Dynamic Brownian bridges	Account for multiple distinct behavioural states	Assume independent steps, do not account for surrounding environment or density	Kranstauber et al., 2012	Prosser et al., 2016
Host density or mobility	Mechanistic/ Environmental	Master equations	Account for neighbourhood effects on space use	Assume independent steps, do not account for density or other social factors	Moorcroft & Barnett 2008; Potts et al., 2014	Merkle et al., 2018
Host density or mobility	Mechanistic/ Environmental	Least-cost paths/ resistance surfaces	Isotropy does not matter, accounts for landscape structure	Dependent on and sensitive to the underlying habitat selection surface	Hanks & Hooten, 2013; Marrotte & Bowman, 2017	Remais et al., 2010
Host density and mobility	Mining	Path segmentation	Various methods that decompose movement trajectories into contiguous blocks of similar steps	Classifications may be sensitive to the timescale of the data; categories may not directly connect to behaviours	Reviewed in Edelhoff et al., 2016	Garcia Fontes et al., 2021
Mobility	Descriptive/ Geographic	Great-circle distances, Gaussian kernels	Simplicity, analytical tractability, direct links to some formulations of diffusion	Tail of Gaussian kernels can underestimate rare, long-distance moves	e.g. Meyer et al., 2012	Gerbier et al., 2008
Mobility	Descriptive/ Geographic	Estimated (or non-parametric) dispersal kernel	Flexibility to capture deviations from standard parametric dispersal models	Assumes independent steps, no effect of habitat resistance, no effect of density or social structure, (usually) isotropic, depends on available data which can miss rare movements	Many including Skellam, 1951, Kolmogorov, 1937, Clark, 1998	Tsao et al., 2020

TABLE 2 (Continued)

Component	Tactic	Method	Strengths	Weaknesses	Methods example	Disease modelling example
Mobility	Descriptive/ Geographic	Spatial adjacency networks	Geospatial data	Rely on structural, as opposed to functional, connectivity and consequently miss transmission tied to long-distance movement	Kraemer et al., 2019	Davis et al., 2004; Salkeld et al., 2010
Mobility	Descriptive/ Geographic	Movement networks	Does not require habitat surface; directly tied to GPS data	Scope of inference limited to the landscape at hand	Bastille-Rousseau et al., 2018	(none that we are aware of)
Mobility	Descriptive or Mechanistic	Discrete-time CRWs	Can incorporate multiple behavioural states	Discrete time	Morales et al., 2004; Jonsen et al., 2005; McClintock et al., 2012	Morton & Finkenstadt, 2005
Mobility	Descriptive or Mechanistic	Continuous time movement models (CTMMs)	Moves from discrete to continuous time	Depends on particular movement model, but generally do not account for migration or other life histories with multiple sites of intensive use	Fleming et al., 2014; Fleming et al., 2015; Calabrese et al., 2016	Yang et al., 2021
Mobility	Mechanistic/ Environmental	CTMM with state switching	Can account for life-history events like migration or other forms of state-switching	Computationally intensive, and can generate large uncertainty estimates	Hanks et al., 2011	(none that we are aware of)
Mobility	Mechanistic/ Geographic	Gravity model	Accounts for density at each node, and intervening distance	Phenomenological, does not account for landscape resistance, relies on local spatial data	Zipf, 1946, Viboud et al., 2006; Masucci et al., 2013	Riley & Ferguson, 2006; Charu et al., 2017
Mobility	Mechanistic/ Geographic	Radiation model	Captures density at node, density in proximity to node and intervening distance	Relies exclusively on population density, which likely proxies for resource availability in human systems (where the critical resource is jobs)	Simini et al., 2012	Kraemer et al., 2019
Mobility	Mining/ Environmental	Bivariate random forest	Accommodate nonlinear fits; limit requisite a priori specification of covariate structures	Fitting is constrained to the domain of the sampled covariates, which can limit transportability	Wijeyakulasuriya et al., 2020	(none that we are aware of)
Contact	Mechanistic/ Geographic or Environmental	Selection of network generation model based on transmission patterns	Can infer contact structure retrospectively on the basis of manifest transmission events; allows multiple network-generating processes to compete	Assumes true generation mechanism is represented in the set of networks explored. Can require high collaring intensities and disease testing data	Groendyke et al., 2012 (with exponential random graph models)	Grear et al., 2013; Manlove et al., 2017
Contact	Descriptive/ Geographic	Proximity logger-based models of empirical contact	Directly infer contacts on the basis of measured data (that do not rely on direct observation and are thus subject to a different form of sampling variation)	Estimates depend on logger densities, and there are statistical challenges associated with separating individual and dyadic effects	Cross et al., 2013	Aiello et al., 2014
Mobility and contact	Descriptive/ Geographic	Revisitation analysis	Decouples intensity of use from average density	Assumes fixes derived from collared individuals are representative	Bracis et al., 2018	Mysterud et al., 2021

(*continued*)

TABLE 2 (Continued)

Component	Tactic	Method	Strengths	Weaknesses	Methods example	Disease modelling example
Mobility and contact	Descriptive/ Geographic	Networks built from association indices (not home-range overlap)	Availability of appropriate data (e.g. Krause et al., 2013)	Coarse spatial and temporal scales	Farine & Whitehead, 2015; Farine, 2017; Long et al., 2014	Cooper et al., 2010; Drewe, 2010
Mobility and contact	Descriptive/ Geographic	Networks built from continuous-time movement models	Can account for spatiotemporal co-occurrence	Constrained by assumptions of CTMM (no migration etc.)	Martinez-Garcia et al., 2020; Gurarie & Ovaskainen, 2013	Richardson & Gorochowski, 2015; Wilber et al., 2022
Mobility and contact	Descriptive/ Geographic	Networks from connectivity models built on infectious agent molecular data	Track transmission paths through geographical or social space, as opposed to contacts (so, no assumption that the 'contact' definition matches true transmissible contacts)	Can be difficult to parameterise depending on depth of pathogen sequencing and evolutionary dynamics	Hall et al., 2016; Firestone et al., 2019	Biek et al., 2007; Kamath et al., 2016
Mobility and contact	Mining/ Environmental or Geographic	Binary Support Vector Machines, potentially smoothed by hidden Markov support vector machines, Long short-term memory algorithms	Can handle Markovian processes (but circumvents need to directly specify state-transition dynamics). Can handle very large state spaces	'Black box' method. Variable importance may not always be tractable	Martiskainen et al., 2009; Grünewälder et al., 2012; Wijeyakulasuriya et al., 2020; Reviewed in Wang, 2019	(none that we are aware of)
Mobility and contact	Mining/ Environmental or Geographic	Voting ensemble (with 5 base models: gradient boosting, logistic regression, random forests, artificial neural network)	Can produce very high-quality predictions	'Black box' method. Variable importance may not always be tractable	Brewster et al., 2018	(none that we are aware of)

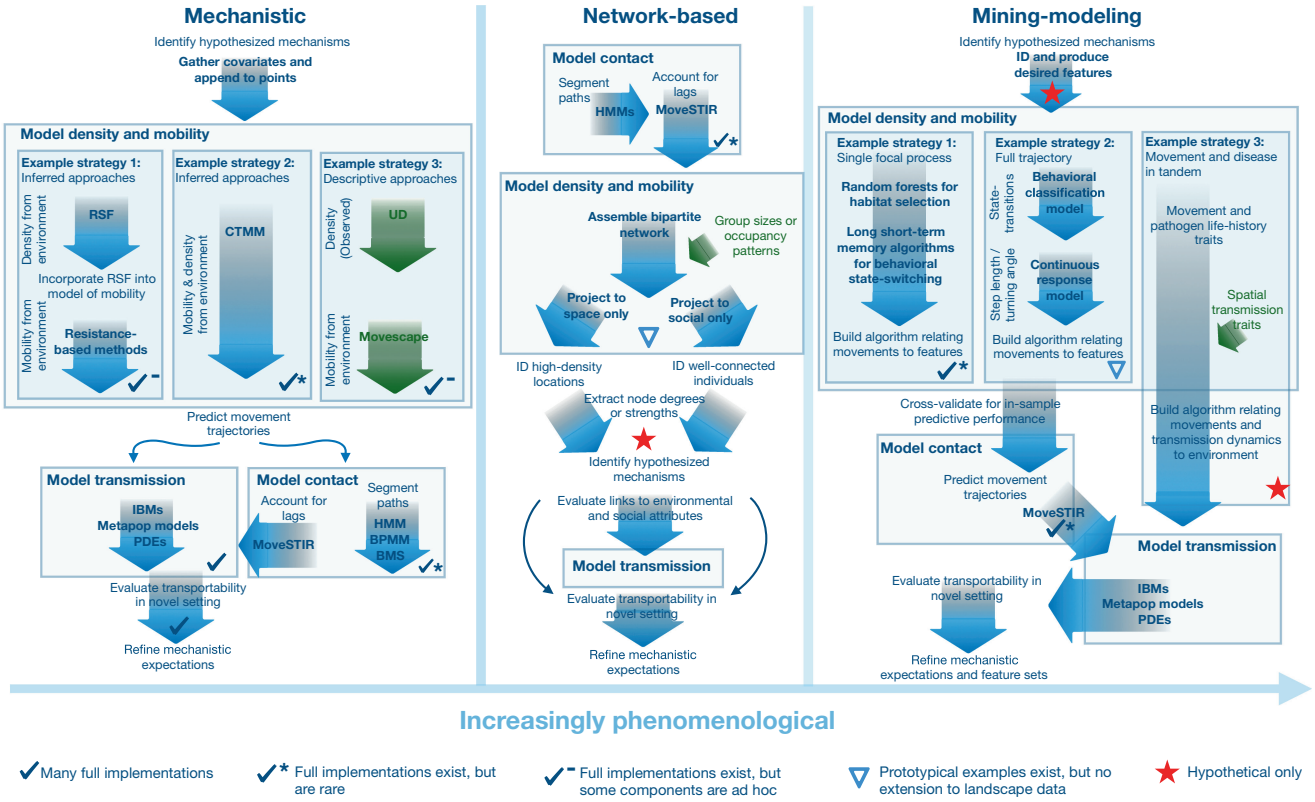

FIGURE 3 Hypothetical workflows using the epidemiological landscape. Each workflow uses animal movement trajectories to forecast movements from environmental covariates, adjusts movement forecasts according to time lags imposed by pathogen life-history, and integrates with disease models (Box 2) to predict spatiotemporal transmission dynamics. Auxiliary data can enter the workflows (examples indicated by steps flagged with dark green arrows), but could limit model transportability. Many of the methods already exist (checkmarks), although it is not always clear how they should be connected. Other methods have been proposed and prototyped, but are as-yet untested on real-word data (blue triangles). A third group remains strictly hypothetical (red stars). 'RSF' = resource selection function; 'SSF' = step selection function; 'CTMM' = continuous time movement model; 'UD' = utilisation distribution; 'CTMC' = continuous time Markov chain. 'MoveSTIR' accounts for temporal lags between pathogen deposition and acquisition (Wilber et al., 2022). Method details are in Table 2.

potential, a short reproductive window in the host-as-environment, and an even-shorter reproductive window in the pure-environment. CDV's infectious periods are longer than its environmental persistence, thus CDV predominantly occupies the host-as-environment PCAM. As a consequence, its densities and mobilities closely match those of its hosts (Almberg et al., 2010; Craft et al., 2011). These traits make CDV most successful (able to produce large outbreaks) when hosts are plentiful, contacts are frequent and host turnover is high (e.g. in ephemeral aggregations of susceptible hosts; Table 1; Supplemental Text S2.1.1).

Devil facial tumour disease (DFTD; a transmissible cancer of Tasmanian devils) exemplifies the infection-driven *K*-selected pathogen group (Pearse & Swift, 2006). DFTD generates long infections (it transitions slowly from host-as-environment to pure-environment), but like CDV, its survival outside the host is brief: cancerous cells must transition quickly from pure-environment to host-as-environment or risk fading out. As a consequence, DFTD spends most of its time in the host-as-environment PCAM, and its density and mobility should be well-approximated by those of its host. However, because DFTD is transmitted through bites (Hamede et al., 2013), it is less equipped than CDV to capitalise on large but short-lived host aggregations for transmission. Instead, DFTD transmission maps to areas where hosts engage in appropriate contact (for DFTD, at locations where hosts mate or fight; Supplemental Text S1.1.2 and S2.1.2), regardless of local host densities. This could be a risky strategy—DFTD's transmission is vulnerable to disruptions in host metapopulation structure (Durrant et al., 2021)—but because DFTD's transmitting behaviours are tied to its host's mating system, transmission opportunities arise at pseudo-regular intervals, limiting dead-end infections and reducing variance in reproductive output.

The prions causing chronic wasting disease (CWD; a prion-driven encephalopathy primarily affecting cervids) exemplify the infection-and-environment-driven *K*-selected pathogen group: CWD's transitions are slow from both host-as-environment to pure-environment and pure-environment to host-as-environment (Miller

et al., 2004; Miller & Conner, 2005). Because CWD can survive for long periods of time in both PCAMs, its density over space should be a convolution of both environment types (its intensities should be elevated around environmental reservoirs, but also around areas of high host densities; Almberg et al., 2011). CWD's mobility is also a mixture of its mobility in the pure-environment and the host-as-environment, but since its movement capacity in the pure-environment is limited, host mobility patterns are the most important determinants of its spread.

Bacillus anthracis (Anthrax) exemplifies the environment-driven *K*-selected pathogen groups: it transitions slowly from pure-environment to host-as-environment, but quickly from host-as-environment to pure-environment. *Bacillus anthracis* concentrates at environmental reservoirs (Weiss & Dishon, 1971), its movements are determined by its mobility in the pure-environment (Turner et al., 2014; Supplemental Text S1.1.3), and its ability to contact hosts depends on host encounters with reservoir environments.

The *movement-pathogen pace-of-life hypothesis* (Box 1) unifies these examples by proposing that the epidemiological landscape component (density, mobility or contact) with the strongest influence on spatial patterns of pathogen transmission is determined by the pathogen's first passage time through both the environment and the host (Lloyd & May, 2001; Figure 2). Classifying pathogens according to their first passage times clarifies expectations about how each epidemiological landscape component influences when and where the pathogen is transmitted. When first passage time in the host is longer than first passage time in the environment (i.e. the pathogen's life-history places it above the dashed diagonal line in Figure 2a), transmission is primarily direct, and concentrates at locations where hosts encounter one another. When first passage time in the environment exceeds first passage time in the host, transmission concentrates at locations where hosts encounter environmental reservoirs. Transmission of 'fast' *r*-selected pathogens concentrates at locations where hosts form large groups (near the bottom and left-hand side of Figure 2a). Transmission of *K*-selected pathogens concentrates at locations with the highest time-averaged densities (for infection-driven *K*-selected pathogens) or reservoir contact rates (for environment-driven *K*-selected pathogens; Figure 2b). The hypothesis assumes that first passage times through both the host and the environment are constant, so environmentally variable pathogen persistence or spatially explicit transmitting behaviours can lead patterns to depart from movement-pathogen pace-of-life expectations. When this occurs, movement-based analyses can be refined and the movement-pathogen pace-of-life hypothesis can operate as a contrasting null.

MOVEMENT MECHANISMS CONNECT HOSTS, PATHOGENS AND ENVIRONMENTS

Specific mechanisms shape how the host, pathogen and environment interact. For example resource selection shapes patterns of host density (Supplemental Text S1.1), thus resource selection functions could be used to predict host densities; and landscape resistance shapes patterns of host mobility, thus modelled resistance surfaces could be used to predict host mobility patterns (McRae et al., 2008). Movement mechanisms could also inform spatially explicit multipartite networks (Manlove et al., 2018; Silk et al., 2018) or models from machine learning (Han et al., 2020; Wijeyakulasuriya et al., 2019) aiming to predict the epidemiological landscape. Metrics like the number of individuals within some neighbourhood of a focal animal or the turning angle required for the host to orient towards a particular environmental feature are commonly used in movement ecology and could also inform spatially explicit models of pathogen transmission.

Some mechanisms can lead systems to depart from movement-pathogen pace-of-life expectations, and several of these mechanisms are already well-understood. Mass aggregations (Cross et al., 2005; Lloyd-Smith et al., 2005) and stable social bonds (Sah et al., 2017) affect patterns of contacts, as do the pathogen's mode of transmission and environmental persistence (Table 1; Supplemental Text S2.1). Synchronous host life-history events (like birth pulses) can produce seasonally pulsed transmission after an influx of new susceptible hosts (Peel et al., 2014). Density, mobility and contact can also vary according to feedbacks between the pathogen and the host (Supplemental Text S1.1.4 and S2.1.4) that arise through either physiological pathways or behavioural shifts. Physiologically, fighting infection might lead to fatigue, causing hosts to move less or self-isolate; and immune functions could change resource requirements (e.g. by increasing water requirements during febrile responses). Behaviorally, neurotropic pathogens like rabies or toxoplasmosis can directly alter host conduct (Hughes et al., 2011; Stockmaier et al., 2021; Weinstein et al., 2018).

Other mechanisms are implicitly embedded in existing frameworks for modelling spatially explicit transmission for humans, livestock and wildlife. In spatially explicit transmission models for humans and livestock pathogens, locations where individuals interact (e.g. houses, transit centers, feedlots) are often assumed to be discrete and fixed through time (Haw et al., 2020; Keeling et al., 2001; Riley & Ferguson, 2006). Site-to-site mobilities depend on intervening distances and local and surrounding host densities (Simini et al., 2012; Tizzoni et al., 2014; Viboud et al., 2006). Once site-to-site movements occur, transmission-appropriate contacts are modelled according to local host density (following a functional form usually

based on a priori knowledge about mode of transmission), and infection rates ultimately depend on the probability of transmission given contact.

These assumptions reflect attributes of human movement ecology that might not hold for other species. Most humans regularly return to home sites (but see Bharti et al., 2011), so assigning humans to fixed locations and movement patterns might not affect spatially explicit transmission predictions. Humans spend little time on random walks (Meekan et al., 2017); instead, they make directed moves from starting points to preordained destinations, with mobilities rarely slowed by environmental barriers (e.g. mountains, rivers; Table 1). Place-of-residence for non-human hosts might be better described through intensity surfaces that shift through time. Non-human host densities and mobilities are often tied to the abundance, quality, timing and spatial distribution of resources, along with the structure of the intervening landscape (Table 1). However, what constitutes a 'resource' depends on the host's ecology (Miller et al., 2019) and its internal state (Nathan et al., 2008; Supplemental Text S1.1.1), and understanding host ecology and physiology are important for generating accurate space use predictions.

In reality, the a priori directed movements of humans might occupy one extreme in host movement decision-making, while movement patterns derived from resource-driven random walks occupy the other. Movement dynamics for most host species probably fall somewhere in-between. Fleshing out these movement continua (Carbone et al., 2005; Han et al., 2015) could inform general expectations about how particular host taxa move, improving spatially explicit transmission predictions especially for understudied species. Biological underpinnings and current integrations between movement and disease ecology, especially with regards to how environment and host social ecology inform patterns of host and pathogen movements, are included in the Supplementary Text.

Next steps

In the immediate-term, disease ecology would benefit from incorporating the following considerations into movement analyses destined for epidemiological frameworks. First, we need to account for spatial and temporal lags between pathogen shedding and acquisition in order to weight pathogen transmission potentials from the pure-environment to the host-as-environment (e.g. Richardson & Gorochowski, 2015; Wilber et al., 2022; Supplemental Text S1.3.1). Second, we need a clearer understanding of how to link the timescales of movement and movement data to the timescales of pathogen transmission (Supplemental Text S1.3.2). Third, we need better frameworks to guide data collection and spatial allocation of tracking devices, especially for studies prioritising contact (Supplemental Text S1.3.3). Fourth, we need to consider whether and how to update movement forecasts dynamically in response to changing epidemiological contexts (Supplemental Text S1.3.4). Fifth, spatial epidemiology might sometimes require entirely new epidemiological theory and methods, which we need to identify and develop (Supplemental Text S1.3.5).

To better incorporate the social environment and quantify socially driven aspects of contact and mobility, we first need methods that can scale up from subsets of tracked individuals to draw inference across entire populations (Supplemental Text S2.3.1). Second, we need to directly incorporate social covariates into models of movement to measure the influence of social factors on animal movements (Supplemental Text S2.3.2). Third, we need to explore the ability of multi-layer network modelling approaches to capture environmental drivers (Supplemental Text S2.3.3). Finally, we need to formalise connections between fission-fusion dynamics and contact network structures to more precisely incorporate environmental and social drivers of movement (Supplemental Text S2.3.4).

Each of these tasks will require (and add to) integration of movement and disease ecology, to benefit of both domains.

MODERN WORKFLOWS FOR CONNECTING ENVIRONMENTAL DRIVERS WITH EPIDEMIOLOGICAL MODEL INPUTS

Predicting pathogen transmission locations is a methodologically diverse objective. Approaches range from mechanistic tactics reliant upon fundamental attributes of the system to phenomenological tactics reliant upon geographical metrics. Emerging methods for Eulerian data (which describe spatio-temporally varying densities without tracking specific individuals) can connect transmission kernels to the environment (Garlick et al., 2011; Hefley et al., 2017; Box 2), but Eulerian methods perform best with patterns that change over both space and time. Eulerian approaches might prove most useful for understanding novel pathogen spread, especially for *K*-selected pathogens, but they might be less-useful for managing endemic transmission or transmission of *r*-selected agents. Here, we focus instead on workflows for Lagrangian data that explicitly track movements of known animals (e.g. through GPS collars or other animal-borne sensors), which are better-equipped to investigate high-resolution environment–movement interactions.

Conventionally, researchers used Lagrangian data to identify drivers of spatial transmission by: (1) correlating transmission with aspects of the environment; (2)

building forward from those correlations to separately investigate each component of the epidemiological landscape and (3) re-combining component-specific estimates to generate overall predictions. These steps are usually ad hoc, and disease ecologists lack clear guidance about which covariates to explore, which components to prioritise, or how to appropriately propagate error. Modern workflows offer new opportunities to overcome each of these challenges (Figure 3).

All workflows (Figure 3) start with a preliminary correlative inquiry relating pathogen prevalence to environmental attributes (preferably using datasets that track changes in relevant environmental covariates). Researchers should then consider how host movement interacts with pathogen pace-of-life, and identify plausible mechanisms relating movement and pathogen persistence to the environment (Table 1). After this point, the workflows diverge.

In the mechanistic workflow, researchers separately model density, mobility and contact as functions of the physical environment, using different datasets and methods for each component (biological processes and integrations with movement described in the Supplementary text; methods summarised in Table 2). Density and mobility can be estimated separately or together depending on data availability, but contact estimates often require distinct datasets (e.g. from proximity loggers or direct observations). The epidemiological landscape can be constructed by predicting densities and mobilities from environmental covariates, and assigning each site specific contact rates depending on local environmental conditions and host densities. Predictions should be validated with pathogen surveillance data whenever possible, but mobility and density models can be validated using movement data alone if necessary. The strength of the mechanistic workflow lies in its ability to draw causal inference from underlying drivers to emergent movements and transmission, which should improve resulting model transferability (but see Section 'Connecting the epidemiological landscape's central components'). Its main weakness is that it can easily overlook host social ecology, so this workflow might work best in systems where host movements can be regarded as independent.

The network-based workflow places social and spatial drivers on common footing from the start. This approach requires defining a spatiotemporal contact function (e.g. a cut-off distance and time) describing the intensity of individuals 'associations', extracting association strengths or events from tracking data, mapping contacts to geographical locations, identifying environmental correlates of those locations, projecting other contact locations across the landscape using the identified environmental correlates, and finally simulating pathogen transmission across the network to estimate site-specific transmission potentials. For example to identify hotspots of *Mycoplasma ovipneumoniae* transmission in bighorn sheep, we might define a 'contact' to be concurrent locations within 50 m of one another within a 2-h time interval. We could then extract all contacts from a set of bighorn sheep telemetry data, perhaps using a continuous-time movement model for times between fixes (Wilber et al., 2022). Next, we could map the contact events back onto the landscape, and match contact locations to environmental covariates (e.g. by fitting a resource selection function directly to the contact events). We could then connect individual animals to contact locations to build a bipartite network, and scale the network up by increasing the number of individual and spatial nodes to reflect the population's size and spatial extent (achieving a model of density and mobility within the system; Figure 3). Uncertainty in edges could be reduced by applying marginal information about group sizes and individual-level habitat selection to assign deer to locations (Cross et al., 2019; Manlove et al., 2018; Silk et al., 2018). Finally, we could simulate transmission on either a static or a dynamic representation of the network and extract cell-specific transmission potentials (similar to White et al., 2018). Whether forecasted hotspots actually harbour more host aggregations can be validated using local movement data, and whether those aggregations lead to transmission can be validated using pathogen data when those data exist. The strength of the network-based workflow is that it balances spatial and social forces. Its main weaknesses are its dependence on tracking intensities high-enough to capture contacts and its currently limited application in real-world settings. How to best discretise space and adjust for spatial autocorrelation are areas of open inquiry.

The final workflow builds from mining-modelling approaches for disease dynamics (Han et al., 2020). Data mining can be applied to a specific process within the epidemiological landscape (Example Strategies 1 and 2 in Figure 3), or to entire animal movement trajectories (Example Strategy 3 in Figure 3). The crux of this workflow is in translating hypothesised mechanistic drivers into quantifiable 'features' that can be measured along individual movement trajectories. For example if our goal was to model movement trajectories of wild pigs, we might include features describing environmental context at several scales (e.g. percent cover, distance to water, etc.). Other features might capture the pig's movement trajectory, including its step lengths and turning angles at various timelags, as well as the angle it would need to turn to orient towards nearby resources (e.g. the angle it would need to turn from its current heading to orient towards water). On the social front, we could include features like distance to all other collared pigs, as well as the turning angles required to re-orient towards them. The set of features would then be used to train a machine learning algorithm, which would be validated against a subset of withheld movement trajectories and then used to forecast movements across all individuals. Pathogen pace-of-life attributes could be overlaid on the predicted movements to simulate transmission,

again resulting in predicted site-specific transmission potentials. Feature importance measurements could be extracted to inform future research isolating and testing specific mechanisms. The strengths of the mining-modelling workflow are its ability to weight social and spatial factors in tandem, and its ability to scale up across individuals to infer density, contact and mobility across entire landscapes. Its weaknesses lie in its dependence on researcher-identified features, its inability to identify causal mechanisms, and its abbreviated track record of application in movement ecology and spatial epidemiology.

Accurate prediction does not ensure effective intervention if the system's mechanistic drivers remain unknown. Mechanistically defining the epidemiological landscape can add insights that purely phenomenological multipartite network or mining-modelling approaches cannot. Ideally, the phenomenological workflows would be used in iteration with mechanistic inquiries.

OPEN CHALLENGES

Connecting the epidemiological landscape's central components

Methods to integrate the epidemiological landscape components (density, mobility and contact) and correctly propagate error remain in short supply (Jerde & Visscher, 2005; Ruckelshaus et al., 1997). Propagating uncertainty is an important challenge for any workflow that moves information across scales or processes to generate predictions. Bayesian integrated modelling (and to a lesser extent, mixed modelling) methods can handle this challenge (e.g. Muff et al., 2020; Schaub & Abadi, 2011), but posing a robust model in the presence of a tower of uncertainties can be difficult. A broad spectrum of ecologists and statisticians are confronting these problems (e.g. Tredennick et al., 2021), and the opportunities from Bayesian approaches are improving as tailored sampler designs become more accessible (e.g. through platforms like NIMBLE; de Valpine et al., 2017). We encourage disease ecologists to collaborate with experts in error propagation and model validation when constructing spatially explicit models of transmission. Appropriate integration, first of means and then of errors, is an urgent need in spatial epidemiological inquiry.

Identifying the correct level of detail

Which biological details to include depends on data resolution and project objectives. Mechanisms should be included if they are central to the overarching question or change resulting predictions. Decisions about which biological details to include should precede decisions about model construction, since some methods cannot capture certain mechanisms. The relative timescales of host movements, environmental fluxes and pathogen pace-of-life are also informative: processes that change slowly relative to system epidemiology could be treated as constant; but processes that change quickly might need to be dynamic (Funk et al., 2015).

Validating spatial patterns of transmission requires the well-designed collection of pathogen surveillance data. The best way to gather surveillance data depends on the focal system's diagnostic methods. Sampling designs for transmission have been reviewed elsewhere (e.g. Plowright et al., 2019), so we focus more narrowly on considerations for movement data. Movement data's temporal resolution is often under researcher control, though resolution trades off against device longevity through battery and memory capacities (Kays et al., 2015). Background knowledge about the timescales of relevant host movements and pathogen life-history traits (i.e. infectious periods and periods of environmental persistence) can inform temporal resolution (Benhamou, 2014; McClintock et al., 2014). Since rare, longer-distance moves drive pathogen invasion speeds, optimal disease invasion tracking might rely on slower fix rates and longer tracking periods. On the other hand, extracting direct contacts from continuous time movement models might require much higher-resolution data, typically with fix rates faster than one point per hour. As a consequence, different tracking rates might be appropriate for developing (faster fix rate) and validating (slower fix rate) models of transmission.

The spatial resolution of movement data is often determined by the technology employed, but researchers often control the geographical and environmental contexts in which devices are deployed. Estimating environmental effects will be most efficient when the environment varies substantially across the study's spatial domain, but if direct contact data are required, tracking densities should remain high in some areas. Ideally, one should know the ratio of tracked animals to total hosts across the study area. Habitat attributes that are consistent across the study area can be excluded from local predictions, but should be considered if the model is used to predict dynamics elsewhere.

Finally, constructing reliable density, mobility and contact estimates often takes substantial effort (e.g. through improved descriptions of important covariates, increased performance of disease diagnostic tests or refinements to statistical methods). This burden might diminish over time, but shortcutting variable development can be costly, and investing in some form of data improvement is often necessary.

Extending outside the measured context

The relation between density, mobility or contact and environmental attributes may depend on the attribute's

local availability (similar to a functional response; Mysterud & Ims, 1998). Availability-dependence is the rule, not the exception, in animal movement (Avgar et al., 2020), and while availability-dependent relationships between movement and the environment do not preclude extrapolation, their presence should be considered when predicting transmission in unsampled geographies or environments. Meta-analyses of how habitat selection, step selection or other attributes of movement vary across environments could offer baseline expectations about how availability affects density and movement, especially for hosts of common interest.

Disease feedbacks can also produce nonlinear functional responses. A low-density population recovering from disease could have different rates of long-distance movement than nearby populations where densities are high; and age-specific mortality burdens can influence social structure, especially if mortalities concentrate among older and more knowledgeable individuals. The relationship between time since pathogen deposition and instantaneous rate of transmission could also be nonlinear (Almberg et al., 2011; Richardson & Gorochowski, 2015), but guidance about how to weight the force of infection arising from different modes of transmission is limited (Breban, 2013).

Finally, predictions can fail in environments containing spatial or social features that never arose in the training data. Host populations with seasonal birth pulses could have different habitat selection patterns than populations where birth pulses are diffuse, and habitats that are seasonally abandoned at some latitudes might be occupied year-round at others. In these cases, researchers could fall back to mechanistic approaches drawn from first principles of system biology.

CONCLUSION

The interface between movement and disease ecology offers exciting opportunities to improve spatially explicit models of pathogen transmission and motivate research into mechanisms shaping animal movement more generally. Rapid advances and new workflows in movement and disease ecology give the interface a strong foundation, and synergistic developments could benefit both fields. However, improving spatially explicit epidemiological forecasts might also require shifts in emphasis. The epidemiological landscape, consisting of environmental processes shaping host and pathogen movements, along with emergent patterns of density, mobility and contact, provides a conceptual bridge connecting environmental mechanisms to spatially explicit patterns of pathogen transmission. Focused inquiry into the mechanisms that underpin the epidemiological landscape and the phenomena that emerge from it could reveal overlooked opportunities for targeted data collection, new applications of tools from movement ecology, and avenues for future method and theory development. We hope that this synthesis sparks conversations that advance perspectives in spatial epidemiology and strengthen the conceptual bridge connecting environment, movement and transmission.

AUTHORSHIP

All authors contributed to manuscript conceptualisation through a series of discussions. KRM built figures and tables and drafted the text. All authors contributed substantially across multiple rounds of manuscript revision.

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This paper contains no data.

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