Unifying spatial and social network analysis in disease ecology

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Abstract

1. Social network analysis has achieved remarkable popularity in disease ecology, and is sometimes carried out without investigating spatial heterogeneity. Many investigations into sociality and disease may nevertheless be subject to cryptic spatial variation, so ignoring spatial processes can limit inference regarding disease dynamics.
2. Disease analyses can gain breadth, power, and reliability from incorporating both spatial and social behavioural data. However, the tools for collecting and analysing these data simultaneously can be complex and unintuitive, and it is often unclear when spatial variation must be accounted for. These difficulties contribute to the scarcity of simultaneous spatial-social network analyses in disease ecology thus far.
3. Here, we detail scenarios in disease ecology that benefit from spatial-social analysis. We describe procedures for simultaneous collection of both spatial and social data, and we outline statistical approaches that can control for and estimate spatial-social covariance in disease ecology analyses.
4. We hope disease researchers will expand social network analyses to more often include spatial components and questions. These measures will increase the scope of such analyses, allowing more accurate model estimates, better inference of transmission modes,

susceptibility effects and contact scaling patterns, and ultimately more effective disease interventions.

Introduction

Spatial structuring is ubiquitous, and can influence all conceivable intrinsic and extrinsic factors in disease ecology. As such, not accounting for space can weaken analyses (Pawley & McArdle, 2018; Pullan, Sturrock, Soares Magalhaes, Clements, & Brooker, 2012; Tobler, 1970). Although spatial effects can potentially touch any process, social interactions may be particularly vulnerable (Adams, Faust, & Lovasi, 2012). Consequently, the relationship between ecology-driven spatial structure and fine-scale social interactions has shaped the study of animal societies for decades. The recognition that social systems are structured by the surrounding environment rather than comprising random arrangements of independent individuals (Crook, 1964; Crook & Gartlan, 1966) was followed by foundational theory stating that ecological factors influence the spatial distribution of individuals within populations, which in turn determines which individuals interact (Clutton-Brock, 1974; Crook, 1970). Recently, the relationship between spatial structuring and sociality has been addressed in the context of animal social networks (Krause, James, Franks, & Croft, 2015; Webber & Vander Wal, 2019); although relatively well-understood in the context of animal behaviour itself, the role of the environment and spatial behaviour requires addressing more frequently in studies that investigate social correlates of disease.

Spatial behaviour can influence social network analyses of wildlife disease through a few principal mechanisms, which we discuss in Section 3. Fundamentally, it is important to remember that the social environment exists within space, so whom an individual spatially overlaps with defines who they can socially interact with (Whitehead, 2008). Consequently, the spatial and social networks often reinforce, or represent, one another, and their correlation may require controlling for (3A), or can be leveraged for operational purposes (3B). Additionally, social network traits can covary with many spatial processes. For example, many pathogens transmit through the environment, so -in this cases- spatial behaviours define relevant ‘contact events’ rather than social ones, or social contact events may be spatially structured (3C).

Likewise, host immunity and susceptibility are determined by environmentally varying gradients in climate and resource availability, which could counteract or artificiate social effects (3D). Finally, a common question in disease ecology concerns the scaling of contact events with population density, known as "density dependence"; in section 3E, we pose this question as a spatial-social question, and outline how spatial-social methods could be used to address the problem in future analyses.

Ultimately, we summarise how spatial and social behaviour can influence infection (Figure 1), and present a conceptual framework of how to analyse them simultaneously (Figure 2). We start by defining both behaviours (Section 1) and discussing why their unified analysis is relatively rare in disease ecology (Section 2), and then outlining reasons to analyse both where possible (Section 3, described above). To help researchers with tackling spatial-social questions, we then outline methods by which space and sociality can be delineated at the data collection level (Section 4; Box 1), particularly focussing on methods that involve approximating social behaviour with parameterisations of spatial behaviours. We then give case studies for considering spatial-social systems (Box 2), and approaches for simultaneous spatial-social analysis (Section 5). Specifically, we discuss the distinction between controlling for space or sociality, and alternative spatial analysis methods that explicitly quantify both spatial and social processes. Finally, we outline important emerging frontiers and model systems in which the ongoing study of spatial and social behaviour is increasingly important and revealing (Section 6). In doing so, we provide an optimistic guide to conducting spatial-social analyses in the future, encouraging new and exciting investigations in the field of network disease ecology.

1. How to define spatial and social behaviour

We define "spatial behaviour" (or "space") as any representation of an individual's context within its surrounding environment (Pullan et al., 2012). This may comprise point locations in space (Albery, Becker, Kenyon, Nussey, & Pemberton, 2019), movement trajectories (Mourier, Lédée, & Jacoby, 2019), space use distributions (Stopher et al., 2012), or a description of surrounding environmental variables (Saito & Sonoda, 2017). Note that in the latter case, environmental variables are counted as a spatial measure, but by definition they must be taken

relative to an organism's spatial context. For example, if a researcher may be interested in the role of environmental temperature in driving between-individual variation in parasitism, they must first decide whether to use temperature readings from near each animal's point locations, or averaged across each individual's home range. Meanwhile, we define "social behaviour" broadly as any social association between individuals (Croft, James, & Krause, 2008). Dyadic social connections can be inferred from all nature of social associations, ranging from direct interactions involving physical contacts (e.g. grooming, mating, fighting), to implied associations such as co-occurrence in fission-fusion social groupings (e.g. pods of marine mammals, foraging flocks of birds) known as the gambit-of-the-group approach (Franks et al 2010). Crucially, just as incorporating multiple social behaviours and network metrics can help with hypothesis testing (Sosa, Sueur, & Puga-Gonzalez, 2020), simultaneously investigating multiple spatial behaviours can be extremely helpful in revealing the underlying mechanisms in a wildlife system (Albery, Morris, et al., 2020).

2. Why is space understudied in disease ecology social network analyses?

Network disease ecology suffers from a lack of methodological workflows and tools for dealing with spatial-social confounding, contributing to our lack of understanding of the relative importance of spatial and social behaviours. Both are hard to investigate, and studies are rarely designed with both in mind, so assessing them simultaneously can be difficult. Many studies experience operational limitations in detecting spatial variation: for example, ecoimmunological sampling regimes often attempt to minimise spatial variation rather than investigating it directly, rarely use spatial analysis methods, and generally have few spatial replicates (Becker et al., 2020), which may reduce their power to detect spatial variation (Becker et al., 2019). Fitting spatial models can require specialist knowledge which may contribute to the widespread impression that space is more difficult to analyse than social connectivity; however, this is no truer of spatial analysis than it is of social network analysis. Additionally, the field of social network ecology has historically employed network permutations that analytically control for the effect of spatial behaviour to ensure that spatial confounding is not responsible for an observed effect (Farine, 2013). On the contrary, rather than perceiving space simply as something "to

control for", it is far more productive to treat space as an exciting and useful component of a system's biology that is worthy of explicitly quantifying in its own right (Albery et al., 2019; Pawley & McArdle, 2018).

Limitations likewise apply to the collection of spatially explicit social data. Because social behaviour can be hard to observe or infer, some social network analyses use spatiotemporal proximity to approximate social interactions (Farine, 2015; Gilbertson, White, & Craft, 2020; Wanelik, 2019). This method is used frequently enough that tools have been developed to calculate social associations directly from spatiotemporal data (e.g. the *spatsoc* R package; Robitaille, Webber, & Vander Wal, 2019). This heuristic may introduce spatial-social confounding in some systems, and it is not necessarily true that social contacts will correlate perfectly (or even that well) with space, so using one to approximate the other may or may not be valid (Castles et al., 2014; Gilbertson et al., 2020; but see Farine, 2015). The definitions for these behaviours are especially important in disease ecology because the field revolves around pathogens that are spread by contact events arising from them. For example, if a study of directly transmitted pathogens assumes that spatial collocations represent social contacts when in fact they do not, the study may be fundamentally unable to draw accurate conclusions about transmission (Section 3C). It is therefore vital that spatial and social behaviours be defined correctly and delineated from each other for disease network analyses to function as intended (Leu, Sah, Krzyszczyk, Jacoby, & Mann, 2020; Manlove et al., 2018; Richardson & Gorochowski, 2015; Sih, Spiegel, Godfrey, Leu, & Bull, 2018).

Encouragingly, there has been considerable recent progress identifying the importance of separating space and sociality in network studies of animal behaviour (Mourier & Jacoby, 2019; Silk, Finn, Porter, & Pinter-Wollman, 2018; Webber & Vander Wal, 2018; see Case Studies). This push is likewise true in disease ecology, as demonstrated by increasing calls for incorporation of spatial effects in network analyses, particularly where indirectly transmitted pathogens are concerned (Sih et al., 2018; Silk et al., 2019; White, Forester, & Craft, 2017). Moreover, there is increasing conceptual and methodological overlap among the fields of movement ecology, network science, and disease ecology (Dougherty, Seidel, Carlson, Spiegel,

& Getz, 2018; Jacoby & Freeman, 2016). As such, the time is ripe for increased synthesis of spatial and social network methodology in disease ecology studies where possible.

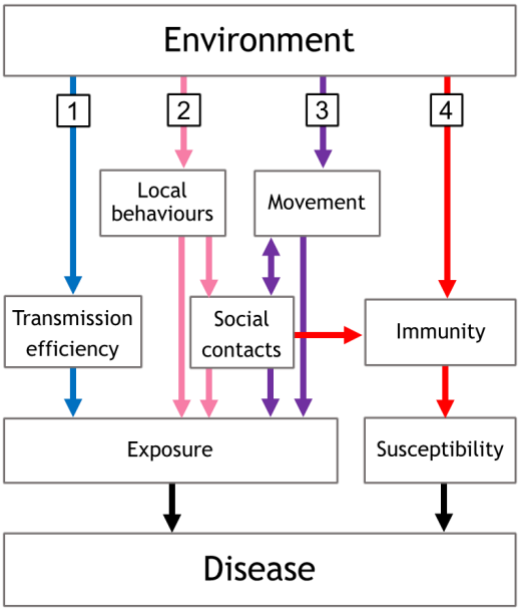

Figure 1: Principal causal pathways among the environment, spatial behaviour, sociality, and disease. 1 (blue lines): Environmental variation in climatic factors affects the transmission efficiency of indirectly transmitted parasites. 2 (pink lines): The environment drives spatial variation in specific social behaviours such as fighting and mating, driving spatial variation in the diseases that are spread by these types of social interactions. 3 (purple lines): Landscape structure and resource distribution determines movement patterns, which themselves determine the social network. Movement patterns determine exposure to indirectly transmitted parasites. The social network determines exposure to directly transmitted parasites, as well as determining susceptibility through changes in resource acquisition and stress. Spatial behaviour and social behaviour can interact. 4 (red lines): The distribution of resources in the environment affects allocation to immunity, creating spatial variation in susceptibility to parasites.

3. Benefits of spatial-social network analysis

Incorporating spatial components into social network analyses can provide important insights into the mechanistic underpinnings of a disease system, as well as potentially offering

operational benefits. Below we consider several of these advantages. Fundamentally, we argue that spatial-social analysis is important because it is challenging to predict where spatial and social behaviours interact, and potentially compete, in influencing disease dynamics. Although spatial-social correlations are common (e.g. Firth & Sheldon, 2016; Mourier & Jacoby, 2019; O'Brien, Webber, & Vander Wal, 2018), these relationships vary considerably across systems, and can be context-dependent (e.g. O'Brien et al., 2018). Unfortunately, little consensus is available on which systems and environments are most likely to exhibit spatial-social correlations due to the rarity of cross-system synthesis. Although recent studies have integrated social networks across a range of animals to make strong comparative conclusions (Sah, Mann, & Bansal, 2018), spatial-social relationships have evaded the same scrutiny. Additionally, fine-scale spatial analyses of wildlife disease are themselves rare and similarly lacking in cross-system comparisons. As such, it is difficult to predict *a priori* which systems and sampling regimes will exhibit the most spatial-social confounding. This uncertainty alone is a strong reason to incorporate spatial analyses into social network studies of wildlife disease.

There likely exist certain systems for which spatial-social analysis is unnecessary, and social network analysis alone is sufficient. However, although it is tempting, we opt not to speculate on these systems for the following reasons: first, the lack of cross-system syntheses means there is currently little empirical evidence which would allow actual assessment, so most such recommendations would be mostly conjecture. Second, the numerous advantages cover so many factors that there are few systems that would not benefit in at least one way by conducting a spatial-social analysis (even if this demonstrated the relative unimportance of space). In the future, greater application of spatial (or spatial-social) analyses of wildlife disease, and increasing application of simulations aimed to answer these questions (e.g. Gilbertson et al., 2020), may help to clarify these issues for a wider range of studies, providing more prescriptive guidelines.

A. Controlling for habitat selection and spatial-social feedbacks

The landscape defines the distribution of resources and potential movement paths, which shapes the structure of the social network through habitat selection (Figure 1; (Albery, Morris, et al., 2020; He, Maldonado-chaparro, & Farine, 2019; Webber & Vander Wal, 2018). Reciprocally,

192 the social environment forms an important component of survival, competition, and dispersal in
193 a heterogeneous environment (Armansin et al., 2019). As such, at fine scales, animals may make
194 space use decisions based on their associates', weighed against environmental cues (Firth &
195 Sheldon, 2016; Peignier et al., 2019). Given this strong mutual causality, it can be difficult to say
196 whether any behaviour represents solely spatial or social processes.
197
198 Empirical attempts to delineate spatial and social behaviour are complicated when considering
199 interactions with disease. Both spatial and social behaviour determine an individual's exposure
200 and susceptibility to infection, and yet behaviour, being highly plastic, can also change in
201 response to infection (Ezenwa, Archie, et al., 2016). For example, sickness behaviours often
202 induce sluggishness and a reduction in social activity (Lopes, 2014; Lopes, Block, & König,
203 2016). It is often mechanistically unclear whether this reduced sociality is an active process,
204 serving e.g. to avoid infecting close relatives or conspecifics, or whether energy-saving
205 reductions in movement merely result in a reduction in sociality by extension (Lopes, Block,
206 Pontiggia, Lindholm, & König, 2018). In addition, parasites commonly affect animals'
207 movement decisions, e.g. through parasite avoidance behaviours, so the spatial distribution of
208 diseases in the environment can determine animals' distributions through a "landscape of
209 disgust" in the same way that predators define a "landscape of fear" (Albery, Newman, et al.,
210 2020; Weinstein, Buck, & Young, 2018). This phenomenon could produce complex covarying
211 patterns: for example, if habitat selection and life history traits covary with immunity and
212 parasite avoidance (Hutchings, Judge, Gordon, Athanasiadou, & Kyriazakis, 2006), the emergent
213 social network could demonstrate artefactual clustering in susceptibility.
214
215 Nevertheless, extricating the roles of spatial and social behaviour in driving disease is not a futile
216 endeavour. Behaviours can be classified on a continuum from "more spatial" (e.g. map locations)
217 to "more social" (e.g. partner choice), and examining and comparing their influence on parasite
218 burden will similarly reveal whether the drivers of parasitism are more likely to be spatial or
219 social. Although some study systems may be poorly suited to spatial-social analysis due to
220 observation difficulties, in most cases fitting both spatial and social behaviours in a model and
221 comparing their effects will likely strengthen inference beyond study designs incorporating only
222 one of the two (see Analysis section).

B. Simplifying measurement approaches

In some circumstances, well-understood spatial-social confounding may be leveraged for operational benefits: for example, streamlining data collection and disease surveillance in wild animal populations with sparse data. Collecting copious GPS data is easier than ever (Kays, Crofoot, Jetz, & Wikelski, 2015) and can be carried out remotely, while social phenomena can be much harder to observe directly (see Box 1). Where spatial data are easier to collect than social interactions, verifying that the two correlate may allow the use of spatial data to approximate social contacts, or social networks and contact events are commonly approximated using parameterised movement data (see below, Box 2 and Section 4). For example, a study of African domestic dog populations used GPS tracking and proximity loggers to demonstrate that individual home range size correlated well with network centrality, which in turn influenced individual propensity to spark simulated rabies epidemics (Wilson-Aggarwal et al., 2019). Similar logic could apply to any system in which ranging behaviour covaries predictably with sociality; however, strong spatial-social correlations are not ubiquitous. Given this uncertainty, we stress that this approach should only be taken cautiously and when accompanied by rigorous validation procedures. In any case, empirical measures of sociality and spatial behaviour will often be imperfect proxies for the interactions that researchers hope to quantify (Farine, 2015). Attempting to incorporate both space and sociality in concert may buffer for this necessity.

C. Identifying pathogen transmission mode

While recent work has considered how the spread of information, or behaviours, may depend on the fine-scale transmission mode between individuals (Firth 2020 TREE), similar considerations also apply to parasite transmission. Indeed, unknown parasite transmission mode is a common reason for conducting spatial-social analyses. Contact events can arise from a variety of spatial/social processes, so the relative importance of spatial and social behaviour depends heavily on the pathogen's transmission mode. Therefore, where transmission mechanisms are unknown, incorporating both spatial and social behaviour helps identify the pathogen's transmission mode, because the behaviour that most closely approximates contact events will best describe variation in infection (Craft, 2015; White et al., 2017). Intuitively, environmental variables will only weakly influence individuals' exposure to directly transmitted pathogens, and

transmission probability will most accurately be represented by social proximity. As such, if space is found to be unimportant relative to sociality, researchers can conclude that direct transmission is likely. For example, in sleepy lizards (*Tiliqua rugosa*), social proximity was a better predictor of *Salmonella* transmission than was spatial proximity, indicating a relatively direct mechanism (Bull, Godfrey, & Gordon, 2012). Conversely, simultaneous use of proximity loggers and GPS tracking revealed that badgers and cattle rarely contact each other directly (despite substantial range overlap), indicating that bovine tuberculosis (*Mycobacterium bovis*) is likely transmitted through the environment (Woodroffe, Donnelly, Ham, Jackson, & Moyes, 2016). An important distinction should be made between pathogens that are transmitted through specific social interactions (e.g., sexually transmitted infections) and those that merely require spatiotemporal coincidence (e.g., aerosol-transmitted viruses). It is possible that both spatial and social behaviours will have detectable, non-interchangeable effects on transmission patterns for the latter group of pathogens, so that both behaviours are needed to gain a full picture of disease dynamics.

Ignoring transmission mode when examining correlates of spatial/social behaviour can produce a confusing picture of a system's ecology. For example, a study in Japanese macaques (*Macaca fuscata*) found that centrality in the grooming network was positively correlated with infection with indirectly transmitted nematodes, which seems mechanistically unlikely (MacIntosh et al., 2012). It is possible that the nematodes' transmission mode is poorly understood, exhibiting a more direct, social component, but it is also possible that the grooming network was spatially structured, so that social network centrality reflected environmental processes rather than sociality itself (MacIntosh et al., 2012). Importantly, because the environment may determine aspects of individual behaviour decisions, some geographic areas may be hotspots for contact events (Albery, Morris, et al., 2020) or for certain risky behaviours, even where the pathogen is directly transmitted. For example, if certain areas lend themselves to fighting or mating grounds for Tasmanian devils (*Sarcophilus harrisii*), this would create enduring spatial variation in the prevalence of Tasmanian devil facial tumour disease despite strictly direct transmission (Figure 1; Hamede, Bashford, McCallum, & Jones, 2009). Therefore, known transmission mode is not sufficient to predict whether or not space is worth investigating in a given host-parasite system, and researchers will benefit from measuring both.

D. Investigating susceptibility effects

Social network analyses commonly focus on the role of social contact events in driving parasite exposure. However, it is important to bear in mind that parasite burden is also a function of susceptibility, that the spatial and social environments can impact host immunity directly, and that these effects may not align (Albery et al., 2019; Becker et al., 2018, 2019). As such, space and sociality should be quantified simultaneously if there is any expectation that they will affect both susceptibility and exposure. Resource supplementation provides an ideal example: increased food should provide more resources for allocation to immunity, reducing susceptibility, yet supplementation commonly leads to aggregation on feeding sites, increasing exposure rates as a result (Becker, Streicker, & Altizer, 2015). Consequently, supplementation could either increase or decrease parasitism, or neither, depending on the balance of these processes. Interestingly, the social environment can also alter susceptibility through stress-induced immunosuppression, potentially counteracting environmental effects on susceptibility or transmission (Ezenwa, Ghai, McKay, & Williams, 2016; Hawley, Etienne, Ezenwa, & Jolles, 2011). Examining both spatial and social behaviour simultaneously may help to extricate sociality-driven changes in susceptibility when examining environmentally transmitted pathogens. One of the foremost advantages of measuring immunity in conjunction with parasitism lies in distinguishing susceptibility- and exposure-driven processes (Bradley & Jackson, 2008). We suggest that studying immunity alongside space, sociality, and parasitism will similarly bolster the strength of inference in determining transmission mechanisms while accounting for susceptibility effects in network disease ecology.

E. Quantifying density dependence

Epidemiological models often make fundamental assumptions about the scaling between population density, contact events, and disease (i.e., “density-dependence”), and the validity of these assumptions can profoundly alter models’ ability to predict disease dynamics (Antonovics, 2017; Hopkins, Fleming-Davies, Belden, & Wojdak, 2020). This question is fundamentally a spatial-social one: how do interactions increase when you add more individuals to the same space? For example, adding more individuals in a given space will generally result in an in-step increase in aerosol inhalation, producing increased contact events for droplet-transmitted

pathogens; however, such increased host density will not necessarily result in a proportional increase in copulation events, so sexually transmitted infections (STI's) are unlikely to scale in this way. As such, STI's are generally considered "frequency-dependent". In reality, all pathogens exist somewhere on a continuum between the two, and identifying where they are placed is an important research priority (Hopkins et al., 2020).

Despite its relative rarity in disease ecology, spatial-social analysis could be incredibly revealing when it comes to empirically identifying pathogens' density dependence and the scaling of contact events. In the absence of disease data, spatial-social analyses could reveal whether increased population density results in a greater frequency of interactions or associations, and this information could be incorporated into epidemiological models. Alternatively, researchers could incorporate both spatial population density and social network metrics at the individual level to identify which best describes disease burden, informing how density and interaction frequency compare (e.g. (Albery, Newman, et al., 2020). Unfortunately, as yet most investigations into density-dependence are conducted *post hoc*, and there is no framework for *a priori* prediction of density dynamics in novel host-pathogen systems. This fact may hamstring efforts to develop epidemiological models and interventions, particularly in the case of novel pathogen emergence, and increasing use of spatial-social approaches could address this gap.

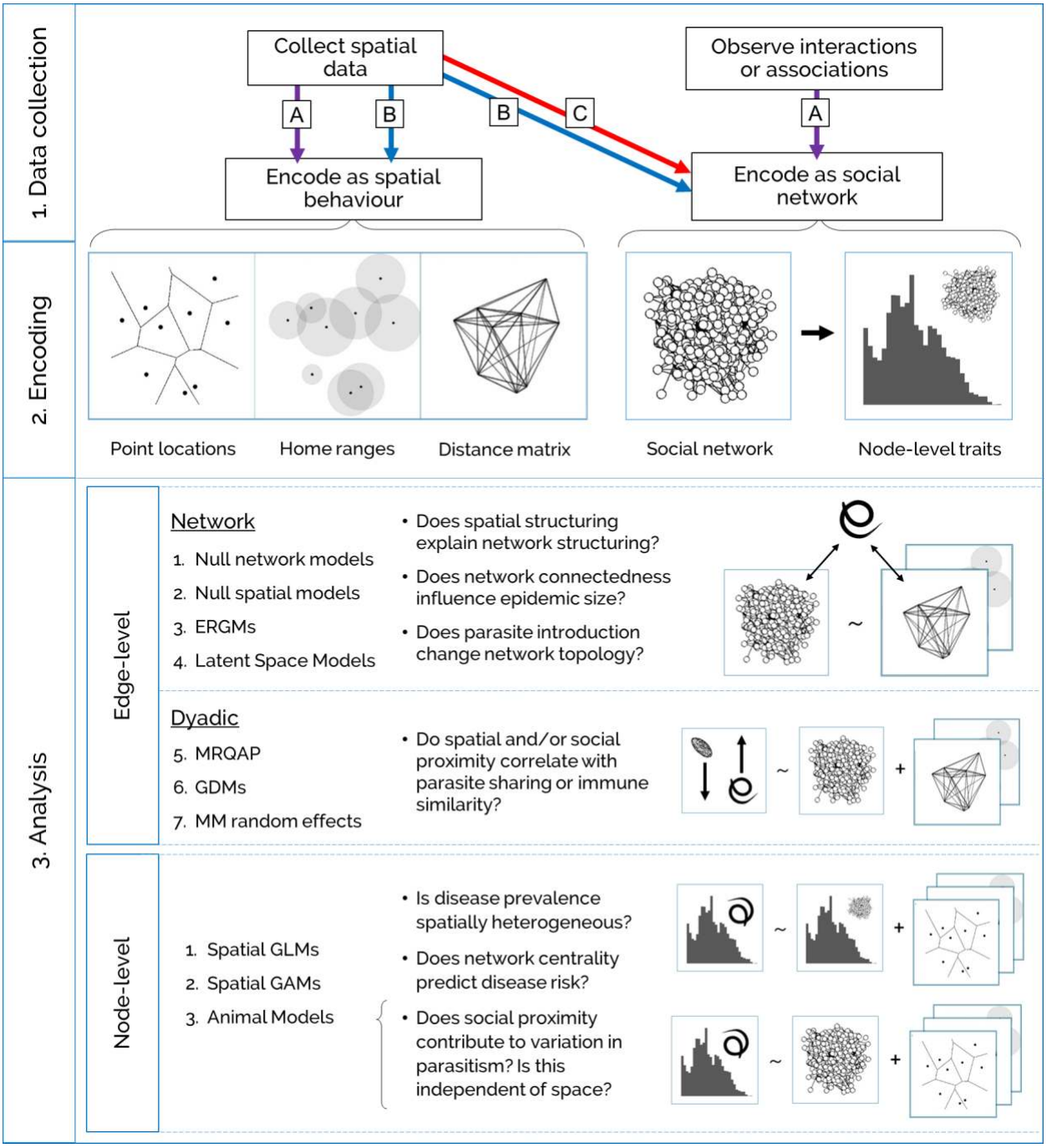

Figure 2: Proposed workflow for collecting, encoding, and analysing spatial data alongside social network data. **Section 1: Data collection**. Purple, blue, and red arrows represent study design options A, B, and C respectively; see "Collecting spatial behaviour with social data". **Section 2: Encoding methods**. Ways to encode spatial behaviour, as either a node-level or dyadic trait. These include: Centroids (point locations) taken from N>=1 observations of individuals. Individual territories have been assigned using Voronoi tessellation (black lines). Point locations can also be used to create home ranges or distance

matrices, or fitted as an autocorrelation function in a statistical model examining node-level traits. Home ranges (grey circles) can be calculated from multiple sightings or derived from movement patterns or kernels, and then coded as a square similarity matrix of range overlaps, to be used in edge-level analyses or as variance components in node-level animal models. Pairwise distances (lines) can be taken between point locations and coded as a square similarity matrix, to be used similarly to home range overlap. Line thickness and opacity are inversely proportional to distance. **Section 3: Analysis methods**. Statistical approaches to analyse spatial-social disease processes and some example questions that each can answer. GLM = Generalised Linear Model; GAM = Generalised Additive Model; Animal Models = a model with a dyadic variance component included; ERGMs = Exponential Randomised Graph Models; MRQAP = Multiple Regression Quadratic Assignment Procedure; GDM = Generalised Dissimilarity Models; MM random effects = Multi-Membership random effects.

Box 1: Methods for collecting spatial and social data simultaneously

Spatial data can take Lagrangian or Euclidean forms, each representing a different way of perceiving movement across the landscape (Nathan et al., 2008; Smouse et al., 2010). Lagrangian data collection (GPS, censusing, and motion tracking) involves the researcher conceptually moving through space, following individuals and summarising their movements. Euclidean data collection (trapping regimes and proximity loggers) uses static sampling locations which collect data on animals moving around them. Lagrangian data are richer and offer greater opportunities for parameterisation; however, Euclidean data collection locations are generally placed by the researcher, so they can be economically distributed in space to cover large areas with minimal effort and/or to accompany visits to locations of biological relevance or experimental manipulation sites (e.g. Firth & Sheldon, 2015). The optimal choice of methods will depend on operational constraints imposed by the study system of interest, e.g. with regards to the size of the animal, the area over which it ranges, and the pathogen and biological process of interest. Here, we outline several methods of spatial-social data collection, including a brief summary of each approach, how they can be used to quantify spatial behaviour and social behaviour, and provide selected illustrative examples from the literature.

GPS: animals are marked and tracked over relatively large distances using satellites.

Spatial: summarise individuals' movements across the landscape.
Social: parameterise activity patterns to identify groups or interactions.
Examples: cattle (Woodroffe et al., 2016); cheetahs (Broekhuis, Madsen, Keiwua, & Macdonald, 2019); feral dogs (Wilson-Aggarwal et al., 2019).

Motion tracking cameras: when the study organism is in a contained space, a large proportion of the population is observed using motion-tracking technology.
Spatial/Social: same as GPS, above.
Examples: carpenter ants (Modlmeier et al., 2019); *Lasius niger* ants (Stroeymeyt et al., 2018).

Census routes: researchers follow a predetermined or random route around a study area and record individual animals' behaviour.
Spatial: record locations of individuals or groups.
Social: record group memberships or interactions between individuals.
Examples: dolphins (Frère et al., 2010; Lusseau et al., 2006); red deer (Stopher et al., 2012).

Spatial proximity loggers: loggers are placed on individuals and in specific environmental locations to identify contact events.
Spatial: use individuals' environmental contact locations to create models of spatial behaviour.
Social: use individuals' contact events to create proximity/interaction/social networks.
Examples: *Mastomys* rodents (Berkvens, Olivares, Mercelis, Kirkpatrick, & Weyn, 2019); great tits (Firth & Sheldon, 2016); European badgers (Woodroffe et al., 2016); reef sharks (Jacoby, Papastamatiou, & Freeman, 2016).

Trapping locations: animals are captured for sampling or camera traps used to identify individuals.
Spatial: record individuals' trapping locations, summarising across repeated trapping events.
Social: record individuals trapped in the same group or within a given spatiotemporal window.
Examples: vole trapping (Davis et al., 2015; Wanelik, 2019); hyena camera traps (Stratford, Stratford, & Périquet, 2019).

Box 2: Frameworks for delineating and analysing spatial and social behaviour

Given the well-understood nature of spatial-social behaviours, there are a great many studies that examine their covariance, and several frameworks have been developed to help untangling and analysing them. Here, we describe some case studies that provide such frameworks to guide researchers carrying out spatial-social analyses of disease processes.

A tripartite network scaffolding for spatiotemporal contact patterns

Manlove et al. (2018) developed a tripartite network which allows characterisation of contact events using three classes of node: space, time, and individuals. Using multiple real-world examples, they demonstrated that this network can be collapsed to form spatial and social networks that are commonly employed in disease ecology. Moreover, the tripartite network was valid for multiple different social systems. Although general and highly flexible, the approach necessitates discretising movement data into spatial nodes, which risks losing information, and the derived contacts are most applicable for directly transmitted parasites (Manlove et al., 2018). An important expansion of the framework will be to incorporate spatiotemporal variation and lag times (Richardson & Gorochowski, 2015; see Timescale Section).

Connecting habitat selection and socio-spatial behaviour with eco-evolutionary consequences.

Webber & Vander Wal (2018) outline a comprehensive eco-evolutionary framework for spatial-social behavioural integration. Specifically, they link individual-level habitat selection behaviours with spatial movements, and then outline how this spatial behaviour results in the development of social networks. They discuss how the resulting framework can be used to examine fitness consequences and ecological dynamics, using animal models, among other approaches (see analysis section). Their incorporation of spatial-social behaviours into quantitative genetic models offers a useful framework for identifying individual-level fitness consequences (and their genetic determinants) while accounting for environmental confounders and density dependence. Their paper offers an interesting scaffold for the investigation of

divergent effects of density-driven susceptibility and exposure effects, and the implied costs and benefits of sociality for disease (Ezenwa, Ghai, et al., 2016).

Networks of networks in reef shark movement ecology

Mourier & Jacoby (2019) used reef sharks as a case study to construct a movement ecology-based framework for spatial-social analysis. In this approach, individuals' movement trajectories are represented as networks, where each node of the network is a Euclidean sampling location, and edges are represented by the individual's movements between these locations. The adjacency matrices from these networks are then nested in a super-adjacency matrix for further analysis, forming a "network of networks". This framework benefits from the fine data resolution it allows, avoiding collapsing individuals' movements into summary statistics such as point locations or space use distributions (Figure 2, Section 2). The authors used this approach to demonstrate high covariance between sharks' spatial and social centrality (Mourier et al., 2019). Like the tripartite model above, this framework is designed for Euclidean sampling locations fixed in space, and has not yet been adapted for Lagrangian data; as such, Lagrangian systems may need to (artificially) discretise their spatial data to take a similar approach.

Competing multiple spatial and social metrics to deconstruct density dependence in a group-living carnivore

(Albery, Newman, et al., 2020) examined parasite burdens in European badgers (*Meles meles*) to investigate socio-spatial drivers. They fitted a series of models with either social metrics (group size and co-trapping networks) or spatial population density, revealing that areas with high population density unexpectedly had lower parasite burdens. Because purely social metrics meanwhile had no detectable effects, cooperative grooming was unlikely to be the cause of the negative density dependence. A series of subsequent analyses revealed that spatial avoidance of parasite transmission was most likely responsible.

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4. Collecting spatial behaviour with social data

If spatial-social analysis is to be carried out, researchers must first collect both data types. Three main study design options can incorporate both spatial and social data collection (Figure 2, Section 1): A) collect both spatial and social data separately, and encode them as different networks; B) collect only spatial data, using spatiotemporal parameters to estimate contact events; or C) collect only spatial data, using these to approximate social contacts without further parameterising – e.g., where spatial proximity is expected to directly represent social proximity. Although the latter is occasionally the only available option for quantifying social behaviour in a given system, we discourage this method for the reasons outlined above.

What spatial measures are available?

Data collection methods for social networks can take many forms, and have been well-reviewed elsewhere (Craft, 2015; Krause et al., 2015; White et al., 2017). Many such methods do not necessarily involve an explicit spatial component, yet they can often be extended to do so with little difficulty. In Box 1, we provide a non-exhaustive list of methods that can be used to collect both spatial and social behaviours simultaneously. Once data have been collected, there are several possible options for encoding spatial behaviour for use in network analyses (Figure 2, Section 2). It is important to consider whether a given spatial measure represents location effects (i.e., where an individual is on a variable landscape) or space sharing effects (i.e., the similarity or proportional overlap of two individuals' spatial environments; Albery, Morris, et al., 2020; Pullan et al., 2012). The two may correlate - e.g., individuals living closer together will share more of their home ranges - but these different types of spatial behaviour can operate differently, potentially offering different insights, and may have additive benefits for inference when considered simultaneously (Albery, Morris, et al., 2020). The relative advantages of the spatial measures used may depend on the system itself: for example, home range overlap will be uninformative for parasitism when species are territorial or at such low density that their home ranges rarely overlap. Pairwise distances and home range overlap matrices can be conceptualised as a spatial network, if this helps with statistical analysis (Figure 2, Section 2; see analysis section; Mourier et al., 2019).

Pairing and delineating spatial and social behaviour

To carry out spatial-social analysis, researchers will need to distinguish social behaviours from spatial activity/occurrence either methodologically or statistically (Figure 2; Box 1). **Methodologically** distinguishing the two involves either combining two data collection methods, each designed to pick up different behaviours, or using multiple types of observations collected by researchers (Figure 2, option A). For example, GPS can provide good wide-resolution spatial data while proximity loggers are used simultaneously to build networks of close-range interactions among individuals (Ossi et al., 2016). Alternatively, researchers conducting behavioural censuses can collect social data by identifying associating or interacting individuals, while also recording spatial locations. The associations/interactions produce a social association network, while the point locations or derived home range estimates provide spatial information.

Distinguishing spatial and social behaviours **statistically** (post-data collection) involves parameterising high-resolution (Lagrangian) behavioural data (Figure 2, option B). For example, GPS-tracking wide-ranging territorial species such as cheetahs (*Acinonyx jubatus*) provides movement data from which contact events can be reasonably inferred purely because individuals rarely come into close proximity of each other (Broekhuis et al., 2019). Meanwhile, the home ranges of the individuals can be independently derived from GPS patterns, and controlled for separately (Seidel, Dougherty, Carlson, & Getz, 2018). Alternatively, study organisms such as ants can be recorded to track the movements of each individual, with contact events identified within this spatial behaviour (e.g. Stroeymeyt et al., 2018). Both of these methods involve selecting defensible criteria for contact events, based on stereotyped behaviours, approach patterns/trajectories (Schlägel et al., 2019), or spatiotemporal proximity (Robitaille et al., 2019). Sophisticated algorithms such as Gaussian mixture models can be used to infer grouping events (Firth et al., 2017; Psorakis et al., 2015) or interactions (Jacoby et al., 2016), avoiding the necessity of defining arbitrary criteria. Encouragingly, even complex, asymmetrical interactions can be identified using only parameterised movement patterns (Jacoby et al., 2016; Schlägel et al., 2019), potentially helping disease ecology researchers to infer specific contact events contributing to transmission.

514 Many studies have examined spatial-social behaviours and their covariance without necessarily
515 tying them to disease ecology; this includes study systems such as great tits (Firth & Sheldon,
516 2016); elk (O'Brien et al., 2018); sharks (Mourier et al., 2019); and many more. Because of the
517 longstanding interest in their simultaneous analysis, several helpful frameworks have been
518 developed; we describe some in Box 2.

519 5. Spatial-social analysis methods in disease ecology

520 Having measured both spatial and social behaviour, statistical approaches must incorporate both
521 data types to compare their effects and/or to ensure they are accounted for when investigating
522 disease dynamics. Controlling for space is a long-standing consideration in ecology (Tobler,
523 1970), so there is no shortage of methods for dealing with spatial structuring. The challenge,
524 then, is incorporating these data into the node-and-edge structure of social network data
525 (Manlove et al., 2018; Mourier et al., 2019; Silk, Croft, Delahay, Hodgson, Boots, et al., 2017),
526 or *vice versa* (Andris, 2016; Mourier et al., 2019). Modelling approaches should take two main
527 forms: investigating the relationship between space and social network structure, and
528 investigating the extent to which space and/or sociality explains variation in disease (or *vice*
529 *versa*). These analyses may take several formats: network-level, dyadic, or node-level (Figure 2,
530 Section 3). The list of network methods we provide is by no means exhaustive, but represents an
531 indicative selection of methods that can be used for spatial-social analysis (Silk, Croft, Delahay,
532 Hodgson, Boots, et al., 2017). For each method, we reference packages or tutorials that can help
533 to carry out the analyses; however, these examples are similarly non-comprehensive, and
534 researchers may seek out and use alternative software in many cases.

535 Considering spatial confounding with network permutations

536 In network ecology, spatial structuring is commonly controlled for by permuting the observed
537 data in a way that maintains the spatial activity of individuals but randomises their social
538 behaviour. These permutations can either be done at the level of the datastream (e.g. randomly
539 swapping individuals' memberships within social groups, but only allowing swaps within the
540 same locations; Farine et al., 2015) or at the network-level (e.g. randomly re-assigning the social
541 network positions of individuals observed in the same place as one another; Firth & Sheldon,

2016). Following the creation of the null networks, any given statistic of interest can then be calculated from them, and the distribution of this statistic expected under spatial structure alone can be generated (Whitehead, 2008). If the same statistic in the observed social network is statistically different from this value, it demonstrates a significant effect above any spatial structuring. This methodology has proven useful for differentiating spatial and social processes, notably in great tits, where individuals' social associations during winter foraging determine subsequent spatial decisions during breeding (Firth & Sheldon, 2016), even more so than expected given winter ranges. Such null network models can be constructed using e.g. the `asnipe` package (Farine, 2013). In a similar sense, "spatially embedded" network models can be used to investigate whether spatial effects can explain social structuring (Daraganova et al., 2012), or spatial measures can be used in concert with contact patterns to derive spatially controlled dyadic traits (Davis et al., 2015), e.g. using the residuals of correlations between spatial and social measures (Whitehead & James, 2015).

Just as 'null social networks' can be created through permuting social behaviour, researchers can create null spatial models (Figure 2) by permuting individuals' spatial activity within the observed dataset while keeping other elements constant. Such methods may aid in comparing the emergent social network to the observed data to investigate whether individuals are actively interacting with (or avoiding) each other, potentially providing insights for disease (Perony, Tessone, König, & Schweitzer, 2012; Richardson & Gorochowski, 2015; Spiegel, Leu, Sih, & Bull, 2016; Woodroffe et al., 2016).

Similarly, permutation can be carried out at any level of the data processing to allow specific null hypothesis testing, whereby particular aspects of the data are controlled for while other aspects are allowed to be randomised. For instance, a permutation may swap the observations within the raw data, or the edges between the nodes in the derived network, or the nodes themselves (Whitehead 2008). In this way, each test comes with its own null hypothesis, and conclusions should be drawn in relation to this hypothesis. For instance, previous studies have noted that permuting the node-level characteristics may be more suited for examining null hypotheses surrounding specific behaviours (Firth et al. 2018) as permuting the raw data under standard datastream permutations only allows for assessing null hypotheses which assume that many

aspects of sociality (such as individual variation in social propensity) are random processes (and thus hold different levels of variation than observed in the real system). <Josh add some sentences about the different levels that permutations can occur at (SS says "node, dyadic and global levels")>.

Furthermore, despite the well-understood nature of network permutations and their widespread use in network ecology, their utility mainly lies in gauging the evidence for the contributions of spatial or social behaviour, rather than accurately gaining estimates of the contribution of both behaviours to a given (disease) phenotype in the form of an effect size (Franks et al 2020 MEE). This is crucial, because (as discussed above) there are many situations in which quantifying spatial effects and directly comparing them with sociality effects is an important component of a study design – for example, where a study aims to identify transmission mechanisms, density dependence, or susceptibility effects (see Section 3). For all such analyses, researchers will likely benefit from approaches that can provide interpretable effect estimates of some sort for both spatial and social behaviours. Similarly, there are specific spatial questions that require alternative spatial analyses: for example, researchers may want to quantify the two-dimensional landscape of network structure, which requires specialised analytical constructs other than standard permutations (Albery, Morris, et al., 2020). All approaches we outline below will provide one or more such pieces of information, allowing greater analytical flexibility, and facilitating a wider range of spatial-social questions. However, it is also noted that each of them can be combined with data permutation tests if deemed useful or necessary, where the tests below can be rerun on different permutations of the observed dataset. Such an approach may, for instance, be useful for initial tests of assurance in these different kinds of tests (e.g. for examining whether the reported test statistics differ from those generated using randomised datasets), for comparing the abilities of different methods, or for drawing general predictions about the dynamics of particular diseases (and our estimates of them) under different reconfigurations of the observed social network (e.g. as done for COVID19 in a real-world human social network - Firth et al. 2020).

Edge-level analyses

Disease analyses commonly aim to investigate how network structure affects pathogen transmission or, reciprocally, how infections alter the network's topology (Craft, 2015; Sah et al., 2018; White et al., 2017). In many cases, multiple spatial and social networks may be necessary to provide clarity on the processes at work: for example, does infection alter the frequency of contact events directly, or does it alter individuals' movements in space, with knock-on effects on the contact network?

Dyadic models

Social, spatial, and disease data commonly comprise pairwise traits between individuals (e.g. distance matrices or pathogen sharing; see Figure 2, Section 2) many of which resist being coded as node-level traits. Analyses that investigate relationships among these data are problematic because similarity matrices are fraught with non-independence: most notably, each row/column represents a replicated individual. Not correcting for this non-independence will inflate the significance of the effects detected, potentially biasing inference. There are a number of specialised ways to deal with non-independence when correlating dyadic data. For example, Mantel tests and Multiple Regression Quadratic Assignment Procedures (MRQAP) produce conservative correlation coefficient estimates and *p*-values through matrix permutations (e.g. VanderWaal, Atwill, Isbell, & McCowan, 2014), and can be carried out using the `asnipe` package (Farine, 2013). Generalised Dissimilarity Models (GDMs) are designed specifically to analyse dyadic data while accounting for non-independence and non-linearities in the data, e.g. when quantifying the relative importance of spatial and social proximity in driving viral transmission in lions (Fountain-Jones et al., 2017). The R package `gdm` will implement them (Manion et al., 2018). Finally, multi-membership random effects can be employed to accurately quantify the importance of node-level traits relative to pairwise interactions (Rushmore et al., 2013), and can be carried out using the packages `MCMCglmm` (Hadfield, 2010) and `mgcv` (Wood, 2011).

ERGMs and Latent Space models

Representing a more complex variation on the theme of dyadic analyses, Latent Space Models (LSMs) and Exponential Random Graph Models (ERGMs) are versatile tools that model edge-level traits as response variables, incorporating both edge- and node-level traits as explanatory variables (Sewell & Chen, 2015; Silk, Croft, Delahay, Hodgson, Weber, et al., 2017; see Silk & Fisher, 2017 for a guide). These variables could include both dyadic spatial/social proximity metrics and individual parasitism, allowing testing of spatial/social components of transmission. Both classes of models can be conceptualised as network-specific adaptations of GLMs, but they differ in the ways they model network structure, and in the process of model fitting (Silk, Croft, Delahay, Hodgson, Weber, et al., 2017; Silk & Fisher, 2017). Importantly, ERGMs may be poorly suited to association-based networks unless sampling biases are absent or well-accounted for (Silk, Croft, Delahay, Hodgson, Weber, et al., 2017; Silk & Fisher, 2017). LSMs and ERGMs can be constructed using `latentnet` (Shortreed, Handcock, & Hoff, 2006) and `ergm` (Hunter, Handcock, Butts, Goodreau, & Morris, 2008), respectively.

Node-level analyses

Network analyses may use node-level traits derived from the social network as response or explanatory variables in statistical models. Below, we outline some ways to control for spatial autocorrelation in network analyses of disease. These models can investigate spatial structuring of social network-derived traits, or estimate spatial processes alongside links between social behaviour and disease.

Spatial autocorrelation variance components

Hierarchical statistical models (i.e., Generalised Linear Mixed Models, or GLMMs) can control for spatial autocorrelation with variance components (random effects), using individuals' point locations to estimate and control for spatial covariance. The analytical workflow for spatial autocorrelation models involves adding the autocorrelation term and comparing it to the base model to investigate whether it changes model fit, accounts for substantial variance, and/or alters fixed effect estimates. In so doing, the spatial effect will account for spatial variation in social behaviour whether sociality is a response or explanatory variable, presenting a good hold-all for

spatial-social disease analyses. Autocorrelation functions include row/column effects (Stopher et al., 2012), wherein individual X and Y coordinates (e.g. latitude/longitude) are fitted as discretised integer values connected by autoregressive processes. Such formulations can be computationally intensive, but modern methods such as the stochastic partial differentiation equation (SPDE) in the Integrated Nested Laplace Approximation (INLA) approach are fast, flexible, and increasing in popularity (Lindgren, Rue, & Lindstrom, 2011; see https://ourcodingclub.github.io/2018/12/04/inla.html for a tutorial). Similar flexible spatial effects can be fitted in Generalised Additive (Mixed) Models (GAMMs), by fitting a tensor smoothing function to individuals' continuous X and Y coordinates. See https://noamross.github.io/gams-in-r-course/ for a tutorial. Available R packages include `mgcv` (Wood, 2011) and `INLA` (Lindgren & Rue, 2015).

Fitting dyadic associations in node-level analyses

Dyadic variance components offer a useful alternative to point-location-based autocorrelation functions, particularly because they allow easy mixing of node-level and dyadic traits in familiar statistical models. Quantitative genetic analyses commonly fit a square matrix of genetic relatedness in the variance component of an "animal model" to estimate genetic heritability in the response variable (Kruuk, 2004). Because these models allow the fitting of multiple such matrices, the models have been supplemented with home range overlap matrices (Stopher et al., 2012). This approach allows extrication of environmental and genetic sources of variation, and can be extended to use social association matrices (Frere et al., 2010; Thomson, Winney, Salles, & Pujol, 2018) to differentiate spatial and social contributions to a given phenotype. For example, do individuals that associate more often have more similar pathogen intensities? Does this result hold when space sharing is accounted for (Webber & Vander Wal, 2018)? These models can be carried out in linear modelling packages including `MCMCglmm` (Hadfield, 2010), `ASReml` (Gilmour, Gogel, Cullis, & Thompson, 2009), and `INLA` (Holand, Steinsland, Martino, & Jensen, 2013).

Considering analytical timescales

The selection of an appropriate timescale is often a necessity of spatial-social analyses, and many available frameworks for spatial-social analysis struggle with incorporating temporal

dependence. The choice of analytical timescale can have dramatic effects on a study's conclusions: for example, Springer, Kappeler, & Nunn (2017) simulated environmental and direct transmission of gastrointestinal parasites in a lemur population, finding that dynamic networks resulted in larger outbreaks than static equivalents. The options for spatial timescale are numerous: a study could use nest or burrow locations to study distributions of vector-borne parasites (Wood et al., 2007) or to investigate whether distance and infection correlate (Bull et al., 2012), or researchers could link chronic parasite infections with an individual's average location over a predetermined timescale – e.g., the previous year (Albery et al., 2019). Landscape structure and climatic conditions can interact with time-dependent habitat selection behaviours, creating spatiotemporal coincidence of individuals and thereby encouraging social associations. Within each study system, researchers need to establish which time periods should be used to summarise an individual's spatial movements and social interactions, and how these behaviours apply to pathogens of varying infectious periods and development times.

Crucially, associations through spatial behaviour can transcend time: that is, individuals can have meaningfully overlapping home ranges even if they were never alive at the same time (Jacoby & Freeman, 2016). In contrast, social contact requires spatiotemporal coincidence (Manlove et al., 2018; Whitehead, 2008). Spatial behaviours' time-independence could be a positive or a negative, depending on the question to hand, and researchers must consider the timescale of the pathogen. For example, space use combined with a temporal delay may be the best way to describe transmission of certain parasites, but not others (Gilbertson et al., 2020; Manlove et al., 2018; Richardson & Gorochowski, 2015). Furthermore, if local environmental variation is stable over long time periods and influences disease risk, spatial associations may predict disease similarity even in the absence of any possible social contacts (i.e. across non-temporally-overlapping generations). This knowledge could inform which behaviours could be important when modelling transmission dynamics – and, conversely, comparing the importance of (temporally lagged) spatial and social behaviours could illuminate the transmission modes or epidemiological dynamics of a given pathogen (e.g. Albery, Newman, et al., 2020; Springer et al., 2017; see Section 3C).

The repeatability of behaviour (sometimes conceptualised as "personality") is an important, rapidly developing area of research (Dingemanse & Dochtermann, 2013; Moirón, Laskowski, & Niemelä, 2019) which is also often considered for movement behaviours (Jacoby & Freeman, 2016; Webber et al., 2020; Webber & Vander Wal, 2018) or social behaviours (Aplin et al. 2015; Firth et al. 2017; Krause et al. 2017). If behaviour is highly repeatable across time, e.g. where individuals inhabit similar home ranges from year to year (Stopher et al., 2012), timescale problems may be somewhat avoidable. This will also depend on the pathogen of interest: environmental parasites may have more constant spatial hotspots driven by consistent climatic factors, so that lifetime home ranges capture substantial variation in parasitism; meanwhile, directly transmitted parasites may exhibit waves of transmission across the population, such that spatial hotspots are more ephemeral and a restricted analytical timescale is vital. Fortunately, many of the analytical frameworks we describe are able to incorporate temporal structures: for example, INLA can fit fluctuating spatiotemporal fields across years and seasons (Albery et al., 2019), and temporal ERGMs (tERGMs) can handle changing network structures through time (Silk, Croft, Delahay, Hodgson, Weber, et al., 2017). Thus, even the enduring problem of timescale selection is solvable when interactions between environment, movement, sociality, and parasitism are understood and analysed properly.

6. Synthesis and future directions

We have so far provided a guide to carrying out spatial-social network analysis in disease ecology, from conception through to analysis. In this section, we discuss ideal empirical systems for addressing spatial-social questions, and we detail potential benefits emerging from the unification of spatial and social analysis.

Model systems

Meta-analysis is a promising option for large-scale investigation of spatial-social influences in disease ecology. The number of published social network analyses has increased exponentially in recent years (Webber & Vander Wal, 2019), and repositories of network data are becoming available as a result (Sah, Méndez, & Bansal, 2019). These resources can help to compensate for the lack of cross-system synthesis in this field so far. By analysing contact data alongside spatial

742 behaviour across the published literature, we can ask broadly informative questions such as: how
743 many social network analyses include spatial data? How often are space and sociality highly
744 correlated? How might this impact studies' findings? Such analyses may identify general
745 indicators of when and where to be concerned about space for social network analyses (and even
746 for disease ecology studies in general), as well as potentially testing the criteria laid out in this
747 review. Furthermore, even if pathogen data are not available for the large majority of spatial-
748 social network datasets, empirically parameterised simulations of disease spread within a meta-
749 analytical framework (e.g. Sah et al., 2018) could be a useful tool for gaining a general
750 understanding of how spatial and social drivers of disease can be untangled, and which kinds of
751 systems and network structures best allow this separation.
752
753 Many empirical systems lend themselves to spatial-social analysis. Fundamentally, any system
754 with extricable/tractable social and spatial behaviour could be used for such analyses, and
755 fission-fusion social systems may be especially well-suited for this reason: censuses and GPS
756 records can regularly identify individuals' group memberships separately alongside their spatial
757 locations, allowing untangling of spatial-social associations (Box 1). Such systems include many
758 well-studied animals, such as dolphins (Lusseau et al., 2006), great tits (Firth & Sheldon, 2016),
759 and deer (Stopher et al., 2012). Ants likewise represent a promising model system for this
760 reason: using motion-tracking cameras, spatial behaviour can be tracked and then social contacts
761 extricated (Modlmeier et al., 2019; Stroeymeyt et al., 2018): for example, trophallaxis or
762 physical touch events can be used to create a contact network, while space use distributions or
763 movement trajectories are used to characterise their spatial behaviour. Although the two will
764 correlate, there is likely to be considerable testable variation: that is, of the ants that overlap in
765 space with one another, only a subset of dyads will give or receive trophallaxis to each other
766 (Modlmeier et al., 2019). Ants' social networks respond predictably to spatial changes
767 (Modlmeier et al., 2019) and pathogen presence (Stroeymeyt et al., 2018), with group-level
768 trends emerging from predictable individual-level behaviours, lending them well to high-
769 resolution movement models.
770
771 Knowledge of a wide range of different pathogens is a further advantage for a potential study
772 system, particularly because this may allow testing of the spatial-social continuum that we

outlined in the pathogen transmission section above. Rodents are some of the best-studied model systems for disease ecology, yet because rodents are generally too small for battery-powered high-resolution GPS tracking, the tools available for studying their spatial behaviour at high resolution in the wild are limited. To fill this gap, the development of lightweight bluetooth technology has facilitated the use of highly sensitive proximity loggers in wild *Mastomys* mice (Berkvens, Olivares, Mercelis, Kirkpatrick, & Weyn, 2019). Using environmentally placed loggers with wide ranges and extended battery lives, it is possible to collect regular spatial locations alongside social contact data, providing an exciting model system with which to investigate space and sociality simultaneously (Berkvens et al., 2019). This methodology could be combined with the considerable literature on trapping-based contact networks in field voles (Davis et al., 2015; Wanelik, 2019) and other rodents (e.g. Grear et al., 2009). Notably, sleepy lizards (*Tiliqua rugosa*) have recently been proposed as an ideal system for the integration of social and spatial analyses, particularly focussing on ectoparasite transmission, and with many exciting future opportunities for joint spatial-social analyses (Sih et al., 2018). As such, the list of potential systems is phylogenetically diverse and extremely promising, with many opportunities for further specialisation under this umbrella.

Connecting environmental, animal, and human health with spatial-social analyses

Unlike human systems where linking real-world disease dynamics to real-world social contact networks is exceptionally rare despite much interest (Firth et al. 2020), there is a great number of real-world social contact network monitoring efforts from natural animal systems (Sah et al. 2017; Sah et al. 2019) meaning these hold unique potential to contribute to understanding broader societal issues relating to disease spread and health. Specifically, aside from strengthening inference and improving model accuracy, the potential practical benefits of unified spatial-social analysis for disease ecology are numerous. Integration will improve our ability to investigate transmission mechanisms and density dependence, while conveying operational benefits (Section 3). Furthermore, better empirical understanding will inform the relevant spatiotemporal scales of transmission dynamics, providing parameters for scalable models of spatial movement that implicitly or explicitly account for social contact-driven transmission

events within them (White, Forester, & Craft, 2018). Building on rapidly developing interest in disease-behaviour-network feedbacks (Section 3A), spatial-social analyses could integrate existing models of spatial-social feedback (e.g. Firth & Sheldon, 2016) with those that identify reciprocal changes in network topology in response to disease transmission (e.g. Stroeymeyt et al., 2018).

All such endeavours will help to predict how altered behaviour will affect disease transmission (and *vice versa*) in the wake of large-scale community perturbations. This includes short-term events (e.g. zoonotic outbreaks or catastrophic events), long-term trends (e.g. climate change-induced alterations to global transport systems), or behavioural animal health interventions (e.g. translocations), all of which will alter contact patterns separately from spatial movements. For example, individual variability in raccoon ranging behaviour can reduce the effectiveness of rabies vaccination interventions (McClure, Gilbert, Chipman, Rees, & Pepin, 2020). Understanding how landscape structure alters raccoons' spatial behaviour, and therefore disease spread, will help to anticipate geographic variation in intervention success. As another example, it is well established that culling British badgers (*Meles meles*) is an ineffective method of control for bovine tuberculosis (*Mycobacterium bovis*). The culling-associated disruption of local population structure provokes badgers to disperse, moving further than they otherwise would and making more social contacts in the process (Carter et al., 2007; Ham, Donnelly, Astley, Jackson, & Woodroffe, 2019; Tuyttens et al., 2000). As such, this perturbation of the social network induces a spatial movement, which is expected to result in a subsequent rearrangement of the social contact network. These changes in network structure may facilitate *M. bovis* spread across the countryside, directly contravening the intended control efforts by infecting cattle in surrounding areas (Donnelly et al., 2007). This example is hard to conceptualise without considering the social and spatial networks in tandem, as well as considering the landscape itself. Under rapid ongoing global change, a proper understanding of the links between the environment, animal movement, and social behaviour will be crucial for understanding how disruptions and natural disasters such as fires, floods, and hurricanes will impact wildlife disease (Silk et al., 2019). Studies have already connected ongoing ecological tragedies such as fire with animal movement and one health consequences (Bonilla-Aldana et al., 2019), and spatial-social analysis is set to be an invaluable tool for anticipating and combatting their effects.

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