OIKOS

Research

Moving infections: individual movement decisions drive disease persistence in spatially structured landscapes

Cédric Scherer, Viktoriia Radchuk, Mathias Franz, Hans-Hermann Thulke, Martin Lange, Volker Grimm and Stephanie Kramer-Schadt

C. Scherer (https://orcid.org/0000-0003-0465-2543) ✉ (cedricphilippscherer@gmail.com), V. Radchuk, M. Franz and S. Kramer-Schadt, Leibniz Inst. for Zoo and Wildlife Research (IZW), Alfred-Kowalke-Str. 17, DE-10315 Berlin, Germany. SKS also at: Dept of Ecology, Technische Univ. Berlin, Berlin, Germany. – H.-H. Thulke, M. Lange and V. Grimm, Helmholtz Centre for Environmental Research–UFZ, Leipzig, Germany.

Understanding host–pathogen dynamics requires realistic consideration of transmission events that, in the case of directly transmitted pathogens, result from contacts between susceptible and infected individuals. The corresponding contact rates are usually heterogeneous due to variation in individual movement patterns and the underlying landscape structure. However, in epidemiological models, the roles that explicit host movements and landscape structure play in shaping contact rates are often overlooked.

We adapted an established agent-based model of classical swine fever (CSF) in wild boar *Sus scrofa* to investigate how explicit representation of landscape heterogeneity and host movement between social groups affects invasion and persistence probabilities. We simulated individual movement both phenomenologically as a correlated random walk (CRW) and mechanistically by representing interactions of the moving individuals with the landscape and host population structure.

The effect of landscape structure on the probability of invasion success and disease persistence depended remarkably on the way host movement is simulated and the case fatality ratio associated with the pathogen strain. The persistence probabilities were generally low with CRW which ignores feedbacks to external factors. Although the basic reproduction number R_0, a measure of the contagiousness of an infectious disease, was kept constant, these probabilities were up to eight times higher under mechanistic movement rules, especially in heterogeneous landscapes. The increased persistence emerged due to important feedbacks of the directed movement on the spatial variation of host density, contact rates and transmission events to distant areas.

Our findings underscore the importance of accounting for spatial context and group size structures in eco-epidemiological models. Our study highlights that the simulation of explicit, mechanistic movement behaviour can reverse predictions of disease persistence in comparison to phenomenological rules such as random walk approaches. This can have severe consequences when predicting the probability of disease persistence and assessing control measures to prevent outbreaks.

Keywords: agent-based model, classical swine fever, disease persistence, host–pathogen dynamics, landscape structure, movement ecology

Introduction

The ability of a disease to establish and persist in a host population depends on sufficient contacts between susceptible hosts and the infectious source which can either be an infected individual in the case of direct transmission or an environmental reservoir or intermediate vector of the pathogen. Contact rates among individuals are thus a key parameter for predicting the spread and persistence of directly transmitted infectious diseases (Altizer et al. 2003, Craft 2015, Arthur et al. 2017, Daversa et al. 2017). Even within species, contact rates exhibit remarkable heterogeneity due to individual variation in movement behaviour (Fraser et al. 2001, Morales et al. 2010, Jeltsch et al. 2013) and due to modular organization of groups (Craft 2015, Sah et al. 2017). In concert with a species' motion and navigation capacity, internal factors such as sociability or behavioural decisions and external factors such as the abundance of resources, shelter, conspecifics or predators act as drivers of the movement decisions of individuals (Nathan et al. 2008). The emerging individual movement paths determine spatiotemporal heterogeneity in host density and contact rates, with potential consequences for the invasion success and the persistence of diseases (Hess et al. 2002, Real and Biek 2007, McCallum 2008, Paull et al. 2012, Craft 2015). Consequently, several recently published frameworks highlight the need to include mechanisms of the host's movement ecology (Nathan et al. 2008) when analysing and simulating dynamics of wildlife diseases (Riley et al. 2015, Boulinier et al. 2016, Daversa et al. 2017, Fofana and Hurford 2017, Sih et al. 2018). Such a mechanistic approach would advance our understanding of how variation in host contact rates in interactions with the landscape heterogeneity affect the dynamics of pathogen transmission in a more realistic setup (McCallum 2008, Daversa et al. 2017, Fofana and Hurford 2017).

Several theoretical studies have investigated the effect of host population structure and contact rates on disease dynamics. However, most approaches model host movement implicitly assuming that the pathogen can spread to any susceptible host with constant rates globally, known as 'homogeneous mixing' (Anderson and May 1991, Grenfell et al. 1995), or depending on the distance from the infectious source (Sato et al. 1994, Keeling et al. 2001). In models with implicit movement, pathogen persistence has been found to be favoured by fragmentation of the population (Dye and Hasibeder 1986, Bolker and Grenfell 1995, Hess 1996, Lloyd and May 1996, Foley et al. 1999, Rozhnova et al. 2014), given a sufficiently large host population exceeding the 'critical community size' (Bartlett 1960, Keeling and Grenfell 1997) and a sufficient rate of pathogen exchange between patches (i.e. distant populations or groups) relative to the infectious period (Keeling and Rohani 2002, Cross et al. 2005). The assumptions of these models may be reasonable in the case that all individuals behave the same way but real systems rarely exhibit homogeneous contact rates and these differences could be critical for the dynamics and persistence of diseases (Bolker and Grenfell 1995, Craft 2015, Sah et al. 2017). Furthermore, feedbacks of the underlying landscape on host movement and pathogen spread are often simplified as being homogeneous or divided in suitable patches and unsuitable matrix (Fig. 1).

Integrating individual movement patterns into epidemiological models allows us to focus on bottom–up processes to understand how differences at the individual level such as movement behaviour affect higher-level processes, namely emerging disease dynamics, in a mechanistic way (Baiser et al. 2013, Jeltsch et al. 2013, Radchuk et al. 2019a). In this context, network models are flexible approaches to relax the assumption of homogeneous mixing and to account for differences on the individual level that arise due to heterogeneity in behaviour, social organization and landscape structure (Bansal et al. 2007, Craft and Caillaud 2011, Ferrari et al. 2011, Godfrey 2013, Jacoby and Freeman 2016). Network models summarize phenomenological movements and can be analysed with regard to network topologies that facilitate or impede the spread of a pathogen among patches that are either host individuals or habitat patches (Ferrari et al. 2006, Fortuna et al. 2009, Craft et al. 2011, Sah et al. 2017). However, network approaches traditionally depict static edges, and connecting the temporal dynamics of real systems with network models is challenging (Ferrari et al. 2011), but first approaches of dynamic networks to capture the temporal dynamics of the spatial and social properties of the individuals are a promising new avenue (Jacoby and Freeman 2016, Sih et al. 2018). Another option are spatially explicit agent-based models (ABMs) simulating individual movement decisions and behaviour that are a means to capture the temporal and spatial variation (Grimm and Railsback 2005, Coulon et al. 2015, DeAngelis and Diaz 2019). Furthermore, ABMs allow the explicit simulation of barriers or low-quality habitat in the landscape that may prevent movement as well as contact rates and transmission events while hosts move through the landscape (Daversa et al. 2017). However, even though ABMs offer the opportunity to include host movement mechanistically, most epidemiological studies so far have used phenomenological approaches by imposing observed patterns, using random walk and correlated random walk approaches (Jeltsch et al. 1997, Nunn et al. 2014, Tracey et al. 2014, Belsare and Gompper 2015).

The effect of directed movement and spatial heterogeneity on host infection risk and disease persistence are not trivial to predict due to emerging variation of contact rates between patches. Segregation or clustering of high-quality patches may reduce the persistence probability due to decreased patch-to-patch contact rates in the first case, or favour persistence under conditions of high exchange rates as found in metapopulation models (Hess 1996, Grenfell and Harwood 1997, Hanski 1997). These effects could be further intensified by individual movement of host animals towards specific areas such as high quality patches in terms of resources, shelter or mates, which may either allow for recurrent infections to neighbouring patches sensu source–sink dynamics or quarantine the infection in these areas
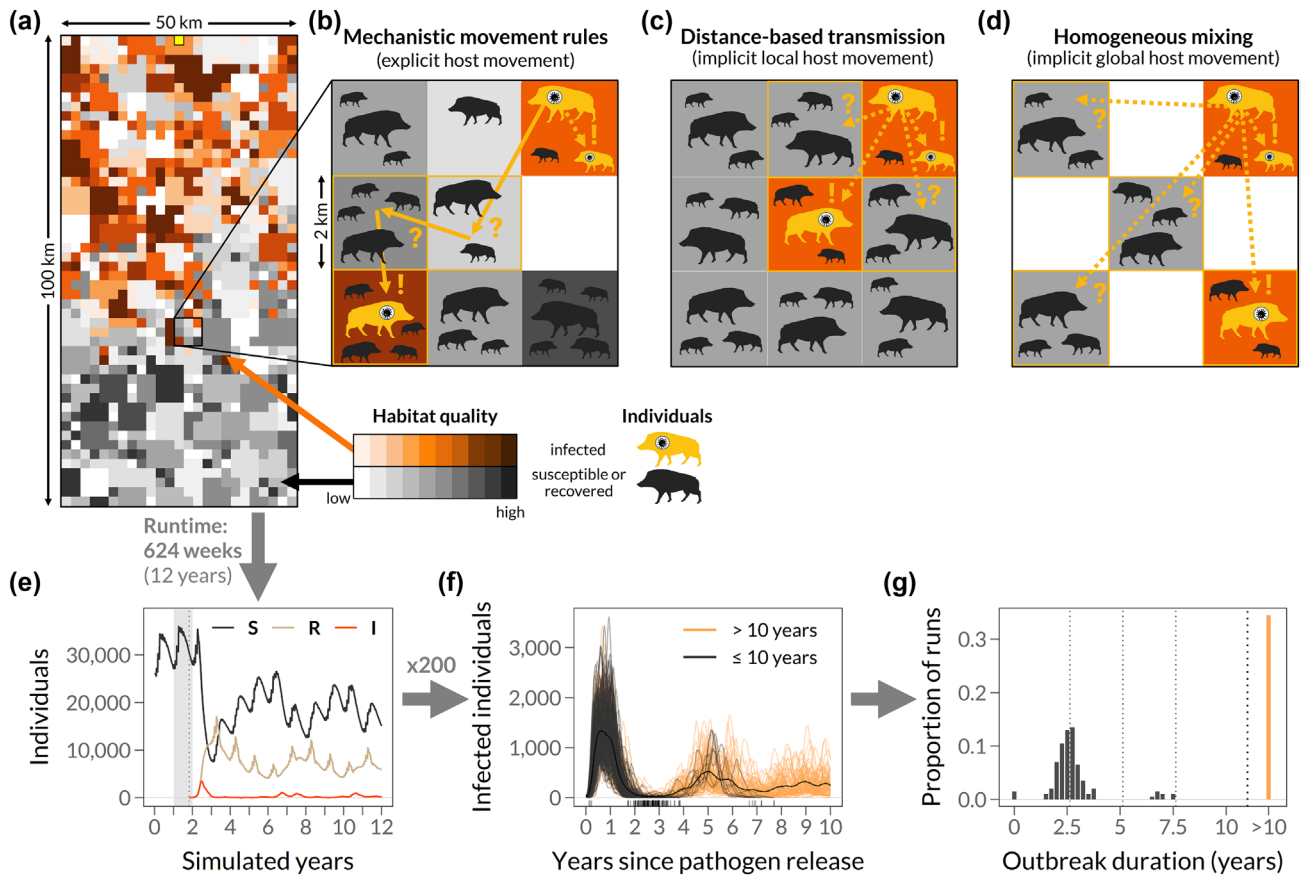
Figure 1. Schematic representation of the agent-based model, possible approaches to simulate movement in epidemiological models and the analytical workflow. (a) Snapshot of the spatially explicit agent-based model with explicit host movement used in this study. The landscape represents 100×50 km (50×25 patches) of heterogeneous wild boar habitat. Colour intensity represents the habitat quality of each home range with consequences for the overall capacity. While grey coloured patches do not host infected individuals (i.e. only contain susceptible or immune individuals), orange coloured patches are currently hosting one or more infected individuals. The yellow patch in the upper row of the landscape indicates the release area of the pathogen. (b–d) Possible modelling approaches to simulate movement in epidemiological models: (b) Explicit movement as simulated in this study. For one infected boar we demonstrate that inter-patch movement is simulated explicitly (straight lines) and movement within the home range is assumed to be implicit (dashed lines). The '?' and '!' indicate possible transmission events to all individuals occupying the visited patches (yellow borders, '?' = no transmission, '!' = new infection). (c) Schematic representation of distance-based transmission (here depicted as 'neighbourhood infection' since the maximum distance of transmission is set to 1 patch) in a homogeneous landscape. Movement to neighbouring patches is assumed to be only temporary (i.e. individuals always return to their home range patch), hence only spread of the pathogen is modelled. This individual-based setup is comparable to metapopulation approaches with distant-dependent transmission kernels. (d) Schematic representation of homogeneous mixing in a binary landscape (grey and orange = habitat, white = matrix) where all individuals are equally at risk to get infected while movement is often assumed to be implicit and only spread of the pathogen is modelled. This individual-based setup is comparable to classical metapopulation approaches. (e) Simulations were run for 624 weeks (= 12 years) with the pathogen being released in the second year (grey area) to allow the pathogen to spread for at least 10 years. We recorded the first and last week of each outbreak together with numbers of each health status over time. The dotted line indicates the first week of the exemplary outbreak simulation (movement rule: competition-driven movement, landscape scenario: small random clusters). (f) For all 200 simulation runs per combination of movement rule (correlated random walk, habitat-dependent movement and competition-driven movement) and landscape scenario (homogeneous and three levels of spatial heterogeneity), we recorded the first and last week of the outbreak, and classified the runs as either 'non-persistent' (less than 10 years since pathogen release; grey) or 'persistent' (more than 10 years; orange). Additionally, we investigated the proportion of persisting runs based on a 2.5-, 5- and 7.5-year threshold (small dotted lines). (g) Based on this information, we calculated persistence probabilities using a 10-year threshold (black dotted line) as well as three shorter time periods (2.5-, 5- and 7.5-year thresholds, grey dotted lines).

(Grenfell and Harwood 1997, Holt and Hochberg 2002, Paull et al. 2012, Faust et al. 2018).

Here, we modified an established agent-based model (Kramer-Schadt et al. 2009, Lange et al. 2012a, b) to investigate the effects of individual movement behaviour and landscape

heterogeneity on disease dynamics, namely on rates of contact and transmission as well as the probability of invasion success and disease persistence. We simulated an economically important livestock disease (classical swine fever, CSF) infecting a socially structured host, the wild boar *Sus scrofa*. In the previous

model versions infections were based on transmission probabilities for individuals within and between groups ('neighbourhood infection' with implicit host movement; Fig. 1). We extended the model to deal with age- and sex-dependent differences in movement behaviour and thus allowed for the emergence of individually differing contact probabilities as it is common in wild boar and many other social wildlife species (Pepin et al. 2016, Sah et al. 2017). To explore the interplay of movement behaviour and the underlying landscape heterogeneity, we ran simulations using four landscape scenarios with different spatial configurations of habitat quality representing local carrying capacities in combination with three types of assumed movement rules. The following types of movement were modelled: phenomenological description of movement (i.e. correlated random walks with a general tendency to continue in the same direction while ignoring external effects such as habitat quality) and mechanistic movement decisions related to the underlying landscape or conspecifics' density.

We expected to find an effect of landscape structure per se on the probability of invasion success and disease persistence. We predicted a lower probability of successful invasions in heterogeneous landscapes due to the spatial variation in host density according to the theory of the 'critical invasion threshold' (Anderson and May 1991, Lloyd-Smith et al. 2005a, Keeling and Rohani 2008). Once the disease is established, homogeneity of patch quality in space should lead to relatively panmictic and fast dynamics, facilitating rapid face-out through exhaustion of the susceptible pool while intermediate levels of clustering might favour disease persistence due to asynchrony between different areas (Grenfell and Harwood 1997, Keeling and Grenfell 1997, Hagenaars et al. 2004). High levels of clustering, however, should lead to rapid fade-outs and thus reduce persistence probabilities due to barrier effects and structural trapping (McCallum and Dobson 2002, McCallum 2008, Rees et al. 2011, Sah et al. 2017). Furthermore, we predicted higher chances of persistence when the explicit host movement is simulated as correlated random walks due to higher exchange of infectious agents since movement is independent of habitat quality in comparison to mechanistic movement rules. However, as described above, due to complex feedbacks that may emerge from individual movement decisions, effects of directed host movement are difficult to predict and may be non-linear and counterintuitive (Hess et al. 2002, Cross et al. 2005, White et al. 2018). In general, we expected habitat-dependent movement rules to increase negative landscape effects since directed movement to high-quality patches should increase spatial heterogeneity of host densities, while competition-driven movement was expected to mask effects of landscape structure by equalizing local differences in host densities.

Methods

Model overview

The spatially explicit agent-based eco-epidemiological model is based on the study by Kramer-Schadt et al. (2009) and subsequent modifications by Lange et al. (2012a, b). In the original model transmission is based on nearest-neighbour group mixing processes, where infection pressure within and between neighbouring groups is based on constant transmission probabilities without movement of the host individuals. The modified version presented here assumes explicit phenomenological, fully imposed movement patterns and mechanistic movement based on individual decisions of hosts. Below we present essential parts of the model necessary for understanding model outcomes. The full model description following the overview, design concepts and details ('ODD') protocol (Grimm et al. 2006, 2010) is provided in the Supplementary material Appendix 1. The NetLogo model and the R code to analyse the simulation results are available on Zenodo (Scherer et al. 2019).

The model comprises two major components: a demographic host submodel and a virus submodel. Host individuals are characterised by sex, age in weeks, location, demographic status (residential, dispersing, ranging) and health status. The health status of the individuals is described by an SIR epidemiological classification (susceptible, infected and recovered; Kermack and McKendrick 1927). The simulation is updated on a weekly basis which equals the approximate CSF incubation time (Artois et al. 2002).

Landscape structure

All processes take place on a rectangular grid where each cell represents an area of $4\,km^2$ encompassing an average home range area of a wild boar group (Leaper et al. 1999, Podgórski et al. 2013, Fattebert et al. 2017). Each cell is characterised by a habitat quality value expressed as breeding capacity, denoting the highest possible number of breeding females and thus equalling the carrying capacity of the cell, or home ranges, respectively. We simulated landscapes representing $100 \times 50\,km$ (50×25 cells) and four types of landscape scenarios with increasing complexity of spatial structure (i.e. with decreasing aggregation and increasing edge density, fractality and Simpson's diversity, Fig. 2, Supplementary material Appendix 1 Fig. A6): a homogeneous landscape with a uniform habitat quality of 4.5 and three landscapes with different levels of heterogeneity in the clustering of patches with the same habitat qualities (small and large random clusters, and a purely random setup) and habitat qualities ranging from 0 to 9 (Fig. 2, Table 1). Two hundred unique landscape maps with an average habitat quality of 4.5 were generated with the R packages NLMR and landscapetools (Sciaini et al. 2018), resulting in the reported density of five wild boars per km^2 (Howells and Edwards-Jones 1997, Sodeikat and Pohlmeyer 2003, Melis et al. 2006). For each landscape scenario, the same 200 maps have been reused for the different parameter combinations of movement rule and case fatality ratio to avoid pure stochastic effects of landscape generation.

Host movement

We explicitly modelled the inter-cell movement of adult male wild boars; movement of resident females and dependent offspring within their home ranges was neglected.

	Homogeneous	Large clusters	Small clusters	Random
Aggregation index:	NA	82.9 ± 2.0	50.2 ± 2.1	10.7 ± 0.6
Edge density:	0	3,922.0 ± 426.7	10,076.5 ± 389.9	17,461.7 ± 112.4
Fractal dimension:	0	14.9 ± 31.8	23.0 ± 1.4	28.3 ± 0.7
Simpson's diversity:	0	0.77 ± 0.04	0.82 ± 0.01	0.90 ± 0.00

Decreasing aggregation index
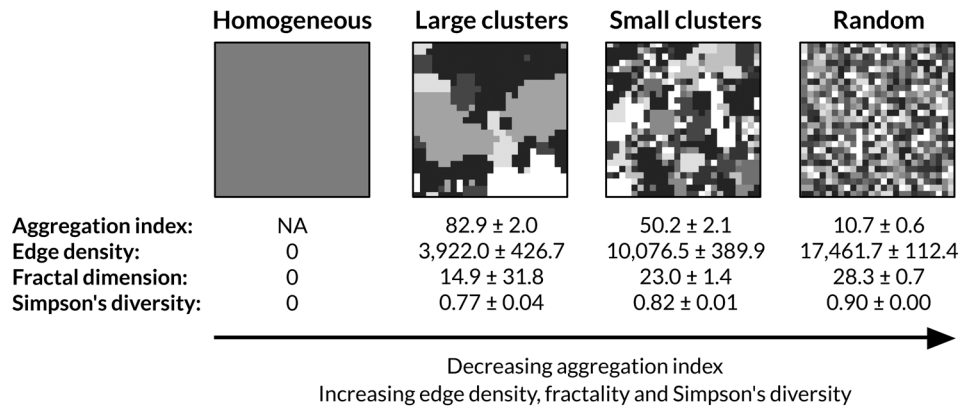Increasing edge density, fractality and Simpson's diversity

Figure 2. Examples and landscape metrics of the landscape scenarios used in the simulation study. Landscape scenarios were ordered to represent gradients in different landscape characteristics such as aggregation index, edge density, fractality dimension and Simpson's diversity index. Note that only an exemplary snapshot is shown and the landscape maps used for the simulation did cover a grid of 50×25 cells.

While adult female wild boars move mainly within their family group (staying strategy with negligible contacts between groups; Spitz and Janeau 1990, Pepin et al. 2016), adult males follow a ranging strategy and move solitarily (Morelle et al. 2015). Individual movement distances were drawn from a truncated median Weibull distribution so that ranging males display mostly conventional (22 km per week) but also long-distance movement behaviour covering distances up to 84 km per week (Podgórski et al. 2013, Morelle et al. 2015). We considered three different movement strategies to study their effects on disease dynamics. Movement was either modelled as correlated random walk (CRW) or emerged from the individual's decision to move towards cells with higher habitat quality (habitat-dependent movement: HDM) or lower

conspecific competition simulated as negative-density dependence (competition-driven movement: CDM). Individuals performing a CRW do not take the underlying landscape structure into account, but exhibit a general tendency to continue in the same direction as the previous movement decision (Kareiva and Shigesada 1983, Codling et al. 2008). In contrast, individuals following rule HDM or CDM make mechanistic decisions related to the underlying landscape directly (HDM) or indirectly (CDM). While habitat quality is static and does not change with time, density of conspecifics can be dynamic as a consequence of demographic processes, the movement rule and disease-induced mortality. The three movement strategies only differ in their way of deciding where to go each step (i.e. follow the previous

Table 1. Variables and parameters that vary from the original model version of the agent-based model simulating classical swine fever in wild boar. Each of 60 combinations of movement rule (three levels), landscape scenario (four levels) and case fatality ratio M (five levels) was repeated 200 times, resulting in 12 000 runs in total. The same 200 landscape maps were used for each combination per landscape scenario to allow for comparability between movement strategies. For a detailed list of parameters used in the model see Supplementary material Appendix 1.

Name	Values
(A) Variables	
Movement rule	(i) correlated random walk (CRW)
	(ii) habitat-dependent movement (HDM)
	(iii) competition-driven movement (CDM)
Landscape scenario (Fig. 2)	(a) homogeneous
	(b) large clusters (p=0.5)
	(d) small clusters (p=0.1)
	(d) random
Case fatality ratio	$M \in \{0.1; 0.3; 0.5; 0.7; 0.9\}$
(B) Parameters	
Mean infectious period (weeks)	$\mu_{mean} = 4$
Maximum infectious period (weeks)	$\mu_{max} = 40$
Transmission probability	$\beta = 0.022$
Pathogen release (week)	$U(53, 104)$
Mean movement distance (cells per week)	$D_{mean} = 12$
Maximum movement distance (cells per week)	$D_{max} = 42$
Individual movement distance (cells per week)	$D_i \{1, D_{max}\} \sim \text{Wei}(26, 1.3)$
Tendency to move randomly	$\rho = 0.7$
Habitat quality (number of breeding females)	$z = 4.5$ for landscape scenario (a),
	$z \in \{0, 9\}$ with $\bar{z} = 4.5$ for landscape scenarios (b–d)

direction or choose cell with highest habitat quality or lowest competition), with 30% of their decisions being random to represent noise in movement decisions. In general, individuals were moving until the individuals' weekly movement distance was reached or movement rules led to the decision to stay in the current cell (i.e. no surrounding cells classified as habitat for CRW, with same or higher habitat quality for HDM, or with same or lower density for CDM).

Pathogen transmission

The disease course is determined by the case fatality ratio M, the probability to become lethally infected or transient infected otherwise, and the infectious period μ of lethally infected hosts. M is age-specific with a lower probability for adults ($M_a = M^2$) and a higher probability for piglets ($M_p = M^{0.5}$) in comparison to subadults ($M_s = M$) (Dahle and Liess 1992). The individual infectious period of lethally infected host is based on μ that is drawn from an exponential distribution with a mean of four weeks and a truncated maximum of 40 weeks. Transient infected hosts shed the pathogen for one week and gain lifelong immunity (Dahle and Liess 1992). Infection dynamics emerged from within-group and on-the-move transmission and individual courses of infection. We verified our model and calibrated the transmission probability by comparing emerging distributions of the basic reproduction number, R_0 (estimated as the number of secondary infections caused by an infective agent in a completely susceptible population; Diekmann et al. 1990) to those of the original model (Supplementary material Appendix 2). On average, an infected individual generated 1.6 new cases over the course of its infectious period ($R_{0,CRW} = 1.57$, $R_{0,HDM} = 1.59$ and $R_{0,CDM} = 1.63$ compared to $R_0 = 1.59$ for the original model with neighbourhood infection). Within family groups, the transmission probability and the number of infectious group members (excluding reproductive males) determine the density-dependent infection pressure. For ranging males, individual per-step transmission probability is calculated as the transmission rate divided by the movement distance to account for the time an individual spends in each cell and may shed or catch the virus. If individuals stopped moving, the remaining per-step infections happened in the current cell.

Process scheduling

The temporal resolution equals the approximate CSF incubation time of one week (Artois et al. 2002). Each time step, the following procedures were executed in the given order: pathogen transmission, host ranging movement, natal host dispersal of males and females, host reproduction, host mortality, host ageing and disease course. Natal dispersal of males and females was limited to week 17 and week 29 of the year, respectively, representing the typical dispersal time for each sex. Each simulation was run for 12 years (624 weeks) in total, with the virus being released in the second year (week 53–104). The virus was introduced to a defined cell in the upper row with a habitat quality of 4.5 (Fig. 1). Per combination of movement rule (three levels: CRW, HDM and CDM)

and landscape scenario (four levels: homogeneous, large clusters, small clusters and random), we ran 200 simulation runs. Furthermore, we varied the case fatality ratio to simulate five different levels of disease severity, ranging from a mostly transient (M = 0.1) to a highly lethal (M = 0.9) pathogen, resulting in a total of 12 000 runs (Table 1). Note that severity had no bearing on transmission since the transmission probabilities were simulated to be independent of the case fatality ratio.

Analysis

Disease persistence and outbreak duration

During each week, we recorded numbers of individuals with each health status. From this, we derived the duration of each disease outbreak, which was used to calculate persistence probabilities. Disease persistence was estimated using a 10-year threshold, i.e. the disease was defined as persistent only in those simulation runs in which infected individuals were present 10 years after the pathogen was released to the naive population. This threshold is an arbitrary choice just like other viability thresholds, for example in population viability analyses (Grimm and Wissel 2004). It was based on previous simulation studies that estimated disease persistence on similar timescales (Visser et al. 2009, Belsare and Gompper 2015, Lange et al. 2016). However, we note that in real-life situations, a pathogen may be classified as persistent in a system after a much shorter outbreak duration. Thus, we additionally estimated disease persistence using a 2.5-, 5- and 7.5-year threshold, respectively. Furthermore, we explored the outbreak duration measured as the weeks until the pathogen went extinct within the system for simulation runs in which the disease did not persist.

Landscape- and movement-related variables

To reveal effects of different spatial complexity and different movement strategies, we recorded for each time step 1) the variation in density of all individuals and ranging males per week across all cells, either occupied or unoccupied (i.e. unbiased estimate of variance in number of individuals per cell), 2) the average realized movement distance as the mean number of unique cells visited by ranging males, 3) the contact rates between infected ranging males and susceptible individuals, grouped by the habitat quality of the cell, 4) the proportion of cells containing infected individuals, 5) the cumulative proportion of affected cells (i.e. the proportion of cells containing infected individuals at any time in the entire run), as well as 6) the number of transmissions within and between patches of the same habitat quality. To calculate these transmission rates, we first defined unique patches of cells of equal habitat quality that where adjacent to each other (Moore neighbourhood). We then measured within-patch transmission rates as the number of infections a moving individual caused within the patch it was infected in, while infections outside of this patch were used to calculate between-patch transmission rates. For each run, all measurements were summarized as mean over a time period of three months (13 weeks) starting from the week the pathogen was released.

Results

Long- and short-term persistence

Probability of long-term persistence, estimated as the presence of infected individuals 10 years after the pathogen release, was considerably higher when movement was simulated as a mechanistic process based on the habitat quality (HDM) or the presence of conspecifics (CDM) instead of a phenomenological pattern of correlated movement steps (CRW; Fig. 3a). In general, persistence probability was very low in homogeneous landscapes (probability of 0.09) compared to heterogeneous landscapes (0.21 or higher), with the highest probabilities arising in more complex landscapes (Fig. 3b). Especially in homogeneous landscapes persistence was very low in the case of CRW and HDM (0.05 and 0.04, respectively) compared to CDM (0.19). In heterogeneous landscapes, mechanistic movement strategies yielded a considerably higher proportion of runs where the pathogen was able to persist for more than 10 years (0.21–0.4 for HDM and 0.33–0.37 for CDM) in comparison to CRW (0.05–0.08; Fig. 3c).

Compared to the results for the 10-year threshold we found similar patterns of disease persistence when evaluating persistence probabilities based on a 5- and 7.5-year threshold (i.e. highest persistence probabilities for heterogeneous landscapes and mechanistic movement strategies, respectively, and lowest persistence probabilities in homogeneous landscapes and when assuming CRW). However, results differed strongly for the 2.5-year threshold (Fig. 3, 4, Supplementary material Appendix 1 Fig. A7). Probability of disease persistence

for more than 2.5 years (afterwards termed probability of short-term persistence) was the highest in homogeneous landscapes for all movement strategies (Fig. 2, 4). The difference in probabilities of short- and long-term persistence was generally low when assuming CRW. Mechanistic movement assumptions exhibited high probabilities of short-term persistence in homogeneous landscapes (0.86 for HDM and 0.98 for CDM) that decreased in the case of non-homogeneous landscape scenarios. This effect was more pronounced in the case of HDM, while CDM led to considerably higher probabilities of short-term persistence across all landscape scenarios, especially in more complex landscapes (0.78 for small clusters and 0.87 for random).

Outbreak duration and pathogen extinction

We further explored the differences between movement strategies by visualizing the outbreak duration, i.e. the time the pathogen was present in the system before it went extinct (Fig. 4, Supplementary material Appendix 1 Fig. A8). In general, during the 10 years of outbreak simulation almost all pathogen extinctions happened within the first five years (always more than 89%). The mean extinction time of runs facing pathogen extinction within 10 years was slightly lower in heterogeneous compared to homogeneous landscapes (Fig. 4). The mean extinction time decreased considerably in the case of HDM when replacing the homogeneous landscape by structured landscapes, but only slightly in the case of CRW and CDM. When individuals followed the HDM rule in heterogeneous landscapes, the outbreak did not last longer
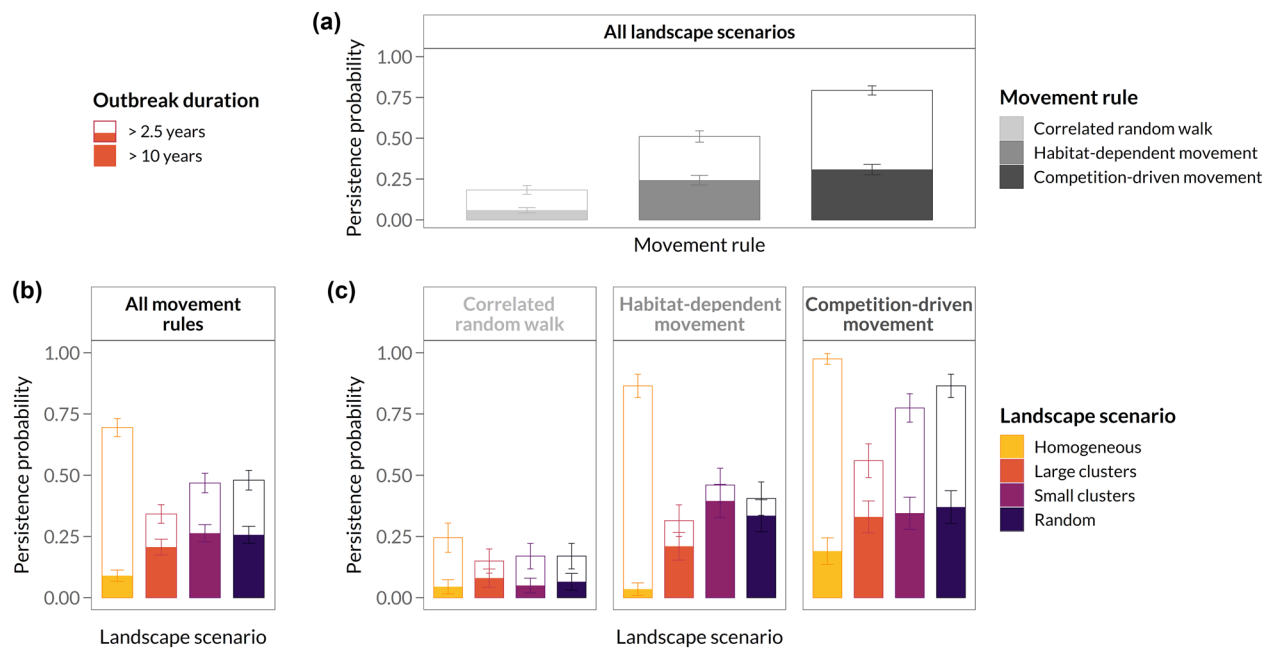

Figure 3. Probability of disease persistence estimated as the proportion of simulation runs that lasted more than 2.5 ('short-term persistence'; empty + filled bars) and 10 years ('long-term persistence'; filled bars) after the pathogen was released out of 200 simulation runs. Probabilities of persistence are grouped by (a) movement rule, (b) landscape scenario and (c) for each combination of those variables. Error bars show the 95% binomial confidence intervals.
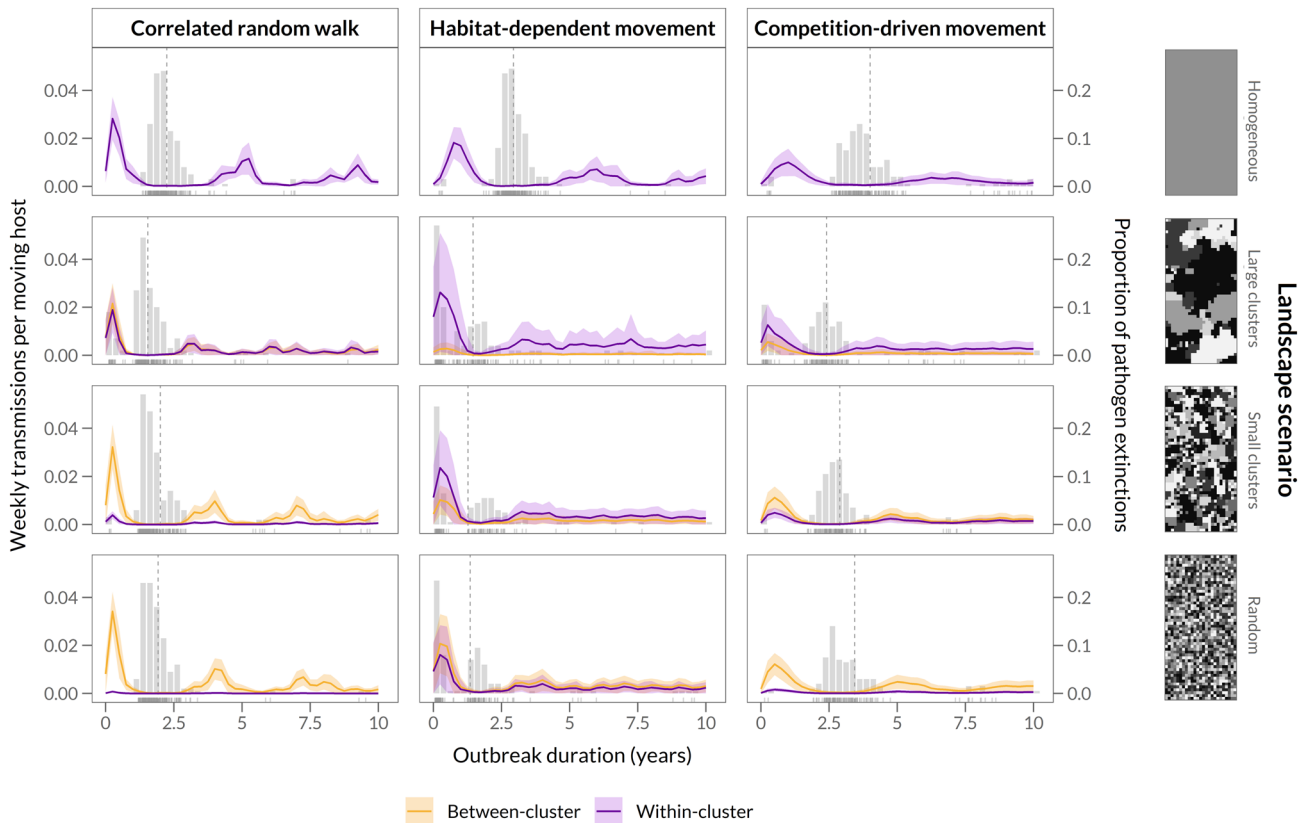
Figure 4. Temporal dynamics of weekly within- and between-patch transmission rates (mean ± SD; orange and violet lines; left axis) for outbreaks that lasted for more than five years. We first defined unique patches of cells of equal habitat quality that where adjacent to each other (Moore neighbourhood). We then measured within-patch transmission rates as the number of infections a moving individual caused within the patch it was infected in; infections outside of this patch were used to calculate between-patch transmission rates. 200 simulation runs were conducted for each combination of movement rule (columns) and landscape scenario (row) allowing the pathogen to spread for a total of 520 weeks (10 years) after release. The histogram depicts the outbreak duration of all runs with an outbreak duration with less than 10 years, measured as weeks until the pathogen became extinct (grey bars and rugs; right axis). The vertical dashed line indicates the mean extinction time of runs that did not last 10 years. For each landscape scenario an exemplary scenario is shown on the right. The proportion of simulation runs lasting for more than 10 years is equal to the probability of persistence (cf. Fig. 3).

than a few months in around 25–30% of the simulation runs. This was rarely the case for all other setups (0.5–11.5%) that exhibited unimodal distributions of extinction time (Fig. 4, Supplementary material Appendix 1 Fig. A8) with the majority of extinction events happening after the initial, invasive spread of the pathogen that led to rather long periods of low spread within and between patches (Fig. 4, Supplementary material Appendix 1 Fig. A9).

Landscape- and movement-related variables

Introducing heterogeneity in habitat quality led to the expected variance of host densities (Fig. 5a), since habitat quality is defined as breeding capacity and thus ultimately limits the group size of a home range. Landscapes with high levels of aggregation of cells with the same habitat quality showed the highest variance in host densities but differences between the three heterogeneous landscapes were small. Spatial differences in densities of all individuals were most pronounced when assuming HDM. Depending on the

movement rule, effects of landscape structure on the variance in densities of ranging males were non-existent (CRW), rather small (CDM), or exhibited clear trends towards high variance of ranging males in more complex landscapes (HDM). However, variances in the distribution of moving hosts were generally higher under CDM when compared to CRW but lower when compared to HDM. In accordance with the absence of any variance in densities of ranging males, the assumption of CRW did not exhibit any differences in the number of unique visited cells, irrespective of the landscape scenario (Fig. 6b). The number of visited cells was considerably lower when assuming movement behaviour as a mechanistic process. For HDM and CRW, the number of visited cells did not differ between pre-outbreak and outbreak period. However, in the case of CDM more cells were visited before the outbreak started than during the outbreak. This pattern was also visible in the variance in density of ranging males (Supplementary material Appendix 1 Fig. A10), with higher variance emerging during the outbreak period compared to the burn-in phase.
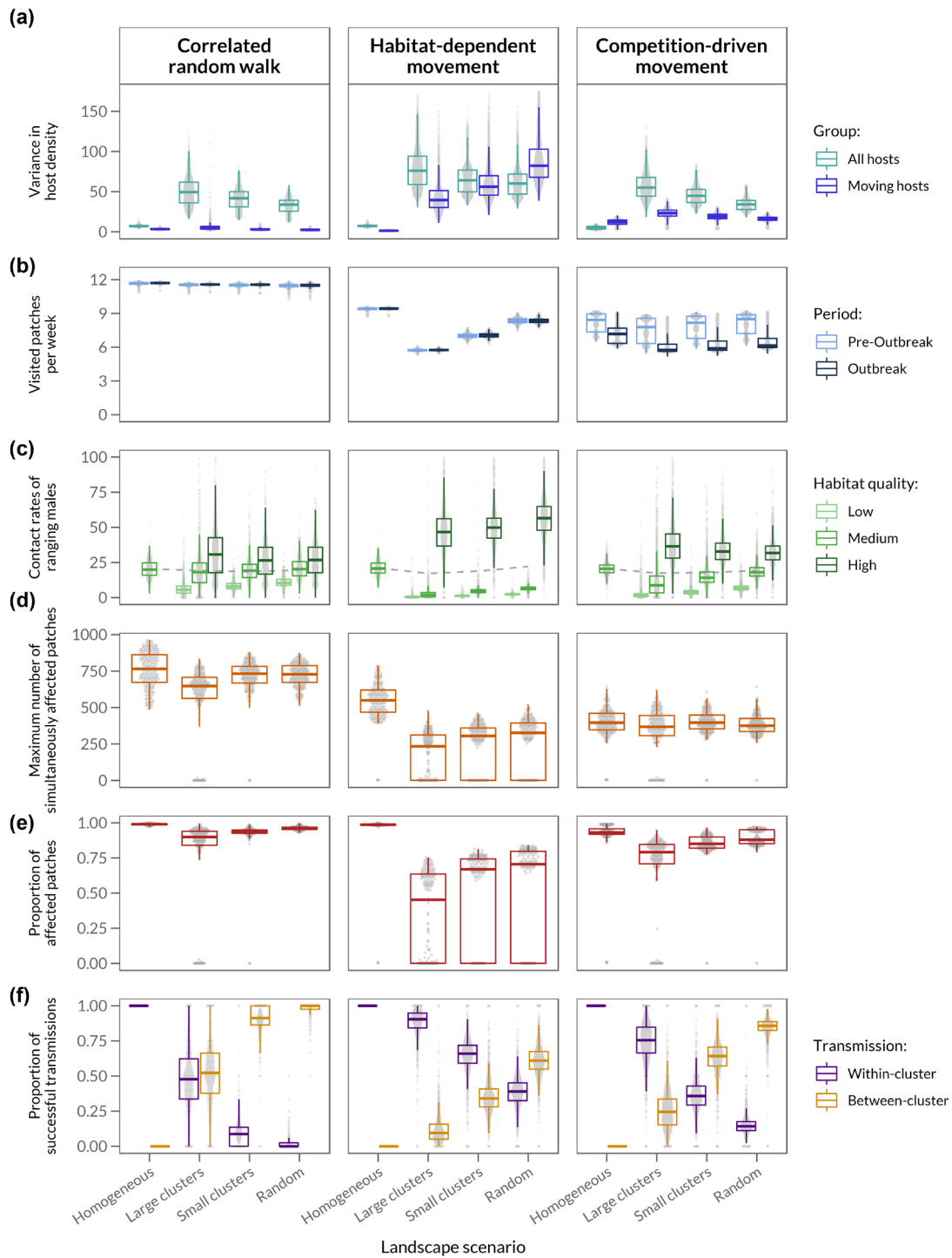
Figure 5. Landscape- and movement-related variables as a function of movement rule (columns) and landscape scenario (x-axis), estimated from 200 model runs. For each run, measurements were summarized as mean over a time period of three months (13 weeks) starting from the week the pathogen was released. (a) Variance of host density, either for all individuals (turquoise) or ranging males only (blue). (b) Mean number of unique visited cells per week before the outbreak started (light blue) and during the outbreak (dark blue). (c) Number of contacts between infected ranging males and susceptible hosts for three groups of habitat quality (low: habitat quality > 0 and ≤ 3, light green; medium: > 3 and ≤ 6, medium green; high: > 7 and ≤ 9, dark green). The dashed line indicates the mean contact rates over all habitat qualities. (d) Proportion of habitat cells that contained at least one infected individual in the same time step. (e) Proportion of habitat cells that contained at least one infected individual during the simulated outbreak. (f) Proportion of infections that were caused by ranging males either within the patch in which they got infected (yellow) or between different patches (purple). Since homogeneous landscapes consist of similar habitat quality, within- and between-patch transmission rates are obsolete for these setups.

(a)

Long-term persistence (outbreak duration > 10 years)

(b)

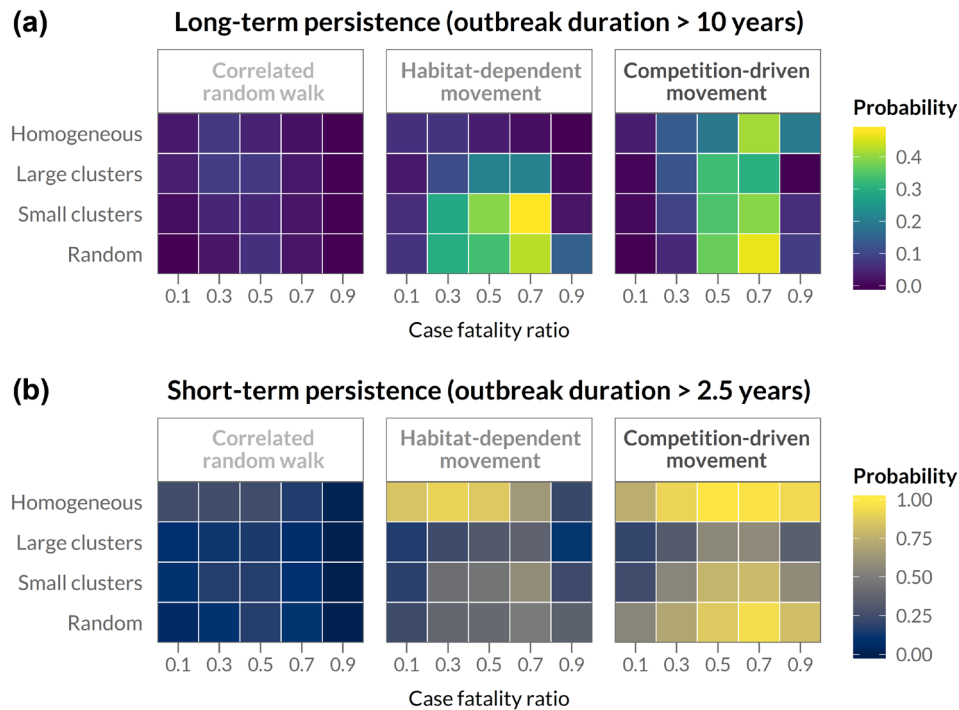**Short-term persistence (outbreak duration > 2.5 years)**

Figure 6. Probabilities of (a) long-term persistence (i.e. outbreaks that lasted longer than 10 years) and (b) short-term persistence (i.e. outbreaks that lasted longer than s.5 years) with variation in the case fatality ratio M (ranging from mostly transient, M = 0.1, to mostly lethal infections, M = 0.9) per landscape scenario and movement rule (cf. Supplementary material Appendix 1 Fig. A8).

Infected ranging males were much more likely to have high numbers of contacts with susceptible hosts in high-quality habitat (defined as the upper ⅓ of the habitat quality; Fig. 6c). At the same time, contact rates in medium- and low-quality habitat (defined as middle and lower ⅓ of the habitat quality, respectively) were lower when assuming HDM than in the case of either CRW or CDM. The contact rates when assuming CDM differed between different levels of habitat quality but the effect was much less pronounced compared to the patterns found in HDM. As before, CRW in general only led to low variation in contact rates between different habitat qualities and landscape scenarios.

Movement behaviour had a strong effect on the proportion of affected cells (Fig. 6d–e). The maximum proportion of simultaneously affected cells, estimated as patches that contain at least one infected individual, was higher in the case of CRW in comparison to CDM and especially HDM in heterogeneous landscapes. Similarly, CRM resulted in the highest proportions of cumulatively affected cells, estimated as patches that contained at least one infected individual during the simulated outbreak, but CDM yielded only slightly lower proportions. In the case of HDM in heterogeneous landscapes, a considerable number of simulations resulted in very low proportions of both simultaneously and cumulatively affected cells. This pattern is also visible in the temporal dynamics of the epidemic size: CRW led to distinct and high peaks of infected individuals and affected cells during the initial spread of the pathogen which was mostly pronounced in heterogeneous landscapes (Supplementary

material Appendix 1 Fig. A11, A12). In contrast to the dynamics under the assumption of CRW, HDM and CDM led to considerably damped but also longer invasion periods in terms of the number of infected individuals.

The interplay of landscape structure and individual movement strategies had a strong impact on the transmission rates within a patch (i.e. within the same group of cells of similar habitat quality that the ranging male was infected in) or between patches (Fig. 4, 6f). While the general pattern of high within- and low between-patch transmission rates in highly clustered landscapes and vice versa was comparable, there were remarkable differences between movement strategies: the proportion of within-patch transmission on average never exceeded 50% when assuming CRW, but accounted for more than 39–90% of all transmissions when assuming HDM in heterogeneous landscapes. The same but less pronounced pattern emerged when simulating host movement as CDM that led to a proportion of 14–75% of within-patch transmissions.

Variation in the case fatality ratio

Probabilities of persistence based on 5, 7.5 and 10 years exhibited the same pattern when varying the case fatality ratio (M; Supplementary material Appendix 1 Fig. A8). Long-term disease persistence was highly unlikely in the case of mostly transient (M = 0.1) or highly severe infections (M = 0.9; Fig. 5a). Short-term persistence (based on the 2.5-year threshold) in homogeneous landscapes was very robust in the case M shifted towards more transient infections and in

general when simulating host movement as CDM. Changes of M towards more lethal infections decreased the probability of short-term persistence especially for CRW and HDM.

Discussion

By extending an agent-based model mimicking an outbreak of an infectious agent in a social host, we demonstrated that including mechanistic movement decisions based on the external factors can reverse the findings of models based on phenomenological movement rules. We expected high rates of exchange, as in the case of correlated random walks (CRW), to increase disease persistence as predicted by other studies (Hess 1996, Jesse et al. 2008). Interestingly, CRW resulted in the lowest probabilities of both short- and long-term persistence of the pathogen. On the contrary, the emerging variation in host density and contact rates in combination with movement arising from individual decisions allowed for persistence probabilities that were up to eight times higher than under random conditions.

Movement decisions increase disease persistence in structured landscapes

In an important theoretical work on the effect of connectivity on disease persistence, Hess (1996) developed an epidemiological metapopulation model where fractions of the susceptible and infected compartments were transferred between populations at equal rates. He showed that the probability that a disease becomes persistent increases as the transfer rates among populations increase, especially in the case of highly contagious diseases of moderate severity. Our findings of a severe decrease in disease persistence when simulating movement as CRW contrasts those of the study by Hess (1996). Although R_0 and the host density were kept constant among simulations, the pathogen depletes the susceptible hosts too fast due to randomly chosen movement directions and long movement distances imposed by CRW. Contrary, the slower and more directed spread of the disease emerging in the case of mechanistic movement decisions ensures continuous sources of both susceptible and infectious hosts (Read and Keeling 2003, Tildesley et al. 2010). In contrast to the homogeneous mixing assumption that all susceptible hosts have the same chance to get infected, in spatially explicit models such as ours the number of susceptible hosts in the focal and surrounding cells is of importance for transmission and persistence (Tildesley et al. 2010, White et al. 2018). Due to the fast spread resulting from CRWs, irrespective of the underlying landscape, a considerably higher proportion of cells is simultaneously infected. In consequence, the pool of susceptible hosts becomes quickly and simultaneously exploited across the entire spatial domain and the system becomes more prone to stochastic fade-outs due to the low density of susceptible individuals, which is corroborated by studies on oscillatory infection cycles (Dietz 1976, Lloyd and May 1996, Grenfell and Harwood 1997, Rohani et al. 1999, Hagenaars et al. 2004).

In contrast, we found that the probability of persistence was considerably higher in heterogeneous, non-binary landscapes when explicitly representing individual movement decisions. As a consequence of the mechanistic movement, host movements were more restricted to certain areas, thereby intensifying spatial variation in host density while causing remarkable spatial heterogeneity in contact and transmission rates. These differences to mean-field approaches result from the feedbacks of directed host movement in response to landscape structure on host density and contact rates (White et al. 2018). The assumption of homogeneous mixing of hosts on a population-level in conventional compartmental metapopulation models does not allow for the observed variation in contact rates (Read and Keeling 2003, Lloyd-Smith et al. 2005b, Cross et al. 2013, VanderWaal and Ezenwa 2016). In contrast, the individual-based model presented in this study allows for emerging changes in contact rates and host density due to individual movements (Swingland and Greenwood 1983, Turchin 1991). Two related epidemiological mechanisms may explain increased probabilities of persistence we have found under decision-based movement: 'disease hotspots' (Real and Biek 2007, 'transmission islands'; Paull et al. 2012) and 'structural delay' ('structural trapping effect'; Sah et al. 2017).

The mechanisms behind disease persistence

Changes in host susceptibility, community structure and contact rates that generate disease hotspots in animal species are frequently associated with anthropogenic impacts such as eutrophication and fragmentation that alter habitat quality and landscape complexity (Patz et al. 2004, Johnson et al. 2010, Paull et al. 2012). The role of these 'disease hotspots', namely areas that contain high densities of (susceptible) hosts leading to heterogeneous contact and transmission rates in populations, has recently received much attention (Hosseini et al. 2004, Cross et al. 2005, Real and Biek 2007, Paull et al. 2012). In our model, high-quality cells served as hotspots, thereby increasing contact rates and restricting spread. While this effect was more pronounced and rather obvious in the case of habitat-dependent movement (HDM), we did not expect to find such a pattern when movement was represented as negatively density-dependent in the competition-driven movement (CMD). However, while this movement rule yielded quite low variation in density during disease-free periods, the infection itself induced movement into areas that were recently affected. Thereby, these hotspot areas did not only exhibit the highest contact rates between infected and susceptible individuals, but also caused a 'structural delay' effect with only a few individuals leaving the infected area and thus delaying the spread to other cells (Sah et al. 2017).

Accounting for other factors in the modeling approach

Our model accounted for spatial variation and temporal changes in host density as well as phenomenological and

mechanistic movement rules to investigate disease dynamics, but several other key factors that might influence disease persistence such as seasonal changes in movement behaviour or the distribution of resources could be addressed in our framework as well. For example in mammals, adult males often exhibit higher levels of activity and space use, especially in the case of polyandrous males during the mating season (Greenwood 1980, Dobson 1982, King et al. 2013). While during this season movement decisions of males may be mostly driven by the presence of females, decisions may be driven by resources and shelter during other seasons of the year. In addition, seasonal or unpredictable short-term disturbances may influence the dynamics of the host population (Radchuk et al. 2019b). Such rare events in wildlife could, however, be decisive for explaining the system's behaviour (Jeltsch et al. 1997, Lloyd-Smith et al. 2005b, Paull et al. 2012). Finally, here we considered a limited set of epidemiological and movement parameters based on the CSF-wild boar system for the sake of simplicity. Since one of the suggested main factors for increased chances of persistence of CSF in wild boar populations is the long-term change from a highly virulent CSF virus strain to a strain of moderate virulence (Mittelholzer et al. 2000, Lange et al. 2012a, b), we have tested several case fatality ratios, highlighting that more moderate strains enable disease persistence in the presence of decision-based movement. However, changes in epidemiological or movement parameters, e.g. infectious period or movement distance, are likely to cause additional feedback on the observed disease dynamics (Cross et al. 2005, Lloyd-Smith et al. 2005b, White et al. 2018). To account for comparability among simulations, we fixed R_0 and allowed for a sufficient long infectious period in relation to the movement rate, thus enabling the pathogen to invade the population successfully in absence of spatial heterogeneity (Lloyd-Smith et al. 2005a) since we were rather interested in long-term persistence than in examining the range of epidemiological parameters that cause fade-outs due to too low rates of spread. An alternative approach would be a fixed epidemic growth rate as a measure of the speed of epidemic growth that conveys more information about the time scale of disease spread as opposed to R_0. However, the decision on the parameterization approach would be more important in the context of predictive modeling in combination with empirical data than in the case of our theoretical model. To account for the time scale and the stochasticity in infectious periods when estimating R_0 we have run the calibration runs for longer than one timestep (one week).

Simulation of individual variation and host movement in disease models

Individual-based approaches are a means to relax the assumption of homogeneous mixing among individuals within a population and also subgroups. When focussing on the individual instead of the population, differences in behaviour that drive social interactions, individual movements and segregation into groups can be included (Grimm and Railsback 2005,

Craft 2015). Ultimately, the social and spatial structuring limits interactions between individuals and thus transmission dynamics might differ within and between groups, forming a heterogeneous network of contacts and transmission (Newman 2002, Altizer et al. 2003, Craft and Caillaud 2011, Ferrari et al. 2011, Sah et al. 2017). A major advantage of a network-centric epidemiological view is the long history of networks in graph theory thereby providing many quantitative tools and mechanisms for the description and the analysis of network models (Keeling and Eames 2005, Ferrari et al. 2011). Network models have become very popular approaches to capture individual variation and have been used to explore the role of different factors such as group size, modularity and transmission rates on global disease dynamics (Meyers et al. 2005, Craft et al. 2011, Sih et al. 2018, White et al. 2018). For example, Craft et al. (2011) characterized the social contact network of Serengeti lions *Panthera leo* and investigated the contribution of nomadic males on the dynamics of the canine distemper virus (CDV). Similar to our model, groups of individuals were assigned to spatial locations and nomadic males were moving among these groups. However, in contrast to our model they allowed for transmission between neighbouring and non-neighbouring groups. These occasional long-range connections were sufficient enough to drive the epidemic while nomads did not qualitatively alter the network structure. This is in contrast to our approach where ranging host individuals are needed to connect spatially distinct groups and thus by definition form the network between groups. Another interesting network model focussing on host movement between family groups is the work by Davis et al. (2008) on plague in populations of great gerbils *Rhombomys opimus*. By modelling transport of infectious flea from burrow to burrow, they showed that, in contrast to conventional theory based on abundance threshold, the local depletion of susceptibles arising from spatial restrictions in contacts among groups leads to so-called 'percolation thresholds'. These spatially explicit thresholds of local host abundance lead, similarly to the structural delay effect, to spatio–temporal barriers and recurrent and occasional spread to distinct groups. This pattern is supported by our findings of reduced euclidian movement distances. This structural delay slows the spread of the pathogen in space and thus allows for persistence of the disease within the host population.

In general, basically each model that simulates structured populations can be seen as a network: in the classical metapopulation model, nodes are formed by populations and any given node is connected with all other nodes, while lattice-based models are regularly connected nodes on a grid (Colizza and Vespignani 2008, Keeling and Rohani 2008, Daversa et al. 2017). While network approaches traditionally depict static edge, recent development of dynamic network models are a promising avenue to enable the incorporation of spatiotemporal variation in contacts and external key factors causing these changes (Godfrey et al. 2013, Craft 2015, Jacoby and Freeman 2016, Sih et al. 2018). One example

are stochastic actor-oriented models (SAOMs), a class of individual-level networks that simulates transitions between discrete time points and that is increasingly applied in animal network studies thanks to recent increases of high-resolution data (Fisher et al. 2017). An alternative approach are agent-based models (ABMs) that are flexible tools to capture the spatial and temporal dynamics found in social and transmission networks. A major advantage is their mechanistic nature from which higher-level processes emerge (Grimm and Railsback 2005, Radchuk et al. 2019a, b). ABMs are thus excellent candidates to simulate the interplay of landscape structure and individual host movement, while incorporating variation in movement based on step length and turning angle or behavioural and environmental preferences (Grimm and Railsback 2005, Avgar et al. 2013, Coulon et al. 2015, DeAngelis and Diaz 2019). In combination with basic assumptions on transmission rates that are a function of contact duration (as in the model presented here) or frequency, individual movement and resulting transmission networks can be upscaled to spatiotemporal patterns on a population scale (Craft 2015, Dougherty et al. 2018b, Sih et al. 2018). Due to their simplicity, random walk approaches are the most widely used tool to model movement of animals in ecological studies (Codling et al. 2008) and have been used in several eco-epidemiological models (Jeltsch et al. 1997, Craft et al. 2011, Belsare and Gompper 2015, Fofana and Hurford 2017). The CRW approach extends the simple random walk model to more realistically account for short-term correlations in the movement direction, thereby imitating observed movement patterns of area-restricted search and long-range movements between these areas (Lotka 1925, Patlak 1953, Kareiva and Shigesada 1983, Bovet and Benhamou 1988). However, classical random walk approaches are purely phenomenological, thus imposing movement patterns similar to those that have been observed (Kareiva and Shigesada 1983, Codling et al. 2008). Hence, these models ignore any feedbacks of the underlying landscape structure and other factors on the movement decision that may affect population-level characteristics such as density, spatial distribution and demography of populations (Gaillard et al. 2010, Morales et al. 2010) with important consequences for encounter and contact rates (Hutchinson and Waser 2007). One of the first epidemiological agent-based models that included host movement explicitly was a study by Jeltsch and colleagues (1997) that highlighted the importance of rare long-scale dispersal on the dynamics of rabies in a population of foxes performing random walks. Using different types of random walk approaches (simple, correlated or biased) in combination with a probability of leaving habitat cells, Tracey et al. (2014) simulated bobcat movements in binary landscapes with varying fragmentation levels to investigate transmission patterns of the feline immunodeficiency virus. Their model showed, similar to ours, that between-patch transmission rates were higher in more fragmented landscapes and that CRW yields the highest proportion of infectives. In our model, we have used two mechanistic movement rules, HDM

and CDM, in addition to the phenomenological CRW rule. Movement decisions based on the in response to environmental factors such as resources and shelter or to conspecifics is a typical driver of animal movement and used as a basis for movement analyses and simulation (Smouse et al. 2010, Doherty and Driscoll 2018). In our approach, HDM leads to crowding of animals in high-quality patches and thus can be seen as a positive density-dependent movement while we simulate CDM as negative density-dependent. Both movement rules led to increase chances of pathogen persistence but only CDM exhibited high persistence probabilities in homogeneous landscapes. This is caused by the response of ranging hosts to conspecifics, leading to heterogeneous distributions within the homogeneous landscape. Consequently, the spatial segmentation of the ranging individuals allows for structural delay effects in the absence of varying group sizes.

Using rather simple behavioural movement rules, our study supports and extends previous findings by showing that decision-based movement has the potential to reduce transmission rates between habitat patches due to decreases in movement distances and emerging spatial variation in the movement. Thereby, our findings underline the importance of mechanistic bottom–up processes caused by individual movement decisions (Nathan et al. 2008) in epidemiological models to investigate emerging contact rates on the population-level. Recent developments in telemetry sensor technology allow to track various animals of the same species with great detail (Nathan et al. 2008, Kays et al. 2015), thereby offering the ability to evaluate spatiotemporal changes in movement behaviour and to incorporate these insights into eco-epidemiological models using mechanistic movement rules (Dougherty et al. 2018b, Sih et al. 2018, Tardy et al. 2018). For example, by combining empirical near-continuous tracking data and behavioural rules, Dougherty et al. (2018a) investigated emerging contact rates between hosts and infectious reservoirs of Anthrax in a theoretical setup, showing that probabilities of contact were always greater than expected by random movement alone but lower than estimated on a home range resolution. These findings together with our model study highlight the possible over- or underestimation of rates of contact, prevalence and ultimately disease persistence when simulating host movement implicitly or explicitly but using phenomenological movement patterns.

Conclusion

Our findings underscore the importance of accounting for spatial context and group size structures in eco-epidemiological models. Our study highlights that the simulation of explicit, mechanistic movement behaviour can reverse predictions of disease persistence in comparison to phenomenological rules such as random walk approaches. The change in emerging contact rates can have severe consequences when predicting the probability of disease persistence and assessing control measures to prevent outbreaks. Thus, individual movement

should desirably be modelled as a mechanistic process based on external abiotic and/or biotic factors rather than as phenomenological pattern to avoid over- or underestimation of contacts leading to potentially biased conclusions about the infectiousness and persistence probability of a pathogen (Hess et al. 2002, Cross et al. 2005, Dougherty et al. 2018a). Our relatively simple yet mechanistically-based movement depiction provides insights for understanding the emergence of contact heterogeneity and the effect of individual movement decisions in interaction with landscape heterogeneity on pathogen spread. Furthermore, approaches such as ours can be easily implemented in existing and future epidemiological ABMs and extended to simulate movement decisions on several criteria simultaneously or depending on the location or season. The rather simple, theoretical rules in the study presented here highlight feedbacks of great importance for the spatial spread and the persistence of directly transmitted diseases.

Data availability statement

The NetLogo model and the R code to analyse the simulation results are available on Zenodo (doi: 10.5281/zenodo.3387444) (Scherer et al. 2019).

References

Altizer, S. et al. 2003. Social organization and parasite risk in mammals: integrating theory and empirical studies. – Annu. Rev. Ecol. Evol. Syst. 34: 517–547.

Anderson, R. M. and May, R. M. 1991. Infectious diseases of humans: dynamics and control. – Oxford Univ. Press.

Arthur, R. F. et al. 2017. Contact structure, mobility, environmental impact and behaviour: the importance of social forces to infectious disease dynamics and disease ecology. – Phil. Trans. R. Soc. B 372: 20160454.

Artois, M. et al. 2002. Classical swine fever (hog cholera) in wild boar in Europe. – Rev. Sci. Tech. 21: 287–303.

Avgar, T. et al. 2013. An empirically parameterized individual based model of animal movement, perception and memory. – Ecol. Model. 251: 158–172.

Baiser, B. et al. 2013. Predicting food–web structure with metacommunity models. – Oikos 122: 492–506.

Bansal, S. et al. 2007. When individual behaviour matters: homogeneous and network models in epidemiology. - J. R. Soc. Interface 4: 879–891.

Bartlett, M. S. 1960. The critical community size for measles in the United States. – J. R. Stat. Soc. Ser. A 123: 37–44.

Belsare, A. V. and Gompper, M. E. 2015. A model-based approach for investigation and mitigation of disease spillover risks to wildlife: dogs, foxes and canine distemper in central India. – Ecol. Model. 296: 102–112.

Bolker, B. and Grenfell, B. 1995. Space, persistence and dynamics of measles epidemics. – Phil. Trans. R. Soc. B 348: 309–320.

Boulinier, T. et al. 2016. Migration, prospecting, dispersal? What host movement matters for infectious agent circulation? – Integr. Comp. Biol. 56: 330–342.

Bovet, P. and Benhamou, S. 1988. Spatial analysis of animals' movements using a correlated random walk model. – J. Theor. Biol. 131: 419–433.

Codling, E. A. et al. 2008. Random walk models in biology. – J. R. Soc. Interface 5: 813–834.

Colizza, V. and Vespignani, A. 2008. Epidemic modeling in metapopulation systems with heterogeneous coupling pattern: theory and simulations. - J. Theor. Biol. 251: 450–467.

Coulon, A. et al. 2015. A stochastic movement simulator improves estimates of landscape connectivity. – Ecology 96: 2203–2213.

Craft, M. E. 2015. Infectious disease transmission and contact networks in wildlife and livestock. – Phil. Trans. R. Soc. B 370: 20140107.

Craft, M. E. and Caillaud, D. 2011. Network models: an underutilized tool in wildlife epidemiology? - Interdiscip. Perspect. Infect. Dis. 2011: 676949.

Craft, M. E. et al. 2011. Disease transmission in territorial populations: the small-world network of Serengeti lions. – J. R. Soc. Interface 8: 776–786.

Cross, P. C. et al. 2005. Duelling timescales of host movement and disease recovery determine invasion of disease in structured populations. – Ecol. Lett. 8: 587–595.

Cross, P. C. et al. 2013. Female elk contacts are neither frequency nor density dependent. – Ecology 94: 2076–2086.

Dahle, J. and Liess, B. 1992. A review on classical swine fever infections in pigs: epizootiology, clinical disease and pathology. – Comp. Immunol. Microbiol. Infect. Dis. 15: 203–211.

Daversa, D. R. et al. 2017. Infections on the move: how transient phases of host movement influence disease spread. – Proc. R. Soc. B 284: 20171807.

Davis, S. et al. 2008. The abundance threshold for plague as a critical percolation phenomenon. Nature 454: 634–637.

DeAngelis, D. L. and Diaz, S. G. 2019. Decision-making in agent-based modeling: a current review and future prospectus. – Front. Ecol. Evol. 6: 237.

Diekmann, O. et al. 1990. On the definition and the computation of the basic reproduction ratio R0 in models for infectious diseases in heterogeneous populations. – J. Math. Biol. 28: 365–382.

Dietz, K. 1976. The incidence of infectious diseases under the influence of seasonal fluctuations. – In: Berger, J. et al. (eds), Mathematical models in medicine. Springer, pp. 1–15.

Dobson, S. F. 1982. Competition for mates and predominant juvenile male dispersal in mammals. – Anim. Behav. 30: 1183–1192.

Doherty, T. S. and Driscoll, D. A. 2018. Coupling movement and landscape ecology for animal conservation in production landscapes. – Proc. R. Soc. B 285: 20172272.

Dougherty, E. R. et al. 2018a. Using movement data to estimate contact rates in a simulated environmentally-transmitted disease system. – BioRxiv (2018): 261198.

Dougherty, E. R. et al. 2018b. Going through the motions: incorporating movement analyses into disease research. – Ecol. Lett. 21: 588–604.

Dye, C. and Hasibeder, G. 1986. Population dynamics of mosquito-borne disease: effects of flies which bite some people more frequently than others. – Trans. R. Soc. Trop. Med. Hyg. 80: 69–77.

Fattebert, J. et al. 2017. Landscape effects on wild boar home range size under contrasting harvest regimes in a human-dominated agro-ecosystem. – Eur. J. Wildl. Res. 63: 32.

Faust, C. L. et al. 2018. Pathogen spillover during land conversion. – Ecol. Lett. 21: 471–483.

Ferrari, M. J. et al. 2006. Network frailty and the geometry of herd immunity. – Proc. R. Soc. B 273: 2743–2748.

Ferrari, M. J. et al. 2011. Pathogens, social networks, and the paradox of transmission scaling. – Interdiscip. Perspect. Infect. Dis. 2011: 267049.

Fisher, D. N. et al. 2017. Analysing animal social network dynamics: the potential of stochastic actor-oriented models. – J. Anim. Ecol. 86: 202–212.

Fofana, A. M. and Hurford, A. 2017. Mechanistic movement models to understand epidemic spread. – Phil. Trans. R. Soc. B 372: 20160086.

Foley, J. E. et al. 1999. The persistence of a SIS disease in a metapopulation. – J. Appl. Ecol. 36: 555–563.

Fortuna, M. A. et al. 2009. The roosting spatial network of a bird-predator bat. – Ecology 90: 934–944.

Fraser, D. F. et al. 2001. Explaining leptokurtic movement distributions: intrapopulation variation in boldness and exploration. – Am. Nat. 158: 124.

Gaillard, J.-M. et al. 2010. Habitat–performance relationships: finding the right metric at a given spatial scale. – Phil. Trans. R. Soc. B 365: 2255–2265.

Godfrey, S. 2013. Networks and the ecology of parasite transmission: a framework for wildlife parasitology. – Int. J. Parasitol. Parasites Wildl. 2: 235–245.

Godfrey, S. et al. 2013. The response of a sleepy lizard social network to altered ecological conditions. – Anim. Behav. 86: 763–772.

Greenwood, P. J. 1980. Mating systems, philopatry and dispersal in birds and mammals. – Anim. Behav. 28: 1140–1162.

Grenfell, B. T. and Harwood, J. 1997. (Meta)population dynamics of infectious diseases. – Trends Ecol. Evol. 12: 395–399.

Grenfell, B. T. et al. 1995. Ecology of infectious diseases in natural populations. – Cambridge Univ. Press.

Grimm, V. and Wissel, C. 2004. The intrinsic mean time to extinction: a unifying approach to analysing persistence and viability of populations. – Oikos 105: 501–511.

Grimm, V. and Railsback, S. F. 2005. Individual-based modeling and ecology. – Princeton Univ. Press.

Grimm, V. et al. 2006. A standard protocol for describing individual-based and agent-based models. – Ecol. Model. 198: 115–126.

Grimm, V. et al. 2010. The ODD protocol: a review and first update. – Ecol. Model. 221: 2760–2768.

Hagenaars, T. J. et al. 2004. Spatial heterogeneity and the persistence of infectious diseases. – J. Theor. Biol. 229: 349–359.

Hanski, I. 1997. Metapopulation dynamics: from concepts and observations to predictive models. – In: Hanski, I. and Gilpin, M. E. (eds), Metapopulation biology. Academic Press, pp. 69–91.

Hess, G. 1996. Disease in metapopulation models: implications for conservation. – Ecology 77: 1617–1632.

Hess, G. et al. 2002. Spatial aspects of disease dynamics. – In: Hudson, P. J. et al. (eds), The ecology of wildlife diseases. Oxford Univ. Press, pp. 102–118.

Holt, R. D. and Hochberg, M. E. 2002. Virulence on the edge: a source-sink perspective. Dieckmann, U. et al. (eds), Adaptive dynamics of infectious diseases: in pursuit of virulence management. Cambridge Univ. Press, 104–120.

Hosseini, P. R. et al. 2004. Seasonality and wildlife disease: how seasonal birth, aggregation and variation in immunity affect the dynamics of *Mycoplasma gallisepticum* in house finches. – Proc. R. Soc. B 271: 2569–2577.

Howells, O. and Edwards-Jones, G. 1997. A feasibility study of reintroducing wild boar *Sus scrofa* to Scotland: are existing woodlands large enough to support minimum viable populations. – Biol. Conserv. 81: 77–89.

Hutchinson, J. M. C. and Waser, P. M. 2007. Use, misuse and extensions of 'ideal gas' models of animal encounter. – Biol. Rev. Camb. Phil. Soc. 82: 335–359.

Jacoby, D. M. P. and Freeman, R. 2016. Emerging network-based tools in movement ecology. - Trends Ecol. Evol. 31: 301–314.

Jeltsch, F. et al. 1997. Pattern formation triggered by rare events: lessons from the spread of rabies. – Proc. R. Soc. B 264: 495–503.

Jeltsch, F. et al. 2013. Integrating movement ecology with biodiversity research – exploring new avenues to address spatiotemporal biodiversity dynamics. – Mov. Ecol. 1: 6.

Jesse, M. et al. 2008. A fully coupled, mechanistic model for infectious disease dynamics in a metapopulation: movement and epidemic duration. – J. Theor. Biol. 254: 331–338.

Johnson, P. T. J. et al. 2010. Linking environmental nutrient enrichment and disease emergence in humans and wildlife. – Ecol. Appl. 20: 16–29.

Kareiva, P. M. and Shigesada, N. 1983. Analyzing insect movement as a correlated random walk. – Oecologia 56: 234–238.

Kays, R. et al. 2015. Terrestrial animal tracking as an eye on life and planet. – Science 348: aaa2478.

Keeling, M. J. and Grenfell, B. T. 1997. Disease extinction and community size: modeling the persistence of measles. – Science 275: 65–67.

Keeling, M. J. and Rohani, P. 2002. Estimating spatial coupling in epidemiological systems: a mechanistic approach. – Ecol. Lett. 5: 20–29.

Keeling, M. J. and Eames, K. T. D. 2005. Networks and epidemic models. – J. R. Soc. Interface 2: 295–307.

Keeling, M. J. and Rohani, P. 2008. Modeling infectious diseases in humans and animals. – Clin. Infect. Dis. 47: 864–866.

Keeling, M. J. et al. 2001. Dynamics of the 2001 UK foot and mouth epidemic: stochastic dispersal in a heterogeneous landscape. – Science 294: 813–817.

Kermack, W. O. and McKendrick, A. G. 1927. A contribution to the mathematical theory of epidemics. – Proc. R. Soc. A 115: 700–721.

King, E. D. A. et al. 2013. Sexual conflict in mammals: consequences for mating systems and life history. – Mamm. Rev. 43: 47–58.

Kramer-Schadt, S. et al. 2009. Individual variations in infectiousness explain long-term disease persistence in wildlife populations. – Oikos 118: 199–208.

Lange, M. et al. 2012a. Efficiency of spatio–temporal vaccination regimes in wildlife populations under different viral constraints. – Vet. Res. 43: 37.

Lange, M. et al. 2012b. Disease severity declines over time after a wild boar population has been affected by classical swine fever–legend or actual epidemiological process? – Prev. Vet. Med. 106: 185–195.

Lange, M. et al. 2016. Relevance of indirect transmission for wildlife disease surveillance. – Front. Vet. Sci. 3: 110.

Leaper, R. et al. 1999. The feasibility of reintroducing wild boar (*Sus scrofa*) to Scotland. – Mamm. Rev. 29: 239–258.

Lloyd, A. L. and May, R. M. 1996. Spatial heterogeneity in epidemic models. – J. Theor. Biol. 179: 1–11.

Lloyd-Smith, J. O. et al. 2005a. Should we expect population thresholds for wildlife disease? – Trends Ecol. Evol. 20: 511–519.

Lloyd-Smith, J. O. et al. 2005b. Superspreading and the effect of individual variation on disease emergence. – Nature 438: 355–359.

Lotka, A. J. 1925. Elements of physical biology. – Williams & Wilkins.

McCallum, H. 2008. Landscape structure, disturbance and disease dynamics. – In: Infectious disease ecology: effects of ecosystems on disease and of disease on ecosystems. Princeton Univ. Press, pp. 100–122.

McCallum, H. and Dobson, A. 2002. Disease, habitat fragmentation and conservation. – Proc. R. Soc. B 269: 2041–2049.

Melis, C. et al. 2006. Biogeographical variation in the population density of wild boar (*Sus scrofa*) in western Eurasia. – J. Biogeogr. 33: 803–811.

Meyers, L. A. et al. 2005. Network theory and SARS: predicting outbreak diversity. –J. Theor. Biol. 232: 71–81.

Mittelholzer, C. et al. 2000. Analysis of classical swine fever virus replication kinetics allows differentiation of highly virulent from avirulent strains. –Vet. Microbiol. 74: 293–308.

Morales, J. M. et al. 2010. Building the bridge between animal movement and population dynamics. – Phil. Trans. R. Soc. B 365: 2289–2301.

Morelle, K. et al. 2015. Towards understanding wild boar *Sus scrofa* movement: a synthetic movement ecology approach. – Mamm. Rev. 45: 15–29.

Nathan, R. et al. 2008. A movement ecology paradigm for unifying organismal movement research. – Proc. Natl Acad. Sci. USA 105: 19052–19059.

Newman, M. E. J. 2002. Spread of epidemic disease on networks. – Phys. Rev. E Stat. Nonlin. Soft Matter Phys. 66: 016128.

Nunn, C. L. et al. 2014. Shared resources and disease dynamics in spatially structured populations. – Ecol. Model. 272: 198–207.

Patlak, C. S. 1953. Random walk with persistence and external bias. – Bull. Math. Biophys. 15: 311–338.

Patz, J. A. et al. 2004. Unhealthy landscapes: policy recommendations on land use change and infectious disease emergence. – Environ. Health Perspect. 112: 1092–1098.

Paull, S. H. et al. 2012. From superspreaders to disease hotspots: linking transmission across hosts and space. – Front. Ecol. Environ. 10: 75–82.

Pepin, K. M. et al. 2016. Contact heterogeneities in feral swine: implications for disease management and future research. – Ecosphere 7: e01230.

Podgórski, T. et al. 2013. Spatiotemporal behavioral plasticity of wild boar (*Sus scrofa*) under contrasting conditions of human pressure: primeval forest and metropolitan area. – J. Mammal. 94: 109–119.

Radchuk, V. et al. 2019a. Transferability of mechanistic ecological models is about emergence. – Trends Ecol. Evol. 34: 487-488.

Radchuk, V. et al. 2019b. The dimensionality of stability depends on disturbance type. – Ecol. Lett. 22: 674–684.

Read, J. M. and Keeling, M. J. 2003. Disease evolution on networks: the role of contact structure. – Proc. R. Soc. B 270: 699–708.

Real, L. A. and Biek, R. 2007. Spatial dynamics and genetics of infectious diseases on heterogeneous landscapes. – J. R. Soc. Interface 4: 935–948.

Rees, E. E. et al. 2011. Understanding effects of barriers on the spread and control of rabies. – Adv. Virus Res. 79: 421–447.

Riley, S. et al. 2015. Five challenges for spatial epidemic models. – Epidemics 10: 68–71.

Rohani, P. et al. 1999. Opposite patterns of synchrony in sympatric disease metapopulations. – Science 286: 968–971.

Rozhnova, G. et al. 2014. Impact of commuting on disease persistence in heterogeneous metapopulations. – Ecol. Complex. 19: 124–129.

Sah, P. et al. 2017. Unraveling the disease consequences and mechanisms of modular structure in animal social networks. – Proc. Natl Acad. Sci. USA 114: 4165–4170.

Sato, K. et al. 1994. Pathogen invasion and host extinction in lattice structured populations. – J. Math. Biol. 32: 251–268.

Sciaini, M. et al. 2018. NLMR and landscapetools: an integrated environment for simulating and modifying neutral landscape models in R. – Methods Ecol. Evol. 9: 2240-2248.

Scherer, C. et al. 2019. Data from: Moving infections: individual movement decisions drive disease persistence in spatially structured landscapes. – Zenodo Digital Repository, <doi:10.5281/zenodo.3387444>.

Sih, A. et al. 2018. Integrating social networks, animal personalities, movement ecology and parasites: a framework with examples from a lizard. – Anim. Behav. 136: 195–205.

Smouse, P. E. et al. 2010. Stochastic modelling of animal movement. – Phil. Trans. R. Soc. B 365: 2201–2211.

Sodeikat, G. and Pohlmeyer, K. 2003. Escape movements of family groups of wild boar *Sus scrofa* influenced by drive hunts in Lower Saxony, Germany. – Wildl. Biol. 9: 43–49.

Spitz, F. and Janeau, G. 1990. Spatial strategies: an attempt to classify daily movements of wild boar. – Acta Theriol. 35: 129–149.

Swingland, I. R. and Greenwood, P. J. 1983. The ecology of animal movement. – Oxford Univ. Press.

Tardy, O. et al. 2018. Interplay between contact risk, conspecific density and landscape connectivity: an individual-based modeling framework. – Ecol. Model. 373: 25–38.

Tildesley, M. J. et al. 2010. Impact of spatial clustering on disease transmission and optimal control. – Proc. Natl Acad. Sci. USA 107: 1041–1046.

Tracey, J. A. et al. 2014. An agent-based movement model to assess the impact of landscape fragmentation on disease transmission. – Ecosphere 5: 1–24.

Turchin, P. 1991. Translating foraging movements in heterogeneous environments into the spatial distribution of foragers. – Ecology 72: 1253–1266.

VanderWaal, K. L. and Ezenwa, V. O. 2016. Heterogeneity in pathogen transmission: mechanisms and methodology. – Funct. Ecol. 30: 1606-162.

Visser, U. et al. 2009. Conservation biocontrol in fragmented landscapes: persistence and parasitation in a host–parasitoid model. – Open Ecol. J. 2: 61.

White, L. A. et al. 2018. Disease outbreak thresholds emerge from interactions between movement behavior, landscape structure and epidemiology. – Proc. Natl Acad. Sci. USA 115: 7374–7379.