A modeling lens on landscapes-of-peril and other ecological drivers of zoonoses

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Abstract. *Ecological systems are driven by a complex suite of environmental conditions, interactions among individuals, and reciprocal behavioral and biological responses of organisms to the ecosystems they inhabit. Individuals may, in turn, reshape those systems, generating feedback that alters the ecological drivers influencing subsequent system dynamics. Extractive and competitive processes are embedded within responses to predation and infection risk, shaped by perceived hazard together with resource value and individual state. Peril need not be perceived, however, to bear on fitness and pathogen transmission. Exposure arises from the intersection of a pathogen landscape that is a property of place with what animals do there, and infection from the state that determines susceptibility to a given dose. These interdependent dynamics can scale upward to influence successional, ecological, and evolutionary processes over time. An emerging framework for incorporating fear and disgust as ecological drivers is the landscape of peril (LOP), which integrates predation and infection risks across heterogeneous environments. LOPs provide a means of characterizing when and where individuals move, how they allocate time among resource patches, and the activities in which they engage at a location in response to spatially and temporally varying hazards. These state-dependent decisions can influence pathogen-transmission dynamics by altering contact between susceptible and infected individuals, the structure of contact networks, opportunities for exposure, host susceptibility, and pathogen shedding. Humans now exert a transformative influence on ecological systems and are major drivers of global environmental change. Humans may also experience ecosystem-dependent landscapes of peril of their own. Their responses to these perceived risks can shape land use, resource distributions, infrastructure, waste, wildlife management, human behaviors associated with tolerance, exclusion, and persecution, thereby engineering the environmental conditions and spatial gradients of risk encountered by other species. Wildlife responses may subsequently feedback*

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to alter human perceptions and actions, although the strength and direction of this feedback will vary with communities and human populations. In this chapter, we develop a framework for identifying the ecological, environmental, anthropogenic, and state-dependent drivers of zoonotic processes that are most salient for agent-based modeling. We explicitly consider the role of human landscapes of peril and the mechanisms through which human perceptions and actions influence the landscapes of peril experienced by wildlife. We propose that future studies incorporate these processes within a modeling framework that we term PEARLS: Peril, Environmental, Anthropogenic, and Resource Landscapes and States. We illustrate the application of this framework using enzootic tuberculosis caused by Mycobacterium mungi, a member of the M. tuberculosis complex, in a population of banded mongoose (Mungos mungo) occurring across a human–wildlife continuum in Northern Botswana.

1 Introduction

An articulation of successional and evolutionary principles requires that we have a comprehensive understanding of population and community ecology, particularly in terms of how environments influence the behavior and movement of individuals, the growth of populations, and interactions among species, including host-parasite dynamics and the transmission of pathogens and parasites across space and time [1–7]. From these interactions, emerge environmental contexts and regulatory forces that, in turn, influence ecological and evolutionary responses to environmental change [8, 9]. This creates feedback cycles of successional destructive and renewal processes, as well as coevolutionary processes resulting in the emergence of ever more complex systems (i.e., increases in diversity at multiple levels) [10].

At much faster time scales, literally, minute by minute, the behavior of individuals becomes important in determining how the conditions encountered at a location drive ecological processes [11, 12], including the spatio-temporal dynamics of zoonotic diseases. Such considerations fall within the ambit of behavioral landscape ecology, which draws upon the behavior of individuals and groups to design experiments and build predictive models [13]. The landscape of fear (LOF) represents spatial and temporal variation in predation risk, including risk associated with humans [14], while the landscape of disgust (LOD) represents spatial and temporal variation in infection, contamination, and parasite risk arising from infected individuals, excreta, carcasses, contaminated resources, and substrates [15, 12]. These landscapes shape where animals move, forage, rest, interact, defecate, scent-mark, and encounter potentially infectious material. Their integration into a single landscape that individuals traverse and evaluate as they move is now referred to as a landscape of peril (LOP) [16].

LOP: Perceived and realized peril

An LOF is a surface of information an animal uses to decide where to go and what to do there, weighed against resource gain in relation to its own state [17–19]. In some cases, the peril itself may not be perceived. An LOD is typically assembled from empirical observations of animals avoid feces, urine, carcasses, and visibly infected conspecifics [11]. Avoidance of detectable waste is, however, a narrow case. Most pathogen occurrence elicits no aversive response, either because it goes undetected or because the site is worth using regardless, and a landscape assembled from aversion alone will not represent a landscape of peril [20].

When incorporating an LOP into a model by formulating an algorithm on how an individual uses information contained in an LOP, we need to distinguish between realized and perceived hazard. Realized hazard bears on an individual's fitness whether or not the organism perceives it. Perceived hazard, influences an organisms movement and other behaviors. Contamination may enter the same evaluation as predation risk and shape what an animal does [12]. It may equally go unperceived, in which case it influences no choice at all, while still bearing on

fitness and on pathogen transmission. Peril, as we use the term, comprises both: the hazard an organism perceives and acts on, and the hazard that bears on it regardless.

The behavioral response to a peril is not the definition of its occurrence. Where a pathogen is perceived, avoidance is one response among several, and attraction is possible where the cue that signals contamination also signals a resource [11]. Where it is not perceived, there is no response to observe. Exposure and transmission arise in either case from the intersection of what is present at a location with what an animal does there, since foraging, competition, and social interaction bring individuals into contact with contaminated material and with each other, whether or not the contamination was evaluated.

Individual state enters at two points. It modulates behavior, since nutritional need, stress, and reproductive demand alter the weight given to risk relative to resource gain wherever a hazard is perceived [19, 21]. It also modulates outcome, since condition, reproductive status, and immune competence alter susceptibility to a given dose. The second role does not depend on the first, so state remains integral to the framework even where perception contributes nothing.

LOP: Natural factors

The role of the LOP in influencing pathogen transmission in wildlife remains poorly investigated, particularly with respect to contact between susceptible and infected individuals, the activities in which they engage across different environments, and the resulting structure of contact networks. A key component of the LOP is the foraging cost of predation, defined as the risk of injury or death while foraging at a particular location, multiplied by the individual's fitness (i.e, its expected present and future survival and fecundity if it avoids death during the current activity) and divided by the marginal fitness value of acquiring additional food [17]. While foraging, an animal may engage in vigilance behaviors that reduce the inherent risk of predation but slow feeding and divert time from resource acquisition [22, 23]. At safer feeding locations, reduced vigilance may permit greater engagement in interference competition. In mongoose, for example, one sees biting or fighting, anal sniffing, scent marking, over-marking, and investigation of scent marks. The fear component of the LOP thus influences both the allocation of time among patches within the home range and the types of activities performed at locations [24]. These responses are determined not only by perceived risk but also by the animal's state and hunger level, with consequences for pathogen transmission through changes in patch use, activity type, contact-network structure, host susceptibility, and pathogen shedding.

Pathogen prevalence and transmission in wildlife species depends on many interrelated factors influenced by where individuals choose to spend time, whom they interact with, and the physical condition or state of the individuals involved. Climatic conditions and seasonal drivers influence host state while simultaneously altering the environmental distribution of food, water, vegetation, ground-cover types, predators including human-associated predators competitors, pathogens, and contamination cues. Individuals in poor condition may ex-

hibit reduced sensitivity to both predation and infection risk, thereby reducing the influence of their perceived LOP by decreasing the costs of using risky or contaminated patches relative to anticipated resource gains. In some locations, a stressed host state may reduce predator vigilance, grouping, and flight behavior. In other locations, it may reduce avoidance of contaminated resources, infected conspecifics, fecal deposits, carcasses, or heavily used locations, thereby increasing exposure to environmentally or socially transmitted pathogens. A stressed host state may also increase competitor vigilance, direct competition, aggression, scent marking, olfactory investigation, and other behaviors that influence transmission potential.

A host's state may consequently alter the relative influence of fear and disgust. Animals may avoid locations perceived as dangerous because of predators while continuing to use contaminated patches when resources are scarce. Conversely, they may tolerate greater predation risk to avoid locations associated with infection or contamination. Fear and disgust may therefore act simultaneously, synergistically, or antagonistically as animals negotiate the relative risks and benefits associated with different patches. These interacting risk landscapes can redistribute individuals across space, alter group size and contact-network structure, and modify behaviors such as vigilance, aggregation, aggression, competition, scent marking, olfactory investigation, grooming, foraging, avoidance, and flight. The same factors may variably influence physiological stress, immune function, pathogen susceptibility, host–pathogen interactions, and pathogen shedding across habitat patches and land-use types.

Predictions of within and between season changes in contact-network structure and pathogen-transmission potential therefore emerge from the combined effects of host state, group size, patch use, resource distribution, and transmission-relevant behaviors across a socioecological system. Anthropogenic and climatic forcings may reshape these relationships by modifying habitat structure, resource availability, predator and competitor distributions, contamination patterns, host condition, and the relative costs and benefits of occupying particular patches. By influencing both the fear and disgust components of the LOP, these forcings may alter where and when animals aggregate, interact, encounter pathogens, become susceptible to infection, and shed infectious agents. Conversely, observed changes in host state, patch use, behavior, network structure, and pathogen transmission within and among seasons may provide insight into the mechanisms and consequences of human forcing across the system. Understanding how wildlife negotiate these overlapping landscapes of fear, disgust, resource, in relation to their internal states provides a mechanistic basis for predicting when and where environmental change may increase pathogen transmission within and among species and elevate the potential for zoonotic spillover. Because these interactions apply across diverse host–pathogen systems, the integrated LOP framework provides a natural foundation for investigating and predicting infectious-disease risk across changing landscapes and climates.

LOP: Anthropogenic factors

Humans, as they occupy and transform landscapes, act as critical ecosystem engineers, restructuring environmental conditions, risk distributions, resource availability, and wildlife community composition, while often reducing biodiversity and biological complexity. Humans may function simultaneously as superpredators that generate fear and avoidance and as providers of refugia, food, water, and other subsidies that attract wildlife and may enhance survival and fitness [25–28]. They may also generate spatially heterogeneous patterns of response to wildlife, ranging from tolerance and protection to harassment, exclusion, and direct persecution. These differences create gradients in human-associated risk that wildlife must perceive and navigate across the landscape.

Human-provided subsidies to wildlife can impose indirect ecological costs by increasing opportunities for parasite transmission through waste-associated pathogen exposure [29], host aggregation [30], or shifts in intraspecific behavior [31–34]. Thus, the same human-modified patch may simultaneously provide food, water, shelter, or refuge while also increasing exposure to persecution, predators, competitors, contaminated materials, or infectious conspecifics. Yet many ecological frameworks continue to represent humans primarily as passive or external disturbance agents, overlooking the dynamic and spatially structured consequences of human perception, decision-making, and behavior.

Both humans and wildlife actively interpret spatial and temporal variation in risk, although they generally do not perceive or respond to the same LOP. Wildlife perceive their LOPs in terms of fear associated with predation and persecution, as well as disgust associated with parasites, pathogens, and contamination. Humans act in response to their own perceptions of threat, contamination, wildlife, economic loss, personal safety, resource need, and other social or ecological concerns. These human perceptions shape land-use, settlement, infrastructure, waste disposal, resource provisioning, wildlife management, protection, tolerance, exclusion, and persecution. Capturing this complexity is essential for advancing a mechanistic understanding of the ecological processes that structure population dynamics, community interactions, and host–parasite relationships in human-altered ecosystems.

Through these actions, humans engineer the environmental, anthropogenic, and resource landscapes encountered by wildlife. Human decisions therefore generate spatial and temporal variation in the risks and resources available to other species, including gradients from tolerance to persecution, refuge to exposure, and subsidy to contamination. Wildlife responses to these human-generated conditions can alter movement, space use, group size, contact networks, physiological state, and parasite transmission dynamics [31, 35, 36, 17, 22, 37, 18, 38, 39]. Capturing this mediated relationship is essential to advancing a mechanistic understanding of the ecological processes that structure population dynamics, community interactions, and host–parasite relationships in human-altered ecosystems.

LOF frameworks and empirical studies [36, 38, 19, 40–55] have discussed and shown how animals adjust movement, space use, and interactions in response to perceived risk, resource availability, and internal state. The application of integrated LOF and LOD frameworks to host–parasite ecology remains limited,

particularly in human-dominated landscapes where predation risk, persecution, tolerance, subsidies, contamination, and other stressors co-occur and interact [56–59]. In parallel, a growing body of work has developed conceptual models describing how human decision-making affects the dynamics of emerging and circulating human pathogens, and vice versa [60–66]. This literature, however, remains largely conceptual and focused on processes occurring exclusively within human populations [67].

Although parasites can influence demographic trajectories, behavior, and trophic dynamics [68–72], we still lack a mechanistic understanding of how various human-engineered landscapes structure wildlife behavior, movement, contact networks, internal state, and parasite transmission dynamics across heterogeneous environments [1, 28, 59, 71]. In particular, we lack models that explicitly capture how spatial variation in human tolerance, protection, disturbance, and persecution combines with resource distributions, environmental conditions, contamination, and host state to influence wildlife decisions and pathogen transmission. Although laboratory studies demonstrate the importance of LOP (infection) feedback [73, 74], these simplified systems lack spatial complexity, multiscale movement processes, and linked human–wildlife interactions necessary to evaluate landscape of peril mediated transmission dynamics in free-living hosts.

Critically, a human experienced landscape of peril (denoted herein by LOP_0), in contrast to the landscape of peril experienced by other animals within an ecosystem (denoted herein by LOP_i for species $i = 1, 2, \cdots$) remains largely absent from mechanistic ecological models. An LOP_0 produces spatial and temporal gradients in perceived threat, contamination, economic cost, wildlife conflict, and other hazards relevant to human decision-making. Societal responses to the LOP_0 are then expressed through behaviors and institutions that transform the system, including settlement patterns, land-use decisions, resource extraction, infrastructure development, waste disposal, wildlife management, protection, tolerance, exclusion, and persecution. The landscape of peril experienced by other animals within the ecosystems is therefore influenced partly by the ecological consequences of human actions undertaken in response to LOP_0.

Humans need not perceive or respond directly to any LOP_i. Rather, their decisions engineer the conditions from which the various LOP_i's emerge by altering habitat structure, predator and competitor distributions, contamination risk, resource availability, refugia, subsidies, and the spatial distribution of human tolerance and persecution. Wildlife subsequently perceives and responds to these engineered conditions through changes in movement, behavior, group structure, physiological state, and contact patterns. Wildlife responses may, in some circumstances, feedback to alter human perceptions and subsequent decisions, but such feedback should not be assumed to occur uniformly. Their strength and direction are likely to vary among species, human communities and individuals, institutions, landscapes, and social contexts. The relationship between LOP_0 and any LOP_i is therefore mediated through human engineering of the socioecological system rather than through an assumption that humans and wildlife perceive or respond to the same risks.

Need for a general framework

Mechanistic ecological models that omit a dynamic LOP_0 and its behavioral and institutional consequences, cannot be used to predict how human-generated landscape change subsequently scales up to shape wildlife host–parasite dynamics, population structure, and broader community processes [1, 28, 59, 71]. Here, we consider the identification of the most critical ecological drivers of zoonotic disease through the lens of dynamical systems modeling. We advance the need for analytical tools and modeling approaches within a unified theoretical framework that integrates human perception and decision-making with human engineering of environmental, anthropogenic, and resource landscapes, and with wildlife peril, internal state, movement, contact structure, and parasite-transmission dynamics. Such a framework is needed to determine which ecological drivers are most salient for predicting changes in zoonotic processes in response to anthropogenic change and disturbance. Ultimately, mechanistic studies will be required to test how spatially structured peril, environmental conditions, anthropogenic influences, resource distributions, and internal state interact to shape host behavior, contact structure, and parasite transmission.

We begin by proposing a general framework that we have termed **PEARLS**: **P**eril, **E**nvironmental, **A**nthropogenic, & **R**esource **L**andscapes & **S**tates. This framework is intended as an overarching conceptual framework for identifying and organizing the interacting processes that may influence zoonotic dynamics, rather than as a prescription for a single model structure. We then discuss how the factors embodied within the PEARLS framework, including empirical landscape layers, organism-specific perceptions and responses, individual states, and resulting behavioral and epidemiological processes, can be identified, evaluated, and incorporated into agent-based models of zoonotic processes. Finally, we demonstrate how the framework can be applied to a banded mongoose population (*Mungos mungo*) [75] in northern Botswana, including how urbanization and resulting host specific LOP factors affecting the movement of mongoose individuals and troops may influence the impact of the *Mycobacterium* tuberculosis complex pathogen, *M. mungi*, and its persistence as an endemic disease within this population [76–78].

2 PEARLS Framework: General Considerations

2.1 Motivation

Landscape of peril (LOP) theory has provided major advances in predicting risk-sensitive behavior and population- and community-level consequences, including altered space use, community structure, trophic cascades, and ecosystem processes [18, 47]. Behavioral responses to hazard follow from an individual's evaluation of that hazard in relation to its own state, including its energetic needs and stress physiology. The individual's states modify risk perceptions and behavioral responses [19, 42, 79, 21] reinforcing the tight link between physiology, movement, and ecological dynamics that underlie parasite transmission. Because

humans are both sources and perceivers of risk and opportunity, applying LOP principles to human-dominated ecosystems demands explicit consideration of human-generated cues and wildlife responses.

Increasingly, human perceptual ecology is recognized as a necessary field for understanding how people interpret and respond to wildlife, most commonly in the context of conflict research and management. Although the presence of humans strongly shapes wildlife behavior, ecological models rarely integrate formalized human perception as a spatially-explicit map, nor do they incorporate human fear. Existing studies demonstrate that humans can generate stronger fear responses in wildlife than natural predators [52], and that conservation outcomes require explicit attention to socio-ecological processes, including human risk perceptions and decision-making [80]. Yet, most ecological applications treat humans as a binary disturbance source rather than as organisms with their own spatially structured perceptions of safety, threat, and opportunity.

Research on wildlife LOPs shows that predation risk and perceived danger strongly influence behavior, movement, and habitat use [48, 81], and growing evidence indicates that human presence intensifies or reshapes these responses [82, 83]. Representing human perceptions as a spatial surface becomes essential because human perceptions of threat, opportunity, conflict, and contamination vary across space and time and influence subsequent human behavior and decision-making. The ecological consequences of human activity, whether expressed as persecution, disturbance, tolerance, or provisioning along some gradient, are therefore heterogeneous in both space and time, and depend on where and when they occur.

Therefore, we argue that a spatially explicit human LOP_0 should represent the distribution of risks and opportunities as perceived by humans, while the behavioral and institutional responses generated by those perceptions—including persecution, disturbance, tolerance, provisioning, protection, and land-use decisions—should be treated as mechanisms through which humans alter the conditions experienced by wildlife. In turn, this approach would enable formal tests of how human perceptions and actions modify landscapes and thereby reshape wildlife movement, behavior, internal state, contact structure, and transmission processes. Ultimately, representing these dynamics explicitly within computational models should improve predictions of wildlife responses across heterogeneous human-modified landscapes and strengthen the capacity to anticipate the ecological and epidemiological consequences of changing human activities for enhanced predictive power.

Overall, there is currently a critical need for an integrated, empirically-grounded framework that:

i) captures interacting human and wildlife landscapes of peril, including hazard that is not perceived as well as those responses to hazards that are
ii) organizes peril, environmental, anthropogenic, and resource landscapes together with the individual states, and links these interacting components to movement, contact networks, and parasite exposure and transmission within the **PEARLS** framework (Fig. 1)

iii) operates at ecologically meaningful spatial and temporal scales.

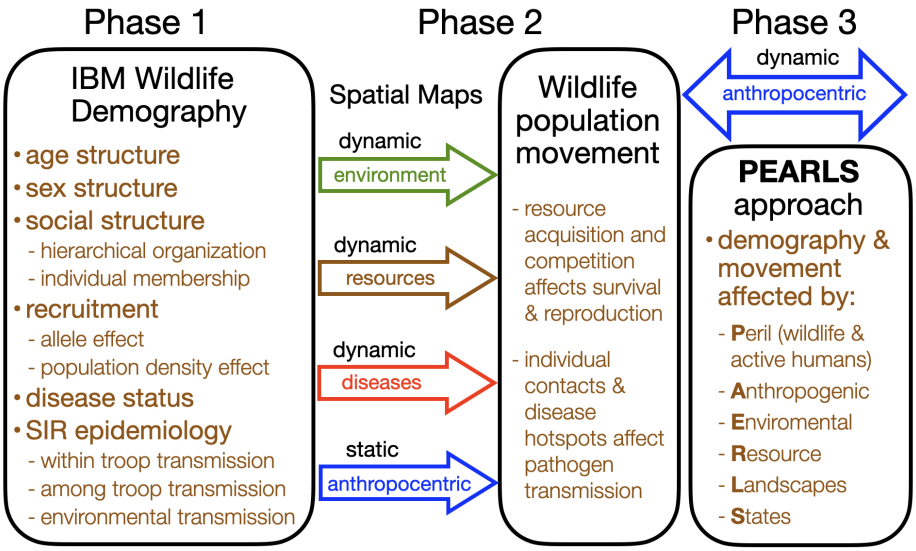

Fig. 1. The **PEARLS** (**P**eril, **E**nvironmental, **A**nthropogenic, & **R**esource **L**andscapes and **S**tates) framework organizes the interacting landscape and individual-level processes that may shape wildlife behavior and zoonotic transmission. The three phases shown above represent one possible sequential strategy for constructing and evaluating models of increasing spatial and socio-ecological complexity; they are not separate components of the framework itself. The three phases identified above are more fully described in the text.

This will require future studies to develop, empirically derive, and experimentally validate spatially-explicit agent-based models that include perceptions of peril, anthropogenic structures on the landscape and the activity patterns of humans, resource distributions, and the internal states of individuals and external environmental drivers into a general framework that incorporates both human and wildlife LOPs. Such models, once validated, can then be used with some confidence to predict and test how heterogeneous, human-modified environments restructure wildlife dynamics and host–parasite interactions, with the potential to scale up and predict changes at the community and ecosystem levels.

2.2 Integrated ABM platforms

A modeling platform that links behavior and disease states of individuals to population-level spatial and temporal infection patterns requires the integration of empirical data into a mechanistic modeling framework. Such platforms will be agent-based (ABM) given the need to include the internal states of individuals

in the response of these individuals to external factors. They will likely discretize time and space at an appropriate level of resolution where the landscape is represented by a pixelated array of cells. The state of each cell at a particular point in time represents the environment of one or more individuals occupying it, and this state varies over time (see [84]). They also need to seamlessly incorporate multiple derived landscape layers, including geographic, topographic, environmental, anthropogenic, resources, risk, and pathogen or contamination together with organism-specific LOP surfaces derived from how these conditions are perceived and evaluated in relation to individual state. The organism-specific LOP is computationally derived to estimate how individuals perceive and evaluate spatially varying environmental, anthropogenic, resource, and peril-related conditions in relation to their internal state and the environmental state of their current location, and how these evaluations shape movement, behavior, contact structure, and pathogen transmission. These computations can be implemented using NetLogo [85], Numerus Model Builder [39, 86, 87], or other agent-based modeling platforms [88].

At the simplest level, only individuals in the focal wildlife population may be treated as agents. Humans, on the other hand, may be treated in terms of a background spatiotemporal distribution of activity levels as influenced by diurnal rhythms. The human activity component could vary with day of the week or season, where appropriate data on such variation is available. The behavior of humans may then be incorporated into the model by including changes induced by the current LOP_0, which itself could depend on the current location of various individuals in the wildlife population and on how humans perceive their disease state and the disease state of other groups of humans nearby.

In turn, these human-driven behavioral gradients modify local environments in ways that alter wildlife movement, space use, and contact opportunities. These behavioral responses get incorporated as spatial modifiers of cell risk and opportunity. Since each individual in our framework is represented by an agent whose movement, activity, and contact-generating behavior are governed by empirically derived decision rules linking landscape conditions and internal state to observed behavior, the framework can explore the impact of LOPs on the incidence and dynamics of zoonoses by comparing outcomes under various scenarios that include or do not include an explicit use of LOPs in the model.

Movement is generally incorporated into an agent-based landscape model using a step selection approach [89–91]. This approach employs behavioral rules to define how agents move over a cellular array and how choices vary with the agent's internal state [92, 93]. In our proposed PEARLS formulation, LOP_0 (human) and LOP_1 (focal wildlife species) surfaces will directly modify the probability that an agent moves from its current cell into neighboring cells at each time step. An agent's movement decision will follow a multinomial habitat-selection function in which the relative attractiveness of nearby candidate cells is determined by the candidate cells' LOP values (reflecting human activity, tolerance–persecution gradients, and associated constraints on wildlife) relative to the current cell's LOP value and resource covariates.

The above prescription allows all movement trajectories to arise mechanistically from empirical relationships linking LOP, resources, and state to observed space-use decisions. A dynamic, heterogeneous contact network provides an additional response layer capturing direct and indirect contacts among individuals of a group or of a shared site. Together, these components allow one to simulate and mechanistically test the ecological drivers of pathogen and parasite transmission in changing landscapes and how these dynamics scale to population-level infection patterns across heterogeneous landscapes. Experimental manipulations provide a direct test of ABM predictions by altering LOP structure in controlled ways and evaluating whether observed changes in movement, behavior, and transmission align with model-forecast responses, thereby validating or falsifying alternative transmission mechanisms.

2.3 Disease transmission network component

Epidemiological networks are often used to model the transmission of infectious diseases through a population, where a contagion (parasite/pathogen) moves from infected to non-infected nodes (individuals) via edges connecting nodes (social or environmental contacts with the potential for pathogen transmission [94–97]). Statistical inference methods can be applied to these networks to predict parasite transmission, estimate epidemic thresholds, identify transmission hotspots, and ascertain the effect of community structure [95, 97, 98].

The above proposed contact network model forms a second layer of input into the ABM framework presented above. Contact networks may have social substructures, such as troops or herds within a more extensive population. To model transmission among the substructures "heterogeneous" contact networks are more appropriate [99]. This approach allows vertices and edges in the contact network to differ in type. In populations with a two-tier structure (i.e., groups within a population or a population of groups) two types of vertices and three types of edges are needed. Specifically:

i) an edge between two groups represents inter-group interaction (e.g., dispersal, fusion, boundary contacts etc.) that can potentially lead to pathogen transmission between groups
ii) an edge between two spatial sites represents a group traveling from one site to the other, which can potentially lead to pathogen transmission across the landscape
iii) an edge between a group and a spatial site indicates that the group is inhabiting that site, which could lead to (indirect) pathogen transmission when another group visits the same site within a certain time interval and contacts environmental sources of the pathogen

Our approach allows the contact network to be dynamic, capturing changes across seasons [94, 100–103]. The dual LOP model can then be tested for robustness using stochastic uncertainty analysis. Predictions can be validated against independent movement and behavior data.

3 PEARLS Framework: Assembly

The problem of investigating behavioral drivers of zoonoses has both theoretical and empirical elements to it. Such investigations typically involve fitting mechanistic or statistical models to empirical data. Behavioral drivers relating to predation and to pathogen exposure are jointly captured by the landscape of peril. Fear and disgust operate in different currencies and are signaled by different cues, and contamination risk further differs in two respects. It is partly generated by the individuals responding to it, so the pathogen landscape is endogenous in a way that topography and physical structure are not, and must be updated from agent occupancy and pathogen shedding at each step. Also, it need not elicit avoidance, since a patch may carry high realized hazard and high net resource value at the same time. What an individual does at a location follows from an integrated evaluation of peril, resources, and environmental conditions made in relation to its own internal state. State is therefore not an additional driver but the reference against which the elements of the landscape are assessed, and the resulting movement, patch use, and activity are outcomes of that integration rather than of any element within it.

The phases we propose for building models capable of identifying and evaluating landscape of peril drivers of zoonoses are depicted in Fig. 1. The first phase is to build a demographically-structured IBM of the wildlife population of interest with disease states that relate to the zoonotic disease of interest. We recommend containing the complexity by assuming spatially homogeneous processes. Once this model has been constructed, fitted to spatially-pooled data, and deemed adequate [92] under the assumption of spatial homogeneity, it can be extended to incorporate as few or many spatial elements as deemed appropriate to address the problem at hand.

In general, Phase 2 typically involves allowing individuals identified in Phase 1 to move over the structured landscape, represented by a cellular array, where each cell has its own time-varying state. These cellular states may include physical environmental variables (elevation, soil or surface type, temperature, precipitation, etc), vegetation (land cover type), resources relevant to the wildlife population under concern (e.g., edible plants, seeds, fruits), relevant occupants (prey items, conspecifics, competitors, predators), level of environmental contaminants or pathogens (the pathogen landscape), and level of risk from predation. They may also include measures reflecting the anthropocentric factors (road coverage, building coverage, garbage levels, etc.). In this phase, any human presence is provided by static (constant) cellular values.

Phase 3 is characterized by the fact that the anthropocentric input is no longer static, but time varying. In addition, these time-varying anthropocentric cellular values change in response to the demographics and movement of the wildlife population itself. These values reflect construction of fences or barriers to wildlife movement, manipulation of resources availability to wildlife, and adjustments to human movement in response to the presence of wildlife.

3.1 Phase 1: Spatially homogeneous wildlife IBM zoonotic models

Characterizing Individuals

In an individual-based approach, the primary components of a zoonotic process in social species (e.g., packs, troops, herds, prides) include the following (Fig. 1):

- the life history processes of recruitment (births), aging, and senescence/death
- the social structuring processes of group formation/consolidation (aka fusion) and dissolution (aka fission)
- the epidemiological processes of disease transmission, disease induced mortality, and recovery with possible permanent or waning immunity

More complicated formulations may involve more than one species and may also track underlying genetic elements that confer particular traits of interest, such as levels of susceptibility to the disease being modeled. We use the designator "type" to cover these possibilities. Consider a single species population (although genetic types or age structure could still be allowed) structured into J social groups, the j^{th} of which ($j = 1, \cdots, J$) is assumed to be of size $n_j(t)$ at time t in a total population of size

$$N(t) = \sum_{j=1}^{J} n_j(t) \tag{1}$$

at time t. Then the i^{th} individual, Id_i, can be represented by a row entry in a data base that has the general form at some time $t = 0, \ldots, T$. This enters into the simulation as:

$$\text{Id}_i(t) := \{\text{Type}_i, \text{Sex}_i, \text{Group}_i(t), \text{Life Stage}(t)_i, \text{State}_i(t)\}, \quad i = 1, \cdots, N \tag{2}$$

We note that the choice of t depends on the longevity of the species involved, as well as the type of disease being modeled. For example, in modeling the spread of a fast-replicating respiratory virus, such as influenza, the time units may be weeks or even days, while when modeling the course of a slow acting pathogen (e.g., as in tuberculosis in many mammals) months or quarters may be appropriate for short-lived animals (e.g., small mammals) or years for long-lived animals (e.g., large mammals). In addition, some of the designators attached to each individual in Eq 2 are fixed value "tags" while others are time-varying "variables." Specifically, "Type" and "Sex" are tags while "Group" (individuals may move among groups during dispersal and fission/fusion events), "Life Stage" (generally age for vertebrates, but more complicated age-stage classes may be germane for some vertebrates and most invertebrates) and "State" (internal state of individuals which beyond disease classes may include physiological variables such as biomass, body quality, temperature, a particular hormone level, or some other relevant variable that can be measured) are time-varying variables.

Model Implementation

The simulation model itself could be expressed as differential equations that model continuous changes over time (though discretization of time is required for numerical simulations of such systems) or in terms of iterative changes at discrete points in time. The latter is much easier to formulate when treating certain processes such as births or pathogen transmission among individuals as discrete events. Given the complexity of the models considered here, we focus exclusively on discrete time formulations.

The tags for each of the N individuals in the model are determined at the time of their introduction to the simulation, either as determined by a procedure that sets the initial population structure at the start of the simulation, or as determined by recruitment processes implemented during the course of the simulation. Recruitment may take place at each time step or may only occur periodically (e.g., t could have units of 3 months while recruitment might occur annually (every 4 time units). On the other hand, the time-varying variables need to be updated at each time step (tick) of the simulation, with separate updating algorithms for each of the different types of variables.

When "Life Stage = Age", the algorithm for updating this variable for each individual is simply part of the algorithm that determines whether or not an individual survives to move up 1 age unit at the end of the age period (essentially, a Leslie matrix process—[104, 105]). On the other hand, the Life Stage variables for holometabolous insects is a sequence of life history stages involving an egg, several larval instars (with moults between them), pupal, and adult stages. The length of time spent in each stage involves an integrative concept called "physiological time" that depends largely on the progression of the temperature profile of the individual over time [106].

In many models, the variable "State" may be confined purely to disease state. In the relatively simple case where individuals are considered to be Susceptible (S), Infected/Infectious (I), and Recovered-with-Immunity (R) a so-called SIR model applies with the most important consideration being how to characterize the pathogen transmission process that determines the probability with which an S individual will become an I individual over the progression of one time step of the simulation (e.g., density-dependent versus frequency dependent transmission or some complex blend of the two: [107]). More complex disease processes may separate out infected-but-not-infectious from an infectious state. There may also be degrees of infectiousness (e.g., superspreader individuals—[108]), or other subtleties to consider (e.g., [109]). Also, other states beyond disease state may be needed, possibly an individual's physiological state, body mass, sex (if individuals change their sex over time), endocrine and reproductive state, as these may impact survival, reproduction, and, in Phase 2 and 3 models discussed below, movement behavior.

Disease Transmission

At the heart of any dynamic infectious disease model are the pathogen transmission processes. Pathogens can be directly transmitted from infectious to sus-

ceptible individuals (i.e. $I \to S$) through direct contact (e.g., those transmitted through bodily fluids) or when these individuals are in close proximity to one another for critical periods of time (e.g., aerosol transmission). More indirect methods apply when individuals come into contact with pathogen contaminated environments. In situations where spatial homogeneity is assumed, one credible way to characterize transmission that may arise from susceptible individuals in social group j ($j = 1, \cdots, J$) being infected with a pathogen from individuals within their own immediate social group, from members outside their social group, or from sources in the environment, is to have a general force of infection term $\rho_j(t)$ for individuals in group j at time t defined additively by an environmental factor $\beta^{\text{env}}(t)$ and a group factor $\beta^{\text{group}}(t)$ that scales with the proportion π_j^{I} of infected individuals in social group j (this is frequency dependent transmission [107]) and a factor β^{pop} that scales with the total number $N^{\text{I}}(t)$ (this is density-dependent transmission [107]) of individuals that are infected in the population at time t (and is assumed time-independent here). In this case, we have

$$\rho_j(t) = \beta^{\text{env}}(t) + \beta^{\text{group}}\pi_j^{\text{I}}(t) + \beta^{\text{pop}}N^{\text{I}}(t) \tag{3}$$

Disease Dynamics

A competing risks approach [110] is most commonly applied to update individuals to account for the continuous processes of natural mortality, disease-related mortality, and disease class transitions (referred to here as parameters μ, α and ρ, respectively, with appropriate age and social membership class super/subscripts designators) as competing processes within the discrete time formulation. The specific formulation follows Getz and Dougherty [111] and Getz et al. [112].

By way of illustration, as a model appropriate [113] for the mongoose example discussed in the next section of this chapter, we present the details of SID disease model (S = susceptibles, I = infectious/infected, D = dead). In this case, over each iteration interval, the deterministic continuous time compartmental model instantiation of an SID process over each time interval $[t, t+1)$ for disease state transition rates $\rho_{a,j}^{\text{S}\to\text{I}}$, $\rho_{a,j}^{\text{S}\to\text{D}}$ and $\rho_{a,j}^{\text{I}\to\text{D}}$ of individuals of age a in social group j takes the form:

$$\begin{aligned}
\frac{dS_{a,j}}{dt} &= -\left(\rho_{a,j}^{\text{S}\to\text{I}} + \rho_{a,j}^{\text{S}\to\text{D}}\right) S_{a,j} \\
\frac{dI_{a,j}}{dt} &= \rho_{a,j}^{\text{S}\to\text{I}} S_{a,j} - \rho_{a,j}^{\text{I}\to\text{D}} I_{a,j} \\
\frac{dD_{a,j}}{dt} &= \rho_{a,j}^{\text{S}\to\text{D}} S_{a,j} + \rho_{a,j}^{\text{I}\to\text{D}} I_{a,j}
\end{aligned} \tag{4}$$

The solutions to these equations over the time interval $[t, t+1]$, if we assume individuals also advance by one age class in each time step (age does not have to be measured in years, but could be measured in quarter-years, months, or even

days, depending on the resolution of the time variable t)

$$
\begin{aligned}
S_{a+1,j}(t+1) &= S_{a,j}(t)e^{-\left(\rho_{a,j}^{\mathrm{S}\to\mathrm{I}}+\rho_{a,j}^{\mathrm{S}\to\mathrm{D}}\right)} \\
I_{a+1,j}(t+1) &= I_{a,j}(t)e^{-\rho_{a,j}^{\mathrm{I}\to\mathrm{D}}} + \\
&\qquad S_{a,j}(t)\left(\frac{\rho_{a,j}^{\mathrm{S}\to\mathrm{I}}}{-\rho_{a,j}^{\mathrm{S}\to\mathrm{I}}-\rho_{a,j}^{\mathrm{S}\to\mathrm{D}}+\rho_{a,j}^{\mathrm{I}\to\mathrm{D}}}\right)\left(e^{-\left(\rho_{a,j}^{\mathrm{S}\to\mathrm{I}}+\rho_{a,j}^{\mathrm{S}\to\mathrm{D}}\right)}-e^{-\rho_{a,j}^{\mathrm{I}\to\mathrm{D}}}\right) \\
D_{a+1,j}(t+1) &= \frac{S_{a,j}(t)}{\rho_{a,j}^{\mathrm{S}\to\mathrm{I}}+\rho_{a,j}^{\mathrm{S}\to\mathrm{D}}-\rho_{a,j}^{\mathrm{I}\to\mathrm{D}}}\times \\
&\qquad \left(\rho_{a,j}^{\mathrm{S}\to\mathrm{D}}-\rho_{a,j}^{\mathrm{I}\to\mathrm{D}})\left(1-e^{-(\rho_{a,j}^{\mathrm{S}\to\mathrm{I}}+\rho_{a,j}^{\mathrm{S}\to\mathrm{D}})}\right)+\rho_{a,j}^{\mathrm{S}\to\mathrm{I}}\left(1-e^{-\rho_{a,j}^{\mathrm{I}\to\mathrm{D}}}\right)\right) \\
&\qquad +I_{a,j}(t)\left(1-e^{-\rho_{a,j}^{\mathrm{I}\to\mathrm{D}}}\right)+D_{a,j}(t)
\end{aligned}
\tag{5}
$$

As a check, the sum of these three equations accounts for all individuals:

$$S_{a+1,j}(t+1)+I_{a+1,j}(t+1)+D_{a+1,j}(t+1)=S_{a,j}(t)+I_{a,j}(t)+D_{a,j}(t)$$

The primary density-dependent components impacting the demography of social individuals—namely competition, typically expressed at the total population level, as well as Allee effects, typically expressed at a low group structure level—can be implicitly incorporated into the population's demographic parameters in various ways. To reduce model complexity, for vertebrates these are typically introduced with respect to the most vulnerable age-classes: namely newborns and senescent adults. Thus, it is not unusual for models to ignore density dependence in all other age classes. In such cases, any age-specific natural and disease-related disease-induced mortality rates, μ_a and α_a respectively apply. In the context of Eq 5, for the age-dependent environmental background transmission parameter β_a^{env}, total population-density transmission parameter β_a^{pop}, and troop frequency-dependent transmission parameter β_a^{group} the various rates featured in Eq. 5 are:

$$
\begin{aligned}
\rho_{a,j}^{\mathrm{S}\to\mathrm{I}} &= \beta_a^{\mathrm{env}}+\beta_a^{\mathrm{pop}}N^{\mathrm{I}}(t)+\beta_a^{\mathrm{group}}\pi_j^{\mathrm{I}}(t) \\
\rho_{a,j}^{\mathrm{S}\to\mathrm{D}} &= \mu_a(t) \\
\rho_{a,j}^{\mathrm{I}\to\mathrm{D}} &= \mu_a(t)+\alpha_a
\end{aligned}
\tag{6}
$$

Recruitment Process

The recruitment processes at time t (this could be at each tick of the simulation clock or with period > 1 if the iteration time is at a finer resolution than the demographic recruitment cycle) generates new individuals as a function of the number of births (or hatchings, etc.) during the time interval $[t, t+1)$ that result in new individuals in the population at time t (i.e., not all newborns survive the first time period). Recruitment is a concatenation of two processes that: 1) determines the actual numbers of births to each gravid female in the

population, and 2.) the survival probability of newborns until the start of the next model iteration point. In general, these two processes are captured by a single "stock-recruitment" function in the parlance of models developed in the field of fisheries management.

In the context of a socially-structured, individual-based model, the following formulation can be applied to the number of individuals recruited into the smallest group for which it is reasonable to assume that the same probability of survival can be applied to all individuals born into that group over the time period $[t, t+1)$ of the simulation. Suppose we have J of these smallest social structures within a population (e.g., troops, prides, herds, etc., or individuals if no social structure is applied). If the function $B_{\mathrm{Id}_i,j}$ designates the expected number of newborns produced by individual Id_i for all Id_i in group j (and do this for $j = 1, \cdots, J$) and if the function $s_{0,j}(t)$ represents the probability that an individual born into group j during the interval $[t, t+1)$ survives until time $t+1$, then:

Number (stochastic or deterministic) of recruits to group j at time t:

$$R_j(t) = \sum_{\mathrm{Id}_i \in \text{group } j} B_{\mathrm{Id}_i,j}(t) s_{0,j}(t), \quad j = 1, \cdots, J \tag{7}$$

Whether this number is stochastic (i.e. drawn from a probability distribution) or deterministic depends on how we implement the functions $B_{\mathrm{Id}_i,j}$ and $s_{0,j}(t)$.

The number of newborns $B_{\mathrm{Id}_i,j}$ that individual Id_i is expected to produce will depend on sex (females only give birth, but parthenogenetic populations could be considered), age/life-stage, and state (epidemiological, physiological, endocrinological), with the details depending on the species. The latter three states will, in turn, be influenced by various ecological factors, but these would only be elaborated and considered in modeling Phase 2 discussed in the next subsection below. In the simplest deterministic model $s_{0,j}(t)$ is just a group independent constant s_0. Whatever the value of $s_{0,j}(t)$ that applies, however, the simplest stochastic equivalent of such a deterministic model is just a binomial drawing that treats the value $s_{0,j}(t)$ as a probability that is applied to all individuals born in the interval $[t, t+1)$: i.e.,

Simplest stochastic implementation given $B_j(t)$ births to group j at time t

$$R_j(t) \sim \text{BINOMIAL}\left[B_j(t), s_{0,j}(t)\right] \quad j = 1, \cdots, J \tag{8}$$

where $x \sim \text{BINOMIAL}\left[n, p\right]$ denotes the number of successes in n independent trials, each succeeding with probability p.

For more complex situations, the survival probability $s_{0,j}(t)$ will be affected by various ecological and social factors, not to mention the disease state of the new born individual—either from possible pathogen transmission at birth, or contracted from an infected group member over the interval $[t, t+1)$. Ecological factors include the availability of resources, competition among social units for those resources, levels of predation, levels of parasites and pathogens, and other

factors that can only be elaborated in detail once the spatial distribution of these factors are included in the model, and the movement of individuals considered as they exploit or encounter these various factors. More of this is discussed below in the implementation of Phase 2. In a Phase 1 model, however, competition for resources, for example, can be incorporated in terms of a survival function that takes total population density at time t into account in a compensatory or over-compensatory manner [114, 115]. Similarly, social factors that induce an Allee effect in the survival of young [116, 117], typically due to phenomenon of groups needing a minimum number of individuals to effectively compete with other troops for resources (food, water, nesting sites, territories etc.) can be incorporated into survival functions taking on a depensatory form [118] at low group numbers.

A simple way to incorporate both depensation (per capita growth declines with declining population size) with regard to troop size J and compensation (per capita growth rate declines with increasing population size) with respect to population size N is to express the survival probability as the product of two switching functions: a switching-on function f_{on} as a function of the number of troops members $J(t)$ (i.e. a function that approaches 0 as troop numbers $J(t)$ drop to zero and approaches 1 as troop numbers become relatively large) and a switch-off function f_{off} as a function of the total population size $N(t)$ (i.e. a function that approaches 1 as total population size $N(t)$ drops to zero and approaches 0 as total population size becomes relatively large. When newborn state only includes its disease state, a new recruits survival probability should be reduced by a factor that reflects the amount of pathogen that newborn individuals are likely to be exposed to over the interval $[t, t+1)$. Thus for example, if a proportion $\pi_j(t)$ of individuals in the troop that are infectious at t, the effects of possible disease in recruited individuals can be taken into account by a function $f_{\text{diseae}}(\pi_J(t))$ that decreases from 1 to some minimal non-negative value as its argument ranges from 0 to 1.

In our formulation of newborn survival, we use the notion of two parameter switching (i.e., sigmoidal) functions f_{on} and f_{off} with switching point $\hat{x} > 0$ and steepness (or abruptness) parameters $\sigma_{\text{on}} > 1$ and $\sigma_{\text{off}} > 1$ respectively (typically, these switching parameters will be around 3-5, or even greater if the switching function is particularly abrupt; [119]):

$$f_{\text{off}}(x) = \frac{1}{1 + x^{\sigma_{\text{off}}}} \quad \text{and} \quad f_{\text{on}}(x) = \frac{x^{\sigma_{\text{on}}}}{1 + x^{\sigma_{\text{on}}}} \tag{9}$$

For these functions, $f_{\text{off}}(0) = 1$, $f_{\text{on}}(0) = 0$, $f_{\text{off}}(1) = f_{\text{on}}(1) = 1/2$ and $\lim_{x\to\infty} f_{\text{off}}(x) \to 0$, $\lim_{x\to\infty} f_{\text{on}}(x) \to 1$.

Newborn survival $s_{0,j}(t)$ (lower case s for survival, upper case S denotes susceptibles) in social group j at time t (recall $a = 0$ is the age index for pups) is given by a maximum pup survival factor $s_0^{\max}$ (assuming $s_{0,j}^{\max} = s_0^{\max}$ for all j). This survival, firstly, is reduced for some force of infection scaling constant $c^{\text{inf}} > 0$ by a factor $e^{-c^{\text{inf}}\beta_0^{\text{group}}\pi_j^{\text{I}}(t)}$ to account for the mortality of newborns as a function of the proportion $\pi_j^{\text{I}}(t)$ of infected individuals in the troop. Secondly,

this survival is increased by a switching-on function of troop size $n_j(t)$ with argument $x = n_j(t)/n^{\text{allee}}$. Thirdly, this survival is reduced by a switching off function of total population size $N(t)$, with argument $x = N(t)/N^{\text{eq}}$, where N^{eq} is a switching location parameter. Thus, it follows that:

$$s_{0,j}(t) = s_0^{\max} \times e^{-c^{\inf}\beta_0^{\text{troop}}\pi_j^{\text{I}}(t)} \times f_{\text{on}}\left(n_j(t)/n^{\text{allee}}\right) \times f_{\text{off}}\left(N(t)/N^{\text{eq}}\right) \quad (10)$$

The surviving new recruits to troop j need to be partitioned into those that are in disease state I and those in S. Following the formulation of Eq. 3, assume that the force of infection on any recruit has a background or external component (i.e. spill over from elements beyond the focal population) β_0^{env}, a density-dependent transmission population-as-a-whole component $\beta_0^{\text{pop}}N^{\text{I}}(t)$, and a frequency dependent social group component $\beta_0^{\text{group}}\pi_j^{\text{I}}(t)$. Thus, the rate at which individuals transfer from S to I over the time interval $[t, t+1)$ is $\beta_0^{\text{env}} + \beta_0^{\text{pop}}N^{\text{I}}(t) + \beta_0^{\text{group}}\pi_j^{\text{I}}(t)$, and we expect the proportion that remain in state S to be:

$$p_{0,j}^{\text{I}}(t) = e^{-\left(\beta_0^{\text{env}}+\beta_0^{\text{pop}}N^{\text{I}}(t)+\beta_0^{\text{group}}\pi_j^{\text{I}}(t)\right)} \quad (11)$$

Social Structure Fission and Fusion

The ecological factors that influence fission (group splitting) and fusion (group merging) in social species is create a tension between the benefits and costs of social group living [120]. The advantages of group living include enhanced predator vigilance, cooperative foraging, and information sharing. The disadvantages of group living include within group competition for resources, increased parasite loads and pathogen transmission facilitated by proximity living (e.g., aggregations of individuals in nest, burrows, etc.), and an increased footprint and visibility of large groups resulting in enhanced detection of groups by predators.

In species ranging from spider monkeys and chimpanzees to elephants and bats, individuals must continuously compromise between nutritional requirements and social needs [121–125]. Ecological factors driving fission include patchy resource distributions (like fruit trees for chimpanzees) where large groups are likely to rapidly deplete these patches resulting in excessive energy spent on movement and exposure to predation during such movements. Ecological factors driving fusion in populations that exploit resources in open habitats or "risky places," (such animals graze in open plains) induces individuals to aggregate, thereby reducing their risk of predation due to the so-termed "dilution effect" (safety in numbers; [126]), as well as benefiting from collective vigilance—the "many eyes effect" [127–129]. An advantage also arises when a few individuals switch groups and transfer genes (out-group mating) and information. There can also be hubs where individuals frequently aggregate for short periods of time (e.g. raven roosts or honeybee clusters fuse to exchange information about food location, then fission to exploit it [130, 131]).

Various approaches have been taken to modeling fission-fusion dynamics, including in an epidemiological context. The most analytical of these has been the formulation of an integro-differential equation (Smoluchowski Coagulation-Fragmentation Equation (see [132]) that describes how a concentration $c(x,t)$

of groups of size x changes over time t. It has been used to analyze the Group Size Distribution (GSD) of large populations such as schools of tuna or herds of African buffalo [133]. Other approaches use mathematical optimization methods. For example, linear programming has been used to demonstrate that a time budget model predicts the upper bound on group size in chimpanzee populations. More particularly, by splitting the troop into smaller foraging parties, chimpanzees can dramatically reduce their daily travel and energetic costs necessary to sustain larger troops in harsh habitats [134]. This results indicates that the temporal dimension of fission-fusion is critical: fissions may be temporary, with subgroups later reuniting, or may become irreversible when social networks cluster to the point where cross-group bonds may disappear.

Network models can be used to capture the social structure of the population through the application of association indices fitted to empirical data [135, 136]. These associations can be generated from direct observation (e.g., following groups of individuals in the field and noting which individuals are within a minimum distance of one another; e.g., in elephants [137]) or using proximity biologging (individuals are fitted with GPs, VHF or PIT tags that log when individuals are within a specific distance of one another; e.g., pit tags mounted on two house sparrow populations, a social weaver population, and a great tit population on three different continents [138]), or using GPS telemetry data (see [139] for a general methods paper to analyse such data). At a cruder level the probabilities of individuals spending time within proximity of one another can be estimated from space-use, home range, or relocation data in which temporal data are explicitly considered [140, 141].

Agent-based models of the group membership of individuals within populations and changes in these groups over time have been used to study fission-fusion dynamics and what types of rules lead to particular social structures. For example, when populations where individuals set out to forage in small groups during part of the diel cycle and nest/roost together during a complementary part of the diel cycle, a set of simple rules might be: "a friendship rule" (prefer to be with long term social partners); "a safety in numbers rule" (show some preference for larger groups over smaller groups when undertaking some particular activity such as resting or grazing), "a thermoregulation rule" (show preferences for larger groups in winter than summer), "a maternal rule" (various options: mothers with young should seek each other out; mothers with young should avoid groups with mature males; etc.). Rules such as these have been shown to predict group size distributions in communally roosting in Bechstein's bats [142] and domestic sheep [143].

In populations where individuals remain with the same group for multiple days, weeks or even months, simple threshold fusion/fission rules may apply. For example, if two groups are each below a fusion threshold then the rule may be that they probably merge. If a group is above a fission threshold (likely to be a least twice as large as the fusion threshold) then the rule may be that it probably splits. These thresholds are likely to be soft rather than hard in the sense that these probabilities increase with decreasing size of the two groups

and the probability that a group will undergo fission increases with its current size. The thresholds themselves are likely to depend on various ecological factors such as the current availability of resources, the current risk of predation, or even levels of parasites in the environment. Such rules are reminiscent of the idea of a "giving up density" in optimal foraging theory [37].

Social structures in populations are known to have a profound effect on epidemic/zoonotic processes and, hence, on zoonoses as well [144–153]. Foremost among these are group size [154], contact networks induced by social behavior [155], the mobility of individuals, and the rates at which individuals make contact or come into close proximity with one another [156, 157]. Thus it is important to take group structure and individual movement characteristics into account when predicting the course of a zoonotic process or when evaluating the risk of pathogen spill overs and possible pandemic outbreaks.

In summary, to assess the risk and control of zoonotic diseases, we need to pay attention to

- **Pathogen Spillover: amplification and dilution effects.** The existence of hubs, such as meat markets, where human contact with wildlife and their pathogens transmitted through bodily fluid or meat consumption greatly amplify the risk of pathogen spillover. On the other hand, alternative or intermediate hosts can sometimes act as a barrier or dilute the risk of pathogen spillover.
- **Population Spatial and Individual Trait Heterogeneity.** Spatial heterogeneity, such as the gathering of individuals at leks, feeding sites, or latrines promotes contact and hence transmission. Similarly, some individuals may possess traits, such as high mobility or gregariousness, that make them superspreaders. These individuals are important to identify and even utilize when applying targeted culling or vaccination actions to control outbreaks.
- **Social Structures.** Most populations exhibit some structure ranging from family groups, herds, troops, packs or prides, to metapopulations. In a network context, these correspond to sparse sets of links between well connected hubs. The spread of disease from one hub to another may be controlled by focusing on strategies that reduce or cut inter-hub (between groups) links.

3.2 Phase 2: Spatial structure

In adding spatial structure to models, we need to account for two orthogonal elements. The first is how to characterize the structure and dynamics of the space over which individuals move. The second is how to characterize the state and movement of the individuals over the space or landscapes of interest. The first typically involves an appropriate discretization of the space into a cellular mapping, or matrix. One then needs to select the key variables representing the properties of each cell, and providing suitable algorithms to update these variables. The second involves developing a set of rules or governing equations that control the movement of individuals. To do this requires selecting key variables to characterize the state of each individual, including the provision of equations for updating an individual's state-variables over time.

Spatio-Temporal Resolution and Process Rates

A key aspect to identifying the ecological drivers of any enzootic or zoonotic process is to select spatial and temporal resolutions that align with the rates of movement of individuals over the landscape, the rates at which pathogens are transmitted within the population, and the rates at which demographic changes unfold in the population. Thus, a fast disease (incubation periods and disease induced deaths measured in days), in short lived populations (sexual maturity within a year, longevity just a few years), that are relatively sessile (home ranges less than hundreds of square meters) such as many viral diseases of rodents [158] or even plague in prairie dogs [159] should be modeled as moving over landscapes whose cellular dimensions are measured in meters rather than hundreds of meters or kilometers, with a simulation time step iteration in days for disease transmission and hours for movement patterns. On the other hand relatively slow progressing diseases in large mammals, such as bovine TB in African buffaloes [160] or chronic wasting disease in white-tailed deer [161] can be simulated on landscapes that have a pixelation resolution of hundreds of square meters, temporal rates of disease transmission measured in weeks or months, although daily movement rates may be needed to capture the contact network at an appropriate level of resolution.

Landscapes

The various factors affecting demographic and disease processes in a population may have different levels of spatio-temporal resolutions at which they can be represented and at which they also affect key dynamics and processes [162]. Thus, for example, precipitation may have a resolution of tens of square kilometers, while changes in resources, risk and terrain influencing movement may occur at the scale of meters [163]. In trying to put all these factors together on a common landscape requires that either information at the finest levels of resolution is averaged out at a coarser level of resolution or information at a coarse level of resolution is interpolated at a finer level of resolution. The resolution will also need to take into account the mobility and speed of individuals moving over the landscape and the spatial resolution at which individuals make decisions on whether to stop or move on.

In some cases the landscape itself may be hierarchically structured, as in the case of migratory animals—migration itself having a profound influence of zoonotic processes [164]. For example, in the case of migratory birds, the landscape may be represented by a network of nodes consisting of breeding ground nodes, overwintering nodes, and stopover nodes [165]. The connectors of the nodes would have distances associated with them. They may also include other information such as temperature or movement-resistance information that would affect how long it takes individuals to traverse nodes and how much energy might be expended in the process. The nodes themselves, if small stopover sites, might be treated as a single cell with regard to resource availability or other salient ecological and environmental factors. If nodes represent relatively large areas,

such as breeding or overwintering grounds, then these in turn may be pixelated and be treated as spatially heterogeneous.

Maps

The following are various maps that might be combined to define the state of each cell within the pixelation of the landscape upon which the population is located for the time period of interest.

- **Topography and elevation**: Various government agencies, such as U.S. Geological Survey (USGS), provide highly detailed topographic and climate maps. Topography and elevation have a major effect on temperature and precipitation and hence on habitat structure and the ecology of their resident populations. Thus it is not surprising that topography, for example, has been shown to be an important driver of arthropod vectored disease such as malaria [166].
- **Physical Structure**: The physical structure of the landscape includes soil type and vegetation structure, particularly relevant as they may pertains to temperature regulation (measured by leaf area index [167], canopy cover or sky view factor [168]), provide protection or cover [169], furnish nesting or roosting sites (snag density and diameter at breast height measures, [170]), provide access to water, and act as barriers to or facilitation of locomotion through least energy expended pathways [171]. Measures related to physical structure can be extracted through field surveys, by planes using LIDAR or 3-d photography, or inferred from satellite imagery [172, 173].
- **Resources**: Field, aerial survey, and remote sensing satellite data can be used to map vegetation [174] and other relevant resource structures (e.g., fruiting tree, [175]) at a landscape level. Resource selection functions can be generated from these data [176] and then used to develop algorithms for generating the movement of animals across these landscapes [177].
- **Disease transmission**: A better understanding of the distribution of animals and their movement across landscapes can be gained by overlaying pathogen landscapes [178], including parasite distributions [20] and environmental pollutants onto the various maps mentioned above [179] to develop algorithms that best explain how animals move across landscapes [180]. In particular, these landscapes can assess the risk of individuals being exposed to a sufficient dose of pathogen to become infected [181, 182].
- **Population interactions**: Creating maps that affect the ecological interactions of individuals, either through competition, or through predation, can be applied at two fundamentally different levels. The first is a real time map of where animals currently are on the landscape. Such a map can be used for predator avoidance [183] or to account for interference competition [39]. The second is to map out an individual's perception of a risk of predation either because of the existence of landscape structures that may be concealing predators, or information regarding the possible proximity of predators (dung, memory) in the creation of a landscape-of-fear [19, 184].

> The landscape of fear may be inferred from vigilance behavior, time allocation (controlling for other variables such as resource availability), herd size or herd compactness, or giving-up densities (the amount of resources remaining from a depletable food source following harvest by the forager) [19].

The information contained in all these various landscapes can be concatenated into a single cellular state vector that complements the individual state vector representation that can be created from the variables listed in Eq. 2. This state vector, then would be made of the variables, many time varying, associated with the landscape's properties relevant to the species of interest and associated with topographic (L^{topo}), physical structure of the habitat (L^{phys}), various resource distributions (L^{res}), resistance to movement (L^{move}), landscapes of fear and disgust combined into a single landscape of peril (L^{peril}), and a landscape of occupants (L^{occ}). In this case the k^{th} cell ($k = 1, \cdots, \#\text{cells}$; a mapping of a two dimensional array onto the integers will be needed to label cells using a single index k) will have the representation

$$\text{Cell}_k(t) := \left\{ L_k^{\text{topo}}(t), L_k^{\text{phys}}(t), L_k^{\text{res}}(t), L_k^{\text{move}}(t), L_k^{\text{peril}}(t), L_k^{\text{occ}}(t) \right\} \tag{12}$$

Time series data will be needed to update time-varying variables including ones the are influenced by others. For instance, resource levels may be driven by external environmental factors (e.g., precipitation, temperature) but will also need to be updated by algorithms computing how much of the resource is extracted by individuals that occupy the various cells as they move across the landscape.

Movement and Disease

The way animals move over landscapes determines the probabilities of coming within proximity of other infected individuals or infectious environmental "hotspots" for pathogen transmission to occur. Detecting the disease state of animal from its movement behavior is a new area of investigation made possible by rapidly advancing technologies for collecting movement data [185] and new ways for segmenting movement paths into basic movement elements [180, 186, 187]. If such detections can be easily implemented using remote sensing technologies that feed movement data directly into AI models for real time assessment, then sick individuals can be quickly identified for collection of samples, and possible capture, isolation, treatment, vaccination or culling before they are able to transmit disease to others [188–190].

Since this is a new endeavor, only a few studies in the literature report the use of movement data—both accelerometer [191, 192] and relocation type data—to infer the disease status of individual animals. For example: accelerometer data has been used to identify individual wild boars that may be infected with swine fever [193]; RFID (Radio Frequency Identification) tag signals from cows fitted with battery-free ear-tag transponders that monitored their peripartum walking activities within a barn was use to detect subclinical ketosis and clinical diseases in dairy cows [194]; and, relocation data collected from horned oryx in Chad

and analysed using Markov chain methods [195, 196], has been used to identify individuals afflicted with Rift Valley Fever, Peste des Petits Ruminants and Babesiosis [197].

3.3 Phase 3: Anthropogenic feedback

Phase 2 of our proposed PEARLS framework deals with the trajectories of natural systems in the context of an environment with anthropogenic factors where these latter factors provide a static background on which the natural system dynamics unfold. In Phase 3, we consider the case where the anthropogenic background is itself dynamic and intertwined with the dynamics of the natural system under consideration. The study of coupled human and natural systems (CHANS) dynamics and the development of models to investigate these complex systems [198] took off in the early 2000s with the establishment of a designated "Dynamics of Coupled Natural and Human Systems" funding program. This program promoted the formation of interdisciplinary teams of modelers, ecologists, geographers, economists, and sociologists to investigate how human engineering of landscapes and management of wildlands influenced the trajectories of natural systems. Teams also evaluated how these natural systems interact with social systems in terms of resource exploitation, wildlands recreation, and facilitation and mitigation of human-wildlife conflict and interactions [199].

At the largest spatio-temporal scales and hence the most aggregated level of analysis are coupled human-Earth system models that typically use systems of ordinary and partial differential equations with a strong foundation in mathematical physics [200]. At finer spatio-temporal scales—i.e. the analysis of geographically localized systems, both systems dynamics and ABM approaches [201] have been used. The former has been applied to systems where resource utilization is the focus of the analysis [202]. The latter, however, is particularly appropriate when the focus is on zoonoses and pathogen spillover, ABM approaches provide a way to focus on the role of individuals in spreading zoonotic diseases.

ABMs have been extensively applied in the public health sector [203] and to human epidemiology [204, 205] and the context of zoonoses involving domestic livestock [206] but less so to zoonoses involving wild animal populations. In this latter context, drivers of the landscape of peril become particularly important in mediating contact between human and wild animal populations. The details of the factors that influences these contacts are highly species specific and thus we leave a fuller discussion of potential factors to the human-mongoose interaction case study presented in the next section.

4 PEARLS Applied to Banded Mongoose-*Mycobacterium mungi* system

The banded mongoose - *M. mungi* system in northern Botswana illustrates why pathogen transmission in human-modified landscapes cannot be understood

through any single ecological driver. The system combines a territorial group-living host; a pathogen transmitted through socially directed olfactory communication; strong spatial and temporal variation in human association, resource availability, and mortality risk; and measurable relationships between landscape conditions, host state, behavior, movement, contact, and pathogen exposure. Therefore, it provides a real-world example of why an integrated framework such as PEARLS is needed to strengthen the mechanistic understanding and predictive capacity for the investigation and control of zoonotic disease transmission involving wildlife hosts.

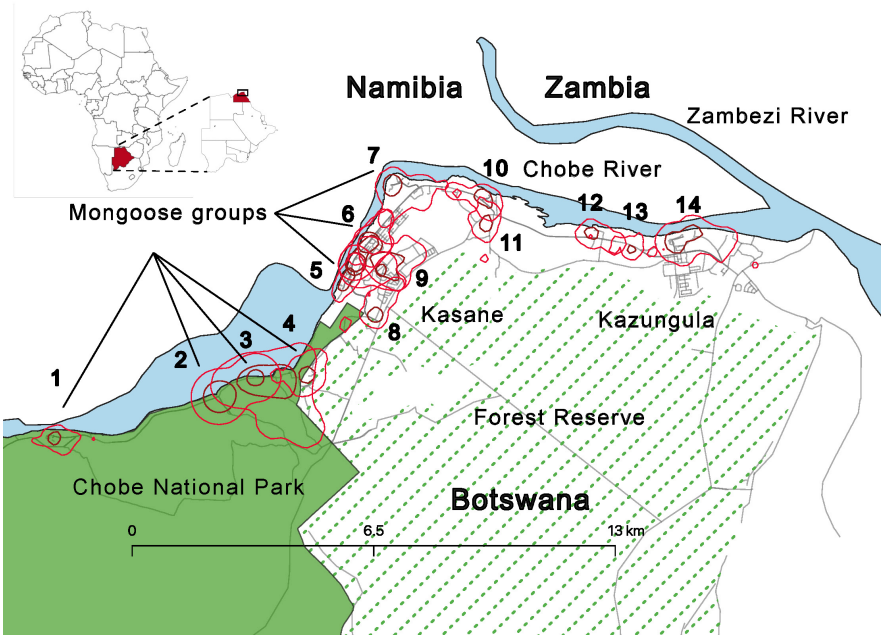

Fig. 2. Spatial distribution of banded mongoose (*Mungos mungo*) troops across protected, lodge, urban, and residential landscapes in Kasane and Kazungula in northern Botswana. Kernel utilization distributions are shown for troops monitored over the last ten years, with outer contours representing the 95% home range and inner contours representing the 50% core area. The map illustrates the distribution of troop ranges across contrasting land uses and spatial heterogeneity in peril, environmental conditions, anthropogenic influences, and resources experienced by this population.

4.1 Behaviorally mediated transmission links landscape use directly to infection risk

Banded mongooses (*Mungos mungo*) live in stable, territorial social groups or troops [75]. In northern Botswana, troop home ranges extend across protected,

lodge, urban, and residential landscapes, creating a pronounced variation in human association and ecological conditions experienced by different troops across the landscape [33] (Fig. 2). This population is naturally infected with *M. mungi*, a member of the *M. tuberculosis* complex [76, 207]. Throughout this discussion, *M. mungi* is treated as a microparasite within the broader ecological parasite–host framework.

The pathogen is shed in urine and anal-gland secretions used during normal olfactory communication and appears to enter susceptible animals through lesions in the nasal planum or skin [76, 78]. Scent marks are deposited on conspecifics and environmental substrates and are subsequently investigated or overmarked by other mongooses. These behaviors provide opportunities for both within-troop and between-troop exposure without requiring direct physical contact between all transmitting and susceptible individuals [76, 208].

Recent modeling work further demonstrates that socially directed, environmentally mediated contacts can contribute substantially to pathogen spread, particularly when infectious secretions persist at sites used sequentially by different individuals or groups [208]. Thus, mongoose movement, scent marking, investigation of marks, den use, and environmental contact are not merely correlates of infection but are mechanistically connected to transmission [76, 26].

Several of these transmission-relevant behaviors can be quantified directly. Camera-trap studies have linked vigilance and olfactory communication to landscape context [209], and triaxial accelerometry has been used to classify behaviors including bipedal vigilance, running, and scent marking in this population [210]. The mongoose-*M. mungi* system therefore provides an unusually direct link between spatial conditions, observable behavior, environmental contact, and pathogen transmission.

4.2 Human-modified landscapes create simultaneous risks and opportunities

Human association alters the distribution and predictability of resources, refugia, den sites, disturbance, and mortality risks encountered by mongooses. These effects are not spatially uniform, nor do they operate independently of season. Human-associated troops showed more concentrated space use during the dry season, when predictable anthropogenic resources modified patterns otherwise expected under natural resource limitation [33].

Anthropogenic structures also alter den ecology. Human-made dens persist longer than natural dens, and use of a den by a troop other than the resident troop was recorded only at anthropogenic den sites. Such sequential use occurred at 6% of the 308 dens examined [26]. Although relatively uncommon, this behavior is epidemiologically important because persistent den sites can connect otherwise territorial troops through indirect environmental contact.

Urbanization also influences connectivity among troops. Genetic analyses identified greater dispersal and gene flow among urban troops, with all probable migrants in that study originating from troops within the urban landscape [211]. Host state modifies this pattern: mongooses showing clinical disease or injury

were less likely to disperse than healthy individuals [212]. Population connectivity therefore emerges from an interaction between landscape structure and individual condition rather than from either factor alone.

Anthropogenic food resources provide another example of interacting risk and opportunity. Refuse can reduce the immediate cost of obtaining food by concentrating predictable resources, but foraging at garbage sites was associated with increased within-troop aggression. Aggression, in turn, predicted injury, and visibly injured animals had a substantially greater incidence of clinical tuberculosis during the study period [34]. Because *M. mungi* can enter through damaged skin and the nasal planum, resource concentration may increase disease risk through a pathway involving competition, aggression, injury, and subsequent pathogen invasion [34, 76, 78].

These findings show why human-associated environments cannot be classified simply as either safe or dangerous. The same anthropogenic feature may provide food, water, refuge, or persistent den sites while simultaneously increasing aggregation, aggression, environmental contamination, or contact among troops [26, 33, 34, 211, 212, 209].

4.3 Landscape context and host state alter the transmission consequences of behavior

The relationship between risk and pathogen-relevant behavior is context dependent. Vigilance interacts with both landscape type and the number of mongooses present to shape the frequency of olfactory communication [209]. In lodge environments, vigilance was positively associated with olfactory behavior, whereas in urban and protected landscapes, where different sources of human- or predator-associated risk occur, the relationship was negative [209].

Consequently, increased vigilance cannot be interpreted universally as either increasing or decreasing transmission potential. Its epidemiological effect depends on landscape context and on the other behaviors with which vigilance is associated. A patch in which animals remain alert while continuing to scent mark may have a very different transmission consequence from one in which vigilance suppresses marking, investigation, or patch residence. These landscape-dependent relationships demonstrate how apparently similar levels of risk responsiveness can generate contrasting contact and transmission outcomes.

Host state adds another layer of complexity. Studies of fecal glucocorticoid metabolites showed that food limitation, rainfall, access to concentrated anthropogenic resources, and reproduction explained more variation in adrenocortical activity than coarse measures of predation risk such as canopy cover or group size [213, 214]. Glucocorticoid elevation was strongly seasonal, with its timing and magnitude moderated by access to anthropogenic food resources [214].

These findings are important because physiological stress cannot be treated as a simple proxy for perceived predation risk. Internal state may reflect nutritional limitation, reproduction, social competition, disease, injury, or several interacting challenges. In turn, state can influence movement, foraging decisions,

vigilance, aggression, susceptibility to infection, and the costs and benefits of occupying particular patches.

Disease may also modify behavior after infection. Diseased or injured mongooses were less likely to disperse [212], but visibly diseased animals were not avoided by troop members during normal social interactions [215]. Infection may therefore reduce one form of connectivity such as dispersal between troops without eliminating close social or environmental contacts within the troop. Disease state, movement, and contact structure must consequently be represented as interacting rather than interchangeable processes [213, 214, 212, 215].

4.4 Why single-driver models are unlikely to predict transmission across landscapes and seasons

The published mongoose research demonstrates that transmission emerges from interactions among peril, environmental conditions, anthropogenic influences, resources, and host state. Peril includes natural predation risk, human-associated disturbance and mortality, social aggression, injury, and the risk of exposure to *M. mungi*. Environmental conditions include seasonality, vegetation structure, visibility, rainfall, and the physical characteristics of den and foraging sites. Anthropogenic influences include urban development, refuse, built structures, roads, tourism facilities, and the spatial distribution of human activity. Resources include natural forage, concentrated food waste, water, den sites, and refugia. Internal state includes nutritional condition, physiological stress, injury, reproductive demands, and infection status.

These components do not operate independently. Anthropogenic resources may reduce food limitation but increase aggregation and aggression. Persistent human-made dens may provide secure refugia while also creating long-lived environmental contact sites. Urban landscapes may increase dispersal and gene flow, whereas disease and injury reduce the likelihood of dispersal. Group size and spatial concentration may alter vigilance, competition, marking behavior, and the number of direct and indirect contacts. Seasonal resource limitation may affect physiological state at the same time that it alters movement, habitat use, and exposure to contaminated sites [26, 33, 34, 209, 213, 214, 211, 212, 215].

A model based only on resources could identify locations where mongooses aggregate but fail to predict how aggression, injury, vigilance, or scent marking alter transmission within those locations. A risk-based model could identify avoidance or refuge behavior but fail to explain why apparently favorable anthropogenic sites can become transmission hotspots. A state-based model could identify differences among individuals but omit the landscape processes that create those states and structure their contacts. A model based simply on human presence would be unable to distinguish between provisioning, tolerance, disturbance, persecution, refuge creation, and other human actions with contrasting ecological consequences.

The relative importance and direction of these mechanisms may therefore change across land types, seasons, troops, and individual states. Predictive fail-

ure may arise not because a particular driver is unimportant, but because its effect depends on its interaction with other components of the system.

4.5 PEARLS connects the missing causal layers

The mongoose system demonstrates that the effects of human activity cannot be represented adequately by a single measure of human presence. Human perceptions and decisions can generate responses ranging from tolerance and provisioning to deterrence, disturbance, and persecution. Within PEARLS, the spatial distribution of risks and opportunities as perceived by humans is represented by LOP_0. The behaviors and decisions arising from those perceptions then modify environmental, anthropogenic, resource, and peril-related conditions across the landscape.

The existing mongoose literature primarily documents the ecological consequences of human-modified environments rather than directly representing the human perceptions and decisions that produce them [26, 33, 34, 209, 213, 214, 211, 212, 215]. This missing causal layer is one reason that the explicit inclusion of LOP_0 is important. Reciprocal effects of mongoose behavior on human perception or activity may also occur, but such feedback is conditional and need not be assumed in every formulation.

PEARLS does not presume that every component will be equally influential in every setting. Rather, it provides a common structure within which the contribution of peril, environmental conditions, anthropogenic influences, resources, and internal state can be compared, their interactions evaluated, and their effects followed through movement and behavior to contact and pathogen transmission. By preventing these processes from being omitted or incorrectly treated as independent, the framework has the potential to strengthen mechanistic inference and improve prediction across changing landscapes and seasons.

5 Discussion and Conclusion

The approach discussed in this chapter suggest ways to advance population and community ecology by establishing a mechanistic framework that integrates human ecology into spatial ecological models. Although LOP approaches in the context of landscapes-of-fear are well developed for predator–prey systems, they have not been mechanistically integrated with how human perceptions of risk and opportunity (LOP_0) generates behavioral, spatial, and trophic structures that reshape wildlife populations through LOP_1 considerations and, hence, zoonoses. By explicitly modeling human perception—how people interpret wildlife cues, evaluate danger, and modify their own activities and movement along provisioning–persecution gradients, our suggested approach introduces a potentially critical causal layer shaping wildlife behavior, encounter networks, and parasite transmission across heterogeneous land types.

The approach proposed in this chapter is explicitly comparative and mechanistic, evaluating competing pathways of parasite transmission, including LOP_1-dominated, resource aggregation–driven, state-dependent, and mixed-interaction

models. It integrates long-term behavioral and demographic data, giving-up densities in experimental food patches, high-resolution movement and accelerometry, experimental manipulations, multi-season occupancy modeling, and hierarchical behavioral models within a novel multi-layer agent-based framework. These components allow direct tests of how human-perceived fearscapes and wildlife LOPs jointly structure wildlife population processes, social connectivity, multispecies interactions, and host–parasite dynamics that scale to population and community levels. Overall, this framework could be developed further to help predict or anticipate potential disease spillover to humans [188].

Although the mongoose (*M. mungi*) system is biologically specific, the ecological mechanisms linking LOP_1 structure to host state, movement, social–spatial contact networks, and parasite exposure and transmission reflect general processes that operate across diverse host–parasite systems. The tractability of this territorial, group-living species—spanning urban to protected landscapes—allows direct measurement of these mechanisms, enabling rigorous hypothesis testing and model validation. This, in turn, supports the development of broadly transferable theoretical constructs, mechanistic models, and analytical tools that extend well beyond this system, providing general insights relevant to wildlife populations, human–wildlife interfaces, and ecological communities undergoing anthropogenic environmental change.

Integrating human activity and perceptions into LOP-based behavioral and spatial models represents a potentially transformative advance. It links sociocultural and ecological processes in a way that yields tractable, testable predictions about host–parasite interactions, population connectivity, and community structure. The resulting theory, analytical tools, and modeling framework are designed to be generalizable to human-influenced ecosystems globally, expanding the conceptual foundations of population and community ecology and enabling mechanistic forecasts of ecological change in human-modified landscapes.

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