When to choose dynamic versus static social network analysis

Damien R. Farine

Department of Collective Behaviour, Max Planck Institute for Ornithology, 78457 Konstanz, Germany
Chair of Biodiversity and Collective Behaviour, Department of Biology, University of Konstanz, 78457 Konstanz, Germany
Edward Grey Institute, Department of Zoology, University of Oxford, Oxford, OX1 3PS, UK
Email: dfarine@orn.mpg.de

Abstract

1. There is increasing interest in using dynamic social networks in the study of animal sociality and its consequences. However, there is a general lack of guidance on the when and how such an approach will be valuable.
2. The aim of this paper is to provide a guide on when to choose dynamic versus static social network analysis, and how to choose the appropriate temporal scale for the dynamic network.
3. I first discuss the motivations for using dynamic animal social networks. I then provide guidance on how to choose between dynamic networks and the 'standard' approach of using static networks. I discuss this in the context of the temporal scale of changes observed, of their predictability, and of the data availability.
4. Dynamic networks are important in a number of scenarios. First, if the network data are being compared to independent processes, such as the spread of information or disease or environmental changes, then dynamics networks will provide more accurate estimates of spreading rates. Second, if the network has predictable patterns of change, for example diel cycles or seasonal changes, then dynamic networks should be used to capture these changes. Third, dynamic networks are important for studies of spread through networks when the relationship between edge weight and transmission probability is non-linear. Finally, dynamic social networks are also useful in situations where interactions among individuals are dense, such as in studies of captive groups.
5. The use of static versus dynamic network requires careful consideration, both from a research question perspective and from a data perspective, and this paper provides a guide on how to evaluate the relative importance of these.

Keywords: disease transmission, group living, information transmission, social network analysis, social organisation

Introduction

All animal populations are, in some way, dynamic. Many factors can shape patterns of connections among individuals, thus changing the social environment that individuals experience (Edenbrow, Darden et al. 2011, Leu, Farine et al. 2016). Broadly, such changes can impact population social structure (Ansmann, Parra et al. 2012) and population processes, such as the spread of disease (MacIntosh, Jacobs et al. 2012, VanderWaal, Atwill et al. 2013, VanderWaal, Atwill et al. 2013, Weber, Carter et al. 2013, Adelman, Moyers et al. 2015) and information (Aplin, Farine et al. 2012, Allen, Weinrich et al. 2013, Aplin, Farine et al. 2015, Farine, Aplin et al. 2015, Firth, Sheldon et al. 2016). As a consequence, the dynamic changes in the patterns of connections among individuals can have major impacts on the evolution and maintenance of group living. Yet we have little understanding of how such changes play out at the individual-level, and how the responses of individuals to change drive population-level outcomes.

A useful tool for quantifying how individuals are connected is social network analysis (Whitehead 2008, Farine and Whitehead 2015, Krause, James et al. 2015). Social networks capture information about individuals (nodes) and the relationships (edges) between them, allowing us to scale from the individual level ('what is the social environment in which individual *i* is embedded?') through to the population ('what is the overall structure arising from the connections among individuals?'). However, these relationships have a temporal component – individual relationships come and go through time (for example with season, or due to births and deaths), while relationships form and disband. Thus, there is increasing call for studies of animal social networks to take such temporal dynamics into account (Blonder, Wey et al. 2012, Hobson, Avery et al. 2013).

In addition to better understanding the nature of social relationships *per se*, dynamic social network analysis is important for studying population processes. Both disease and information transmission have an inherent temporal element: the status of individuals from susceptible to infected, or naïve to knowledgeable, changes

progressively over time. Thus, being able to link the dynamic patterns of connection in time with the changing status of individuals will surely provide more robust conclusions and predictions about the role of social structure in shaping population processes.

However, there is little guidance available about when dynamic networks should be implemented, and when they are likely to be most useful. Dynamic networks can increase the temporal resolution we use to study animal behaviour, but this can sometimes come at the cost of precision. Further, many studies seek to understand general biological patterns, which can be at odds with the specificity of a network constructed at a fine temporal scale from one group or population. Finally there is the question of what temporal resolution is most appropriate when implementing dynamic networks. Such questions impact both studies focused on the dynamic changes in the social network directly (e.g. Franz, Altmann et al. 2015, Ilany, Booms et al. 2015), and those that aim to understand the links between population-level network structure and population processes (e.g. Springer, Kappeler et al. 2017). Thus, while dynamic networks are clearly important in some contexts, they may not be necessary or appropriate for all studies.

In this paper, I provide some guidelines on when it might be useful to analyse observed data as a dynamic network, and how to decide on its temporal resolution. Many of the considerations associated with constructing and interpreting networks are explained in detail elsewhere (e.g. Craft 2015, Farine and Whitehead 2015), and so my focus will be exclusively on addressing their temporal dimension. However, I do note that how the data for networks are collected can influence the inferences we can draw from our models (Perkins, Cagnacci et al. 2009). In that context, I briefly discuss issues of data quality. Finally, I highlight how moving to a finer temporal resolution, while not necessarily important for a given biological question, can help reveal patterns in the data that might be hidden when using a static approach.

General methodological issues

There are two general methodological issues that are prominent to dynamic social network analysis. The first issue is whether enough data are available to construct each of the temporal snapshots of the network. Because networks contain many more relationships (edges) than individuals (nodes), constructing a network requires a great deal of data (Farine and Strandburg-Peshkin 2015). If multiple networks are

constructed (i.e. a dynamic network), then each of these should each contain sufficient data to construct robust networks and have similar (or equal) sampling efforts. Of course all data used to construct the network(s) should be sampled in exactly the same way, as sampling methods can have major implications on the resulting network. Hypothesis testing should also be based on null models, which can help to control for some of the differences among samples (see Farine 2017).

A second issue is that it is widely considered to be problematic to compare differently-sized networks (Croft, James et al. 2008). For example, the population in time period 1 might be larger or smaller than the population in time period 2. Thus, we expect that individuals will have fewer associates (a lower degree) in the smaller network (because there are fewer individuals with whom to associate). On this point, it is important to consider whether the observed change is important biologically. Having a smaller group size will matter for individuals, and so the fact that we *a priori* expect them to have fewer associates does not preclude this having an impact on their behaviour or fitness. Thus, many network comparisons that would be invalid in some cases could be legitimately applied to the same population across time. However, if the difference in the size of the networks is due to methodological reasons, for example fewer samples were collected in time period 2, resulting in fewer observed individuals, then there is clearly a cause for concern.

When are network dynamics important?

Social networks dynamics are applicable to studies of changes in the network structure itself, and studies of transmission dynamics. The former is largely aimed at understanding the building blocks of social systems, whereas the latter is focussed on understanding processes that could be good or bad for individuals in that system. Here I outline different aspects of these two questions that warrant considerations when designing a study and having to decide whether to take a dynamic approach.

Network structure

As the environment changes, so does the social behaviour of many animal species. For example, in great tits, birds form loose mixed-species flocks during the winter but hold territories during the spring breeding season (note that I consider both to represent

social behaviour). This change raises several questions. Do relationships during the winter influence those in the spring? Does winter social behaviour influence reproductive success? In the case of great tits in Wytham Woods, the answer to both questions is yes: birds that winter together breed closer (Firth and Sheldon 2016), and individuals' social environment during the winter is an important predictor of their fitness (via social selection, Farine and Sheldon 2015). More broadly, although the woodland appears to have two distinct states, the behaviour of individual birds is, in fact, temporally-linked across these states (or seasons in this case).

Dynamic social network analysis will be important for understanding how changes in the environment impacts social structure. Drought, floods, fire, and a range of other impulse events can have profound impacts on how animals interact (or with whom), subsequently affecting competition and reproductive success. While many such questions have been explored by ecologists, social network analysis provides a novel window by allowing the impacts to be quantified at the individual level, and the links between past, current, and future behaviour to be carried across contexts.

In addition to broad environmental change, individual traits, such as dispersal status, social rank, or age, can change how animals behave over time. It has also been suggested that the presence or absence of particular individuals can impact the structure of interactions in animal social networks (Flack, Girvan et al. 2006, Barrett, Henzi et al. 2012, Franz, Altmann et al. 2015). Dynamic social network analysis, such as comparing networks before and after a disappearance, has the potential to reveal a great deal about the mechanisms and consequences of particular animals on the social lives of others. For example, which individuals change behaviour most? How do changes result in changes in social network position? What are the impacts on global properties of the social networks in which these individuals are embedded?

An important question when studying dynamics of networks is whether changes in the network are homogenous, or centred on particular individuals (Franz and Alberts 2015). For example, the loss of an individual would reduce the mean degree in the network (there are fewer opportunities to interact). A key question is whether this occurs equally across all individuals (i.e. the relative rank of degree remains stable) or if it changes disproportionately among individuals. For example, in female vampire bats that can rely on food sharing for survival, the loss of an individual from the colony will

disproportionately impact individuals with fewer social bonds (Carter, Farine et al. 2017).

Transmission dynamics

A major aim for studying changing network structure is to understand or predict the effects these changes will have on information or disease transfer. The use of networks for studying transmission dynamics can generally be split into two alternative aims: (i) trying to model or understand an observed epidemic (e.g. Bull, Godfrey et al. 2012, VanderWaal, Atwill et al. 2013) or information spread (Aplin, Farine et al. 2015), and (ii) making general predictions about the susceptibility of a population with a particular social structure to a new disease (e.g. Hamede, Bashford et al. 2009, Springer, Kappeler et al. 2017). These quite different aims also require different considerations when it comes to employing dynamic network analysis. For simplicity, below I mostly focus on studies of disease, but the same considerations are applicable for information transmission.

(i) Studying observed patterns

Improvements in analytical tools, including social network analysis, have greatly enhanced our ability to draw inferences from observed epidemics. For example, Brockmann and Helbing (2013) used data from two global epidemics to highlight the role of air travel as a pathway of disease transmission. Similarly, VanderWaal, Atwill et al. (2013) found that the similarity in *E.coli* infections in giraffes (*Giraffa camelopardalis*) more closely resembled their patterns of social association rather than shared use of waterholes, while social networks predicted similarity in salmonella infections of sleepy lizards (*Tiliqua rugosa*) (Bull, Godfrey et al. 2012). Several studies have also inferred rates of information transmission through networks of animals in the wild (Aplin, Farine et al. 2012, Allen, Weinrich et al. 2013, Hobaiter, Poisot et al. 2014, Aplin, Farine et al. 2015, Farine, Aplin et al. 2015). In all these cases, studies matched networks derived from empirical observations with some independently-measured data on disease (or individual behavioural states) to generate insights into transmission processes.

Better accounting for the dynamic ordering of connections among individuals when constructing social networks will be most important when studying real (as opposed to simulated) diffusion processes, because 1) this will better reveal the transmission

pathways, and 2) it will allow more accurate estimations of the transmission rates. Having information about both the timing of the contacts among individuals (or opportunities for transmission) and the probable occurrence of transmission events will greatly improve the ability to draw conclusions about how associations between individuals promote transmission. For example, take a situation where different groups of animals each have their own source of water. Then say that a drought hits, and for a few weeks most of the waterholes are dry, forcing the population to aggregate at just a few waterholes. Taken over a long study period, static networks representing shared waterhole use will have strong within-group ties, and very weak out-group ties. However, if a sick individual was present during that drought, then that individual could have passed on pathogens to the entire population during that very short period of time, which would be better reflected by the short-term but strong out-group ties during the drought. The outcome of this hypothetical scenario could be even more important if periods of drought coincide with immunodeficiency from heightened stress that increases transmission probability. Thus, a dynamic network, here constructed around the temporal change in climatic conditions, could increase the perceived importance of shared waterhole use for disease transmission relative to the importance placed on shared waterhole use based on a static network.

A static network will also always contain many edges that were not necessarily present at the time when a transmission event actually occurred. This could be important when estimating transmission rates. For example, a static network (Figure 1B) could erroneously suggest that transmission is not always likely between an infected individual and a susceptible individual, when in fact a dynamic network implementation (Figure 1A) would correctly reveal a 100% transmission rate. Note that this is a fundamentally different question to the perspective often presented that a dynamic network is important when simulating outbreaks (see next section) because it captures the fact that not all individuals could become infected based on a specific start point. For example, if either of the lower nodes (those that are blue on Day 1) in Figure 1 first acquired the disease, then the top-most node (shown using the *) could never become infected, which is obvious when using a dynamic network because by the time the disease would reach the right-most node there would be no connections to the top-most node (Figure 1A). However, this detail is lost when using a static representation (Figure 1B).

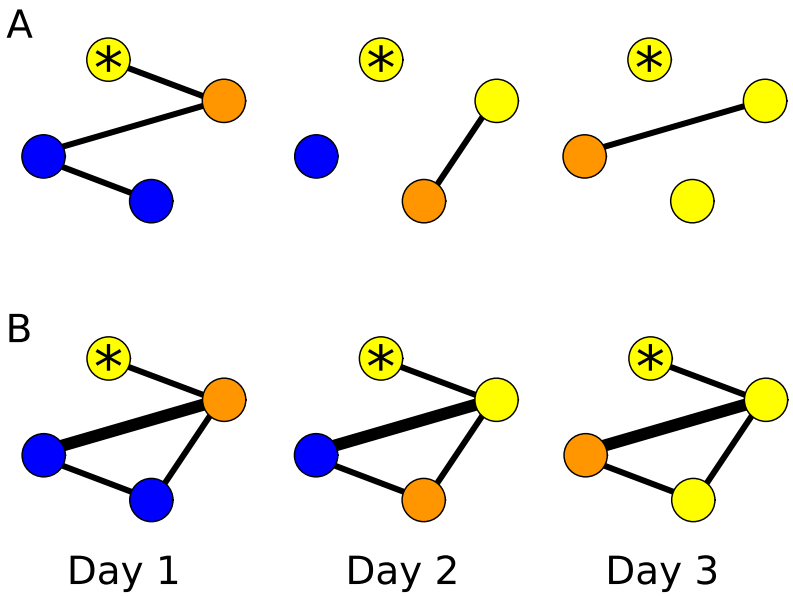

Figure 1. Illustrating the potential importance of accounting for network dynamics when quantifying transmission rates in an observed epidemic. In this toy example, the original individual carrying the disease is marked by a *. Infected individuals can transmit to non-infected individuals (blue), and individuals are marked orange on the day they become infected, but cannot pass on the infection on the same day. (A) The infection can be modelled using a dynamic network. On day 1, the original carrier infects the only individual it is connected to. On the second day, the disease is transmitted between the newly infected individual and its new social partner. On the third day, the disease transmits to the last remaining individual. No doubt this is a highly virulent pathogen. This is clear when taking a dynamic network perspective (A): whenever an edge is present between an individual with pre-existing disease (yellow) and an uninfected individual (blue), the latter becomes infected (orange). Thus, we can conclude that there is a 100% transmission probability. By contrast, when using a static network (B), the disease is not observed to transmit through the double-strength edge on day 2, meaning that the inferred transmission rate would be below 100%.

(ii) Making general predictions

In contrast to studying observed epidemics, many studies have focused on modelling the potential outcome of a pathogen (or new information) entering a network. In this case, simulations involve introducing a disease at one (or more) random individuals at a random point in time. Here, the value of dynamic networks becomes less clear. The crux

of the argument I will lay out below is that when the aim of modelling transmission processes on networks is to make relatively general predictions (such as the size of an outbreak rather than the specific pathway it takes), then static networks are often better representations of an average population. By contrast, a dynamic network is a specific case—generally relevant only to the specific time period in which the population was studied. Simulating an outbreak on an observed dynamic network might reduce the generality of the predictions.

The key question that one should ask when deciding whether to take a dynamic approach to modelling transmission through networks is: are the dynamics repeatable? Because the aim is to simulate a disease entering the population at a random point in time, then observed dynamics are only informative if they are likely to re-occur. Some consistent patterns in the ordering or strengths of connections are clearly important to account for. For example, many animals demonstrate seasonal changes in the patterns of connection among individuals, such as the transition into a breeding season (e.g. Brent, MacLarnon et al. 2013, Rushmore, Caillaud et al. 2013), and these are an important and repeatable part of their life histories. If a future epidemic enters the population, we expect the population to experience these same changes in the same order, and thus accounting for seasonal change will be important for making predictions about transmission outcomes. Take air travel as another example. If we wish to predict the speed at which an epidemic would spread through the global human population, we might well choose to compare a scenario in the middle of May with a scenario where an epidemic hits in late December.

However, accounting for the very fine-scale detail about who came into contact first versus second is often much less important when making general predictions about how an epidemic spreads through a population. Figure 1 highlights why taking a dynamic network approach is beneficial for studying observed epidemics. But, if our aim is to model what could happen at some unknown time in the future, are these dynamics equally important? Say individual A contacts B, then B contacts C; we might conclude that C can never infect A. Now say we re-observe the same three individuals at another time. We might find that this ordering changes (e.g. sometimes A contacts C before contacting B), and that the ordering is largely unpredictable (and could simply be a function of when we started our observations). If this is the case, then our observed dynamics do not provide us with much power when it comes to predicting the outcome of a future epidemic. By contrast, capturing the underlying rates at which these

312 individuals come into contact (i.e. how many times do we observe A contacting B, B
313 contacting C, etc. over a reasonable timeframe given the biology of our study species)
314 will allow more general conclusions to be drawn. This is because a probabilistic model
315 simulating transmission where the probability of transmission is weighted by the
316 strength of the connection among the individuals (i.e. the rate at which they are in
317 contact) will generate the same outcomes as dynamic networks in which the ordering of
318 contacts is random (but of course maintaining who contacts who). As a rule, while the
319 dynamic network in Figure 1A is most important when quantifying an observed
320 epidemic event, the static network in Figure 1B might be more useful for making
321 predictions about the outcome of an epidemic at some unknown time in the future.
322
323 In addition to consistent ordering of contacts, there is a further exception to the rule
324 above. Static networks will not generate the same predictions if the transmission
325 probability scales non-linearly with the strength or intensity of contacts. Take
326 individuals A, B, and C again. At each time step, only one pair of individuals are in
327 contact (the ordering is random), but the contact is strong (say 1 unit, on a range of 0 to
328 1). Thus, the average density of the network (the sum of edge weights divided by the
329 potential sum of edge weights) is 1/3 (there are 3 possible edges that could each have a
330 maximum value of 1). If we make a static representation of the network, then each pair
331 of individuals (i.e. each edge) will be present on average one third of the time, and thus
332 the three edges will all have an edge weight of 1/3 (and thus the static network will
333 have exactly the same density as the dynamic network). In situations where
334 transmission is directly proportional to the strength of the contact, then the average of
335 many simulations run using a static representation of the network will generate
336 identical predictions as the average outcome from many simulated epidemics on a
337 dynamic representation of the network. However, if stronger contacts are
338 disproportionately likely to result in a transmission event than weaker contacts, then
339 the static network could under-estimate the potential of an outbreak. This is because the
340 static network will not capture the nature of occasional but strong contacts. If the
341 relationship between contact rate and transmission probability is weak, then a dynamic
342 network could under-estimate the predicted outbreak size. Having a non-linear
343 transmission probability could explain why recent studies (e.g. Springer, Kappeler et al.
344 2017) have found differences in the outbreaks predicted by dynamic compared to static
345 networks. I illustrate this important point more thoroughly in the case study below.
346 Before I do so, I want to highlight that we could conclude from this case study that it is

always important to model dynamic networks, but to my knowledge the vast majority of models have implemented a linear transmission probability.

Case study: Transmission properties determine differences in simulated spread when using static and dynamic networks

Several studies have compared the impact of disease spreading through static versus dynamic networks (Volz and Meyers 2009, Springer, Kappeler et al. 2017). However, the mechanisms that underlie the differences reported in how diseases (I note this is also relevant to information) spread through different implementations of the same network data have not been well explored. Here I demonstrate how different ways in which the probability of transmission is modelled can generate predictable differences in the spread of a disease.

Because disease transmissions through networks are often modelled as a function of the strength or propensity for individuals to be in contact, a simple prediction is that networks with more contacts will facilitate faster and/or wider spread. However, in a well-designed simulation study, the weighted density of a static network should be exactly equal to the average weighted density of each time slice in a dynamic network, and thus not impact the predictions about how a disease should spread (as highlighted above with the densities of 1/3). I demonstrate this point by constructing a simple simulation model. This model consists of 50 individuals, whose contacts are observed 5 times per day for 10 days. For each of these observations (n=50), I generate a random undirected binary network with a tie probability of 0.2 using the sna package (Butts 2008) in R (R Development Core Team 2017). I then generate a static network from these data, where each edge is defined as the average number of contacts per day averaged over the 10 days, and a dynamic network where each network represents 1 day and the edges represent the number of contacts between each pair of individuals observed on that day. For simplicity I implement only a susceptible-infected model—that is individuals do not recover from their infection. Further, because there are no edges in the static network that were no observed in the dynamic network (and vice-versa), the results are generalisable to more structured networks as generally found in animal populations. This is demonstrated in the supplemental results that contain the same predictions generated using a network based on mixed-species flocks visiting

feeders (as described in Farine, Garroway et al. 2012) contained within the R package *asnipe* (Farine 2013).

First, I simulate the spread of disease on each network using a linear transmission probability. That is, the probability that an individual i acquires a disease from one of its contacts on a given day is proportional to their contact rate: $P(T_{i,j}) = I_j \beta x$, where β is the transmission rate, x is the contact rate per day, and I_j is the infection status of individual j. Note that $P(T_{i,j}) = 0$ when $I_j = 0$. This transmission probability is shown in Figure 2A as a solid black line. The disease spread is simulated over 10 days (for the dynamic network this is a different network each day, whereas the static network uses the same edge values 10 times over). Because the density (i.e. the proportion of realized edges) of the static and dynamic networks are equal, the predicted number of infected individuals at the end of the observation period/simulation is equal, as shown in Figure 2B-C. I note here that the prediction for a single simulation run on both the static and dynamic network representation could be quite different. However, the general patterns over many replicated simulations are identical. For example, in this scenario we predict that in 50% of outbreaks in our population, the proportion of individuals infected will be between 0.35 and 0.65 after 10 days.

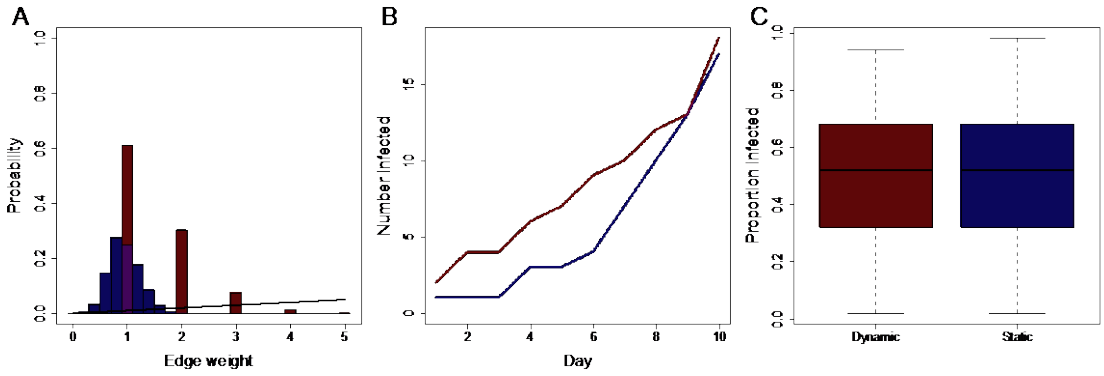

Figure 2. The number of individuals infected is predicted to be equal when the transmission rate is linearly proportional to the contact strength (or edge weight) in the network. Panel A shows the probability distribution of edge weights for a dynamic network (red), a static representation of the same dynamic network (blue), and the transmission probability as a function of edge weight (black line). Panel B shows the cumulative number of infected individuals (population size N=50) over 10 days from one run of the simulation. Panel C shows the proportion of the population infected on day 10 as estimated from 1000 simulations.

However, the relationship between contact rate and transmission probability is not always linear. One scenario is that the relationship between these two is a decelerating function. That is, their relationship increases up until some point whereupon it saturates, thus having stronger contacts does not further increase transmission rate. This type of spread function could represent a scenario where two individuals just have to be in the same area, and any edge (even a weak one) is likely to facilitate transmission. For example, a child just has to be in the same room as another child with chickenpox for 15 minutes to catch it, which contrasts with the intensity of contact required to transmit HIV. Such a function is given in Figure 3A (solid line). If disease transmission occurs at quite weak connections, this means that the edge weight becomes much less important than the presence/absence of an edge (all edges transmit at relatively equal rates). In such a situation, averaging a dynamic network into a static network will increase the number of edges present in each round of the simulation (10 rounds representing 10 days), because edges only have to be present on a single day in order to be present in the static network. As a result, the simulation consistently predicts larger outbreak sizes for the static network (Figure 3B-C). I note here that applying a threshold to reduce the number of weak edges in the static network would be inappropriate, not just because of general issues associated with thresholding (see Farine 2014), but because it would also alter the relative weighted densities of the network.

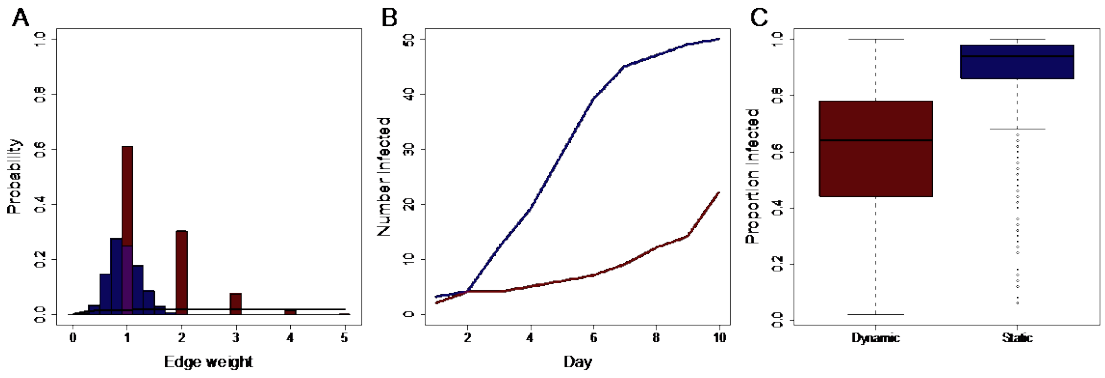

Figure 3. The outbreak of disease is predicted to be larger in static networks when the transmission rate is a decelerating function relative to the contact strength (or edge weight) in the network. Panel A shows the probability distribution of edge weights for a dynamic network (red), a static representation of the same dynamic network (blue), and the transmission probability given an edge weight (black line). Panel B shows the cumulative number of infected individuals (N=50) over 10 days from one run of the

simulation. Panel C shows the proportion of the population infected on day 10 as estimated from 1000 simulations.

A final scenario is the case of an accelerating function, where individuals with very strong contacts have disproportionately high transmission rates. In terms of biology, this could represent close bodily contact between kin (e.g. extended parental care) or pair bonds that does not occur between weaker associates (e.g. the HIV example). Such a relationship is shown in Figure 4A (solid line), although an alternative model could be a step function. Because transmission is much more likely between strongly-connected individuals in the network, simulations typically predict much smaller outbreaks in static networks compared to dynamic networks (Figure 4B-C). This is because of the averaging effect when creating the static network that greatly reduces the spread of the distribution of edge weights (as shown by the histograms in Figure 4A). By contrast, the dynamic network captures the fact that connections can be intermittently strong.

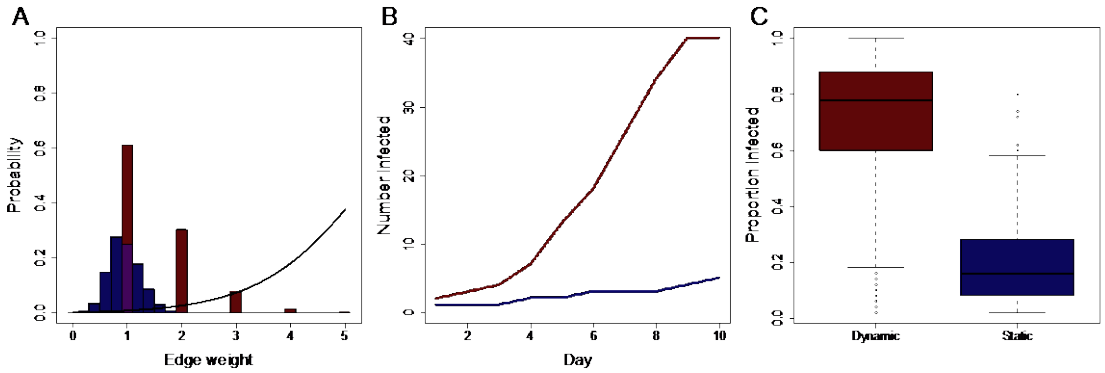

Figure 4. The outbreak of disease is predicted to be larger in dynamic networks when the transmission rate is an accelerating function relative to the contact strength (or edge weight) in the network. Panel A shows the probability distribution of edge weights for a dynamic network (red), a static representation of the same dynamic network (blue), and the transmission probability given an edge weight (black line). Panel B shows the cumulative number of infected individuals (N=50) over 10 days from one run of the simulation. Panel C shows the proportion of the population infected on day 10 as estimated from 1000 simulations.

To conclude this case study, I have shown that the shape of the transmission probability curve (the relationship between edge weight and transmission probability) can have a major impact on the prediction of disease (and equally information) models. This fact could help to explain some of the results found in other studies. Does it justify the use of

dynamic networks for generating predictions about the impact of some future disease? It certainly warrants careful consideration about how the disease spread is modelled. If accurate data are available, both from the network perspective (see below) and the disease transmission function, then perhaps it does warrant using dynamic networks. By contrast, if the purpose of forming the model is to generate predictions for a population or a species, then a more general network (i.e. a static network) with a linear transmission relationship will produce more general predictions.

What temporal resolutions should the dynamic networks have?

A key consideration when transitioning from static to dynamic networks is what time resolution to use. There are two general approaches to implementing a dynamic network: time-ordered and time-aggregated (Blonder, Wey et al. 2012). In a time-ordered network, each edge is present only for the 'exact' period of time over which it occurred. That is, if individual A and B interacted from time 35 to time 45, then the edge is only present for that period. This approach is very precise, but also requires detailed observations (and is typically only useful in situations where all individuals can be observed simultaneously, e.g. Mersch, Crespi et al. 2013, Strandburg-Peshkin, Farine et al. 2015). The alternative method, time-aggregated networks, involves generating replicated networks over relatively short timeframes – for example daily (Boogert, Farine et al. 2014) or weekly (Aplin, Firth et al. 2015) networks. Importantly, the timescale here might be largely dependent on both the data availability and the question. For example, a system where individuals are only observed once per week will not be particularly suitable for weekly, let alone daily, networks (for further discussion on minimum data recommendations for constructing networks, see Whitehead 2008, Franks, Ruxton et al. 2010, Farine and Strandburg-Peshkin 2015, Farine and Whitehead 2015). The temporal patterns of the disease/information transmission process (the ability to observe this or how fast they spread) should also play a part in determining the temporal resolution of the dynamic network (see Cross, Lloyd-Smith et al. 2005). Here, I suggest two considerations to help determine a suitable temporal resolution from the transmission-perspective.

First, many transmission processes can occur over relatively long time-frames. Many diseases have extended incubation periods. For example, chickenpox has an incubation period of 2-3 weeks, and therefore the exact time that the actual transmission took place

could be uncertain (this might generally be less applicable to information, though repeated exposure to information might be important in some systems, such as tool users). In such cases, high-resolution second-by-second temporal resolution of edges will lead to a mismatch between the resolution of the network and the resolution at which we can infer a transmission event. A daily or weekly network might provide a more appropriate model against which to infer transmission characteristics. As with defining the appropriate spatial scale at which transmission is likely to take place (Grear, Luong et al. 2013), the temporal resolution of the networks should be scaled according to the life cycle of the transmission process being measured.

Second, there is often error associated with the measurement of when a transmission event might have taken place. For example, a study might use a weekly sampling regime to test for infections, resulting in a temporal resolution of one week. Again, fitting networks at higher resolution than one week would introduce a mismatch in the temporal scales (or the temporal uncertainty) between the two datasets (the network and the individual states). A similar issue exists for information transmission: the 'information' state of an individual might be determined by when it performs the action, but this provides no data about when it actually acquired the information. Thus, in general, the appropriate resolution to use should be the one that matches the ability to measure individual states and the uncertainty associated with when an individual has transitioned to that state. Take the information example—the uncertainty period might equate the average time between sightings of the individual when it could have performed a new behaviour. This rule applies both to studies of observed epidemics and to those that simulate epidemics, where the aim of the latter is to make testable predictions.

There are many tools available across different disciplines that could be of help for choosing the correct temporal resolution at which to define events in dynamic data. Some of these method involve detecting clusters of events, such as periods in which bursts of transmission events happen (Psorakis, Voelkl et al. 2015). Other methods are aimed at identifying points at which the value of a measure of interest changes, such as change point analysis (Gurarie, Andrews et al. 2009). Methods for identification of timescales in complex systems are particularly promising (Darst, Granell et al. 2016). Finally, a number of statistical modelling approaches have been suggested as useful when evaluating questions of network dynamics (see Silk, Croft et al. 2017). The

application of such tools for dynamic animal social network analysis warrants continued investigation

Can using dynamic networks help my study?

There are a number of trade-offs when considering whether to implement dynamic networks versus static networks. On the one hand, a well-designed study that uses network dynamics at a temporal scale that matches the epidemic/information transmission profile will undoubtedly generate the most accurate conclusions, or allow the most accurate predictions. On the other hand, implementing network dynamics adds complexity to the model, can reduce the generality of the findings, and can overinflate the perceived quality of predictions. These trade-offs are not always clear-cut, and there can be many combinations of factors that will determine which approach is best. In Figure 5, I provide an overview of some of the considerations discussed in this paper. I also discuss a few more nuanced considerations below.

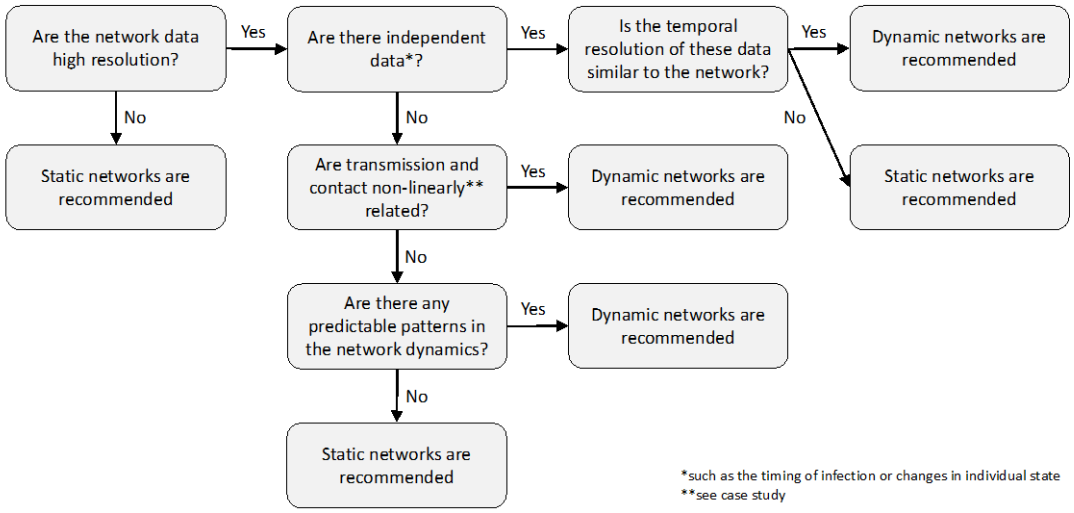

Figure 5. When to choose dynamic versus static social network analysis? This figure summarises the main questions considered in this paper.

One of the motivations for studying networks is that complex outcomes are often not directly reducible to the interactions among individuals. Adding an additional dimension to this problem (i.e. time) further complicates matters. First, it leads to greater difficulties for generating null models (see Croft, Madden et al. 2011, Farine and Whitehead 2015). Second, and most importantly, it becomes more challenging to

565 generate predictions—and thus does not lend itself well to the hypothesis-testing
566 framework biologists are generally used to. Thus, when considering whether to
567 implement a dynamic network approach, one useful step is to ask 'can I make
568 predictions using this framework?'. This will help clarify what network choice is
569 appropriate.
570
571 One case where using dynamics networks is clearly important is situations in which
572 there is some predictability to the dynamics. I have already noted seasonality as one
573 driver of dynamics. Another potentially important factor is whether animals exhibit
574 some consistent differences in their behaviour when they change state. For example,
575 infected mice exhibit sickness behaviours that lead to reduced connections (Lopes,
576 Block et al. 2016). Similarly, female guppies (*Poecilia reticulata*) infected with
577 ectoparasites are less social and also alter the behaviour of the other fish (Croft,
578 Edenbrow et al. 2011). In many cases, patterns arising from individuals changing state
579 will warrant higher-resolved networks (such as updating the network each time an
580 individual becomes sick), and this warrants much greater consideration from modelling
581 studies. However, data from an empirical dynamic network will be most useful if the
582 timing of state changes are also known, hence coming back to the question of matching
583 network data with independent data (see Figure 5).
584
585 Although making predictions about the dynamics of networks can be challenging, using
586 dynamic networks to test other hypotheses that are not dependent on these dynamics
587 can be useful. In particular, using dynamic networks can sometimes help reveal patterns
588 that may be obscured in a more aggregated static network. In well-connected
589 populations (e.g. groups observed in captivity), constructing a static network may make
590 individuals appear much more connected than they actually are. Despite the fact that we
591 have 'weighted' versions of just about every network metric, the impact of the presence
592 versus absence of edges (as a binary form) is still relatively large, even when using these
593 weighted metrics. Thus, compiling all observations into one static network can lose
594 some of the subtle characteristics of the animals' social behaviour. For example, for a
595 study using PIT-tagged zebra finches living in aviaries, Boogert, Farine et al. (2014)
596 created daily networks and fit them as replicated observations in a linear mixed model.
597 The patterns observed with this method were much clearer than those generated with
598 the full 35 days' worth of data compressed into a single network – because the latter
599 was very densely connected with less distinction between strong and weak edges. It is
600 also important to note that, in this case, the continuous collection of PIT tag data

601 throughout the observation period provided sufficient data to create networks at this
602 daily resolution.
603
604
605 **Final thoughts**
606
607 When I initially embarked on writing this manuscript, I was very cynical of dynamic
608 networks—to me they represented another fad where the method drives the question
609 (without appropriate predictions). However, I have largely convinced myself that
610 explicitly accounting for network dynamics will be important in more cases than not, as
611 highlighted by the many pathways in Figure 5 leading to dynamic network analysis.
612 Where the data allow it, most empirical studies will benefit from accounting for changes
613 in patterns of connections over time (even when they do not explicitly test questions
614 about the dynamics). I have also highlighted that models need to take much greater care
615 to consider the effects of the relationship between contact rates and transmission, and
616 to properly explore causes underlying any differences in predictions between
617 simulations based on dynamic and static networks that are generated by the model.
618 More broadly, empirical studies are urgently needed to verify existing modelling studies
619 and inform future ones.
620
621 There are many motivations for studying the links between the environment, social
622 structure, and transmission processes. Ultimately, all studies will benefit from robust
623 analytical approaches. Social network analysis has many advantages, notably by
624 providing a common set of tools and approaches among different studies and model
625 systems. Dynamic networks will play an important role in increasing the robustness of
626 network analyses. By providing some guidance on their application, I hope to have
627 helped researchers with making some of the difficult decisions that arise when
628 designing studies of social systems.
629
630
631 **Acknowledgements**
632
633 I sincerely thank Neeltje Boogert, Mauricio Cantor, and two anonymous reviewers for
634 taking the time to give me constructive feedback on this manuscript. Many of the ideas
635 contained in this manuscript were developed while I received funding by grants from
636 the European Research Council (AdG 250164) and BBSRC (BB/L006081/1) awarded to

Ben C. Sheldon, the NSF (NSF-IOS 1250895) awarded to Margaret C. Crofoot, and the Daimler und Benz Stiftung (32-03/16) awarded to me.

Data Availability

The simulations shown in the case study and supplemental results can be replicated using the supplemental R code.

References

Adelman, J. S., S. C. Moyers, D. R. Farine and D. M. Hawley (2015). "Feeder use predicts both acquisition and transmission of a contagious pathogen in a North American songbird." Proceedings of the Royal Society B **282**: 20151429.

Allen, J., M. Weinrich, W. Hoppitt and L. Rendell (2013). "Network-Based Diffusion Analysis Reveals Cultural Transmission of Lobtail Feeding in Humpback Whales." Science **340**(6131): 485-488.

Ansmann, I. C., G. J. Parra, B. L. Chilvers and J. M. Lanyon (2012). "Dolphins restructure social system after reduction of commercial fisheries." Animal Behaviour **84**(3): 575-581.

Aplin, L. M., D. R. Farine, J. Morand-Ferron, A. Cockburn, A. Thornton and B. C. Sheldon (2015). "Experimentally induced innovations lead to persistent culture via conformity in wild birds." Nature **518**: 538-541.

Aplin, L. M., D. R. Farine, J. Morand-Ferron and B. C. Sheldon (2012). "Social networks predict patch discovery in a wild population of songbirds." Proceedings of the Royal Society B **279**(1745): 4199-4205.

Aplin, L. M., J. A. Firth, D. R. Farine, B. Voelkl, R. A. Crates, A. Culina, C. J. Garroway, C. A. Hinde, L. R. Kidd, I. Psorakis, N. D. Milligan, R. Radersma, B. Verhelst and B. C. Sheldon (2015). "Consistent individual differences in the social phenotypes of wild great tits (Parus major)." Animal Behaviour **108**: 117-127.

Barrett, L., S. P. Henzi and D. Lusseau (2012). "Taking sociality seriously: the structure of multi-dimensional social networks as a source of information for individuals." Philosophical Transactions of the Royal Society B-Biological Sciences **367**(1599): 2108-2118.

Blonder, B., T. W. Wey, A. Dornhaus, R. James and A. Sih (2012). "Temporal dynamics and network analysis." Methods in Ecology and Evolution **3**(6): 958-972.

Boogert, N. J., D. R. Farine and K. A. Spencer (2014). "Developmental stress predicts social network position." Biology Letters **10**(10): 20140561.

Brent, L. J. N., A. MacLarnon, M. L. Platt and S. Semple (2013). "Seasonal changes in the structure of rhesus macaque social networks." Behavioral Ecology and Sociobiology **67**(3): 349-359.

Brockmann, D. and D. Helbing (2013). "The Hidden Geometry of Complex, Network-Driven Contagion Phenomena." Science **342**(6164): 1337-1342.

Bull, C. M., S. S. Godfrey and D. M. Gordon (2012). "Social networks and the spread of Salmonella in a sleepy lizard population." Molecular Ecology **21**(17): 4386-4392.
Butts, C. T. (2008). "Social Network Analysis with sna." Journal of Statistical Software **24**(6): 1-50.
Carter, G. G., D. R. Farine and G. S. Wilkinson (2017). "Social bet-hedging in vampire bats." Biology Letters **13**: 20170112.
Craft, M. E. (2015). "Infectious disease transmission and contact networks in wildlife and livestock." Philosophical Transactions of the Royal Society B-Biological Sciences **370**(1669).
Croft, D. P., M. Edenbrow, S. K. Darden, I. W. Ramnarine, C. van Oosterhout and J. Cable (2011). "Effect of gyrodactylid ectoparasites on host behaviour and social network structure in guppies Poecilia reticulata." Behavioral Ecology and Sociobiology **65**(12): 2219-2227.
Croft, D. P., R. James and J. Krause (2008). Exploring animal social networks. Princeton, Princeton University Press.
Croft, D. P., J. R. Madden, D. W. Franks and R. James (2011). "Hypothesis testing in animal social networks." Trends in Ecology & Evolution **26**(10): 502-507.
Cross, P. C., J. O. Lloyd-Smith, P. L. F. Johnson and W. M. Getz (2005). "Duelling timescales of host movement and disease recovery determine invasion of disease in structured populations." Ecology Letters **8**(6): 587-595.
Darst, R. K., C. Granell, A. Arenas, S. Gomez, J. Saramaki and S. Fortunato (2016). "Detection of timescales in evolving complex systems." Scientific Reports **6**.
Edenbrow, M., S. K. Darden, I. W. Ramnarine, J. P. Evans, R. James and D. P. Croft (2011). "Environmental effects on social interaction networks and male reproductive behaviour in guppies, Poecilia reticulata." Animal Behaviour **81**(3): 551-558.
Farine, D. R. (2013). "Animal Social Network Inference and Permutations for Ecologists in R using asnipe." Methods in Ecology and Evolution **4**: 1187–1194.
Farine, D. R. (2014). "Measuring phenotypic assortment in animal social networks: weighted associations are more robust than binary edges." Animal Behaviour **89**: 141-153.
Farine, D. R. (2017). "A guide to null models for animal social network analysis." Methods in Ecology and Evolution.
Farine, D. R., L. M. Aplin, B. C. Sheldon and W. Hoppitt (2015). "Interspecific social networks promote information transmission in wild songbirds." Proceedings of the Royal Society B **282**: 20142804.
Farine, D. R., C. J. Garroway and B. C. Sheldon (2012). "Social network analysis of mixed-species flocks: exploring the structure and evolution of interspecific social behaviour." Animal Behaviour **84**(5): 1271-1277.
Farine, D. R. and B. C. Sheldon (2015). "Selection for territory acquisition is modulated by social network structure in a wild songbird." Journal of Evolutionary Biology **28**(3): 547-556.
Farine, D. R. and A. Strandburg-Peshkin (2015). "Estimating uncertainty and reliability of social network data using Bayesian inference." Royal Society Open Science **2**: 150367.
Farine, D. R. and H. Whitehead (2015). "Constructing, conducting, and interpreting animal social network analysis." Journal of Animal Ecology **84**(5): 1144-1163.

731 Firth, J. A. and B. C. Sheldon (2016). "Social carry-over effects underpin trans-
732 seasonally linked structure in a wild bird population." Ecology Letters **19**(11):
733 1324-1332.
734 Firth, J. A., B. C. Sheldon and D. R. Farine (2016). "Pathways of information
735 transmission among wild songbirds follow experimentally imposed changes in
736 social foraging structure." Biology Letters **12**: 20160144.
737 Flack, J. C., M. Girvan, F. B. M. de Waal and D. C. Krakauer (2006). "Policing
738 stabilizes construction of social niches in primates." Nature **439**(7075): 426-429.
739 Franks, D. W., G. D. Ruxton and R. James (2010). "Sampling animal association
740 networks with the gambit of the group." Behavioral Ecology and Sociobiology
741 **64**(3): 493-503.
742 Franz, M. and S. C. Alberts (2015). "Social network dynamics: the importance of
743 distinguishing between heterogeneous and homogeneous changes." Behavioral
744 Ecology and Sociobiology **69**(12): 2059-2069.
745 Franz, M., J. Altmann and S. C. Alberts (2015). "Knockouts of high-ranking males
746 have limited impact on baboon social networks." Current Zoology **61**(1): 107-
747 113.
748 Grear, D. A., L. T. Luong and P. J. Hudson (2013). "Network transmission
749 inference: Host behavior and parasite life cycle make social networks meaningful
750 in disease ecology." Ecological Applications **23**(8): 1906-1914.
751 Gurarie, E., R. D. Andrews and K. L. Laidre (2009). "A novel method for
752 identifying behavioural changes in animal movement data." Ecology Letters
753 **12**(5): 395-408.
754 Hamede, R. K., J. Bashford, H. McCallum and M. Jones (2009). "Contact networks
755 in a wild Tasmanian devil (Sarcophilus harrisii) population: using social network
756 analysis to reveal seasonal variability in social behaviour and its implications for
757 transmission of devil facial tumour disease." Ecology Letters **12**(11): 1147-1157.
758 Hobaiter, C., T. Poisot, K. Zuberbuhler, W. Hoppitt and T. Gruber (2014). "Social
759 Network Analysis Shows Direct Evidence for Social Transmission of Tool Use in
760 Wild Chimpanzees." Plos Biology **12**(9).
761 Hobson, E. A., M. L. Avery and T. F. Wright (2013). "An analytical framework for
762 quantifying and testing patterns of temporal dynamics in social
763 networks." Animal Behaviour **85**(1): 83-96.
764 Ilany, A., A. S. Booms and K. E. Holekamp (2015). "Topological effects of network
765 structure on long-term social network dynamics in a wild mammal." Ecology
766 Letters **18**(7): 687-695.
767 Krause, J., R. James, D. W. Franks and D. P. Croft (2015). Animal Social Networks.
768 Oxford, UK, Oxford University Press.
769 Leu, S. T., D. R. Farine, T. W. Wey, A. Sih and M. Bull (2016). "Environment
770 modulates population social structure: experimental evidence from replicated
771 social networks of wild lizards." Animal Behaviour **111**: 23-31.
772 Lopes, P. C., P. Block and B. Konig (2016). "Infection-induced behavioural
773 changes reduce connectivity and the potential for disease spread in wild mice
774 contact networks." Scientific Reports **6**.
775 MacIntosh, A. J. J., A. Jacobs, C. Garcia, K. Shimizu, K. Mouri, M. A. Huffman and A.
776 D. Hernandez (2012). "Monkeys in the Middle: Parasite Transmission through
777 the Social Network of a Wild Primate." Plos One **7**(12).

778 Mersch, D. P., A. Crespi and L. Keller (2013). "Tracking Individuals Shows Spatial
779 Fidelity Is a Key Regulator of Ant Social Organization." Science **340**(6136): 1090-
780 1093.
781 Perkins, S. E., F. Cagnacci, A. Stradiotto, D. Arnoldi and P. J. Hudson (2009).
782 "Comparison of social networks derived from ecological data: implications for
783 inferring infectious disease dynamics." Journal of Animal Ecology **78**(5): 1015-
784 1022.
785 Psorakis, I., B. Voelkl, C. J. Garroway, R. Radersma, L. M. Aplin, R. A. Crates, A.
786 Culina, D. R. Farine, J. A. Firth, C. A. Hinde, L. R. Kidd, N. D. Milligan, S. J. Roberts,
787 B. Verhelst and B. C. Sheldon (2015). "Inferring social structure from temporal
788 data." Behavioral Ecology and Sociobiology **69**(5): 857-866.
789 R Development Core Team (2017). R: A Language and Environment for
790 Statistical Computing. Vienna, Austria, R Foundation for Statistical Computing.
791 Rushmore, J., D. Caillaud, L. Matamba, R. M. Stumpf, S. P. Borgatti and S. Altizer
792 (2013). "Social network analysis of wild chimpanzees provides insights for
793 predicting infectious disease risk." Journal of Animal Ecology **82**(5): 976-986.
794 Silk, M. J., D. P. Croft, R. J. Delahay, D. J. Hodgson, N. Weber, M. Boots and R. A.
795 McDonald (2017). "The application of statistical network models in disease
796 research." Methods in Ecology and Evolution **8**(9): 1026–1041.
797 Springer, A., P. M. Kappeler and C. L. Nunn (2017). "Dynamic vs. static social
798 networks in models of parasite transmission: Predicting Cryptosporidium spread
799 in wild lemurs." Journal of Animal Ecology.
800 Strandburg-Peshkin, A., D. R. Farine, I. D. Couzin and M. C. Crofoot (2015).
801 "Shared decision-making drives collective movement in wild baboons." Science
802 **348**(6241): 1358-1361.
803 VanderWaal, K. L., E. R. Atwill, S. Hooper, K. Buckle and B. McCowan (2013).
804 "Network structure and prevalence of Cryptosporidium in Belding's ground
805 squirrels." Behavioral Ecology and Sociobiology **67**(12): 1951-1959.
806 VanderWaal, K. L., E. R. Atwill, L. A. Isbell and B. McCowan (2013). "Linking social
807 and pathogen transmission networks using microbial genetics in giraffe (Giraffa
808 camelopardalis)." Journal of Animal Ecology **83**(2): 406-414.
809 Volz, E. and L. A. Meyers (2009). "Epidemic thresholds in dynamic contact
810 networks." Journal of the Royal Society Interface **6**(32): 233-241.
811 Weber, N., S. P. Carter, S. R. X. Dall, R. J. Delahay, J. L. McDonald, S. Bearhop and R.
812 A. McDonald (2013). "Badger social networks correlate with tuberculosis
813 infection." Current Biology **23**(20): R915-R916.
814 Whitehead, H. (2008). Analyzing animal societies. Chicago, USA, University of
815 Chicago Press.
816