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Contact and contagion: Probability of transmission given contact varies with demographic state in bighorn sheep

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Abstract

1. Understanding both contact and probability of transmission given contact are key to managing wildlife disease. However, wildlife disease research tends to focus on contact heterogeneity, in part because the probability of transmission given contact is notoriously difficult to measure. Here, we present a first step towards empirically investigating the probability of transmission given contact in free-ranging wildlife.

2. We used measured contact networks to test whether bighorn sheep demographic states vary systematically in infectiousness or susceptibility to *Mycoplasma ovipneumoniae*, an agent responsible for bighorn sheep pneumonia.

3. We built covariates using contact network metrics, demographic information and infection status, and used logistic regression to relate those covariates to lamb survival. The covariate set contained degree, a classic network metric describing node centrality, but also included covariates breaking the network metrics into subsets that differentiated between contacts with yearlings, ewes with lambs, and ewes without lambs, and animals with and without active infections.

4. Yearlings, ewes with lambs, and ewes without lambs showed similar group membership patterns, but direct interactions involving touch occurred at a rate two orders of magnitude higher between lambs and reproductive ewes than between any classes of adults or yearlings, and one order of magnitude higher than direct interactions between multiple lambs.

5. Although yearlings and non-reproductive bighorn ewes regularly carried *M. ovipneumoniae*, our models suggest that a contact with an infected reproductive ewe had approximately five times the odds of producing a lamb mortality event of an identical contact with an infected dry ewe or yearling. Consequently, management actions targeting infected animals might lead to unnecessary removal of young animals that carry pathogens but rarely transmit.

6. This analysis demonstrates a simple logistic regression approach for testing *a priori* hypotheses about variation in the odds of transmission given contact for free-ranging hosts, and may be broadly applicable for investigations in wildlife disease ecology.

Key-words: bighorn sheep, disease ecology, force of infection, *Mycoplasma ovipneumoniae*, pathogen transmission, probability of transmission given contact, social network, wildlife disease

Introduction

Understanding factors that drive variation in host transmission propensity is critical for predicting and managing infectious disease events (Lloyd-Smith *et al.* 2005). For directly transmitted pathogens, transmission can be distilled into two stages. First, a susceptible and an infected host must contact one another; and second, the pathogen must take advantage of that contact to move between hosts and establish in a new individual. Partitioning

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observed heterogeneity into separate stages attributable to contact, and pathogen movement and establishment is a first step towards understanding and managing pathogen transmission. Theoretical and empirical studies describe how contact heterogeneity shapes transmission (e.g. Bansal, Grenfell & Meyers 2007; Craft *et al*. 2011), but a key open question is whether measured networks can also offer insights about the probabilities of transmission given contact.

To date, network-based studies of disease transmission fall into two general groups. The first group simulates epidemics on empirical or simulated contact networks to assess the relative importance of well-connected individuals or groups for pathogen transmission (e.g. Cross *et al*. 2005; Craft *et al*. 2011). Inferences usually focus on population-level epidemic outcomes, such as epidemic size (Cross *et al*. 2004) or duration (Keeling *et al*. 2001). The second group of studies directly measures transmission using physically or genetically marked pathogens (e.g. Zohdy *et al*. 2012; VanderWaal *et al*. 2014), and empirically evaluates the relationship between host behaviour and transmission. Both groups attribute transmission heterogeneity to the contact process, so that epidemic features are treated as functions of contact, with the probability of transmission given contact assumed constant.

Recent studies have begun to link heterogeneity in duration, quality, or order of contact to disease transmission in wildlife (e.g. VanderWaal & Ezenwa 2016). For example, experimental contacts between desert tortoises (*Gopherus agassizii*) underscored the importance of contact quality in transmission of *Mycoplasma agassizii* (Aiello *et al*. 2016); and Quevillon *et al*. (2015) demonstrated how interaction patterns between black carpenter ants (*Camponotus pennsylvanicus*) belonging to different functional grounds (foragers, nest ants, queen, etc.) could theoretically constrain pathogen transmission.

For some human pathogens, knowledge of how probability of transmission given contact shapes epidemic progression is even more explicit. The probability of HIV transmission during a single sexual contact is much higher if the infected individual is within the first 15 months of infection than if the same contact occurred during the subsequent period of viral latency (Wawer *et al*. 2005), indicating a critical role for infection age in determining transmission. While the factors leading to superspreader hosts are often unclear, in some cases, specific demographic attributes relate directly to increased transmission risk. For instance, pregnant women infected with *Plasmodium falciparum* malaria experience higher *Plasmodium* loads in their blood (Bouyou-Akotet *et al*. 2005) and higher vector bite rates (Lindsay *et al*. 2000) than other human host groups, therefore disproportionately contributing to the force of infection.

In these human disease examples, variation in host infectiousness or susceptibility was established by directly measuring pathogen load or immune dynamics within individual hosts, either longitudinally or in widespread cross-sectional sampling pulses. In either case, acquiring sufficient measurements requires extensive animal sampling, which is often not possible in free-ranging wildlife systems; a key next step in epidemiological modelling in wildlife is to account for the contact network with empirical observations, and assess the probability of transmission given contact. Although the probability of transmission given contact is *assumed* constant in models attributing heterogeneity to contact, models could instead condition on *empirically measured* contacts, allowing direct inquiry about probability of transmission and establishment given contact. Here, we outline a general approach for drawing such inferences simply by using social contact information and disease outcomes. We demonstrate the method through a case-study of *Mycoplasma ovipneumoniae* transmission in bighorn sheep.

GENERAL APPROACH

Individual-level network metrics are not independent of one another, in that one animal's connections are not independent of those of its neighbours (e.g. Whitehead 2008). Although network dependencies are usually viewed as inferentially problematic, here, we capitalize on their existence to evaluate how well shared contacts predict common epidemic outcomes among a set of hosts.

Our approach is inspired by adaptive evolutionary models that infer selective pressures over different regions of phylogenetic trees across evolutionary time (Butler & King 2004). These models treat the networks – in their case, phylogenetic trees and here, social contact networks – as known. Node-specific outcomes – in their case, observed phenotypes; here, individual disease outcomes – are assumed to covary in proportion to shared edge attributes. If outcomes for nodes with many edges in common do not covary strongly, this suggests that their shared edges contribute only weakly to the observed outcome. Hypotheses about edge-specific effects can be presented as alternative weightings of the network by categorizing all edges connected to each individual according to hypothesized type, summing edge-weights for each type, and recasting those sums as covariates driving the model's mean so that the corresponding hypotheses can be tested with standard linear modeling techniques (Fig. 1).

Consider an epidemic progressing along a weighted network where nodes represent individuals and edges represent contact intensities. The epidemic produces disease occurrences along the network, with individual outcomes depending on both contact structure (edges and edge-weights) and the probability of transmission given contact. If the probability of transmission given contact does not vary, individual outcomes should covary in proportion to their shared edges (contacts). Alternatively, if some host groups have systematically higher probabilities of transmission given contact, then a more detailed model allowing probabilities to vary for different host groups will better describe the epidemic.

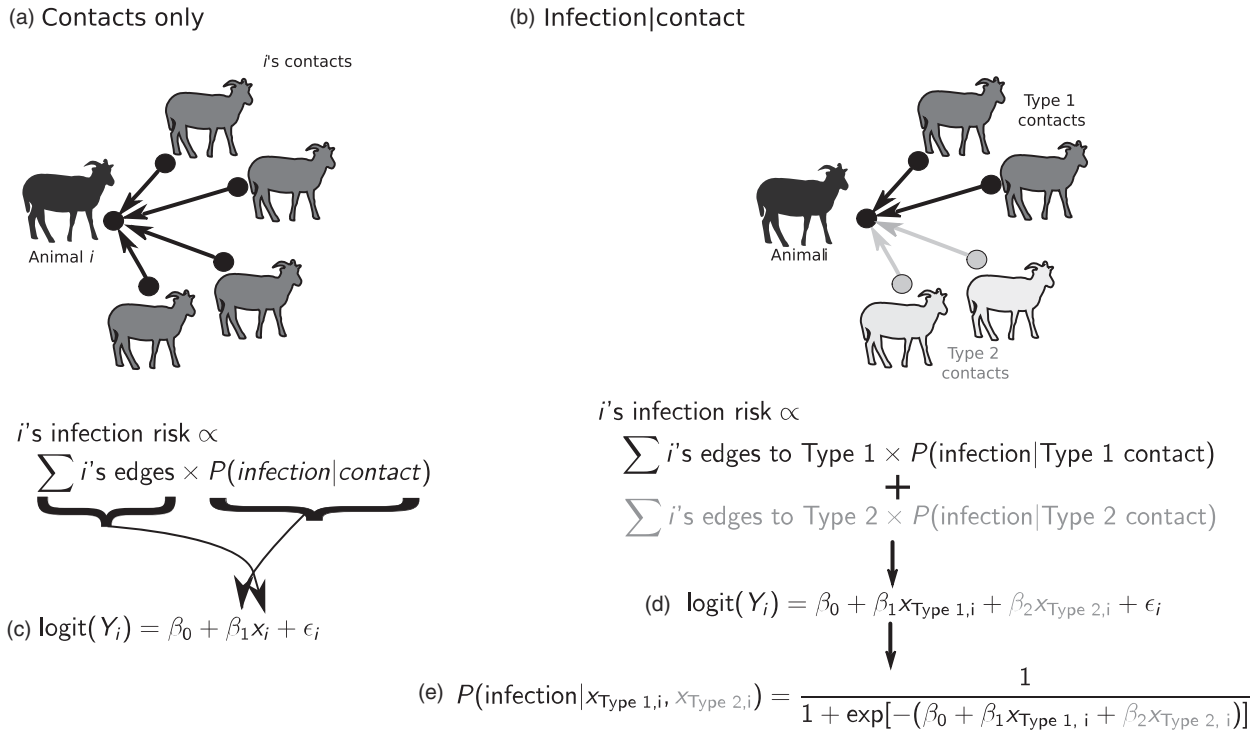

Fig. 1. Conceptual underpinnings. When all animals impose the same infection force on their neighbours, the probability a focal animal (Animal *i*) gets infected is simply a function of the sum of its edge-weights (a). However, when different types of animals impose different probabilities of transmission given contact, the edges are differentially weighted depending on individual identities (b). When individual outcomes and contacts are known, individual outcomes can be regarded as binary response variables, *Y*, and summed edge-weights can be treated as covariates, *X*. This structure allows estimation of an individual's conditional probability of transmission given contact through transformation of the linear predictor from a logistic regression model relating *Y* and *X*, as shown in (c) and (d). Transmission coefficients, β, can be transformed to obtain estimates of the odds ratio of transmission from animals of different types under identical contact conditions. Additionally, this structure allows estimation of conditional probability of transmission given contact with individuals of a particular type (e).

A pure contact model might treat an individual's risk as a function of its network centrality. For individual i whose contacts are listed in the set $I = \{1,2, \ldots, I_n\}$ with edge-weights linking i to $j \in I$ denoted e_{ij}, a pure contact model postulates that risk of infection is a function of a single covariate equal to the sum of i's edge-weights, $\Sigma_{j \in I} e_{ij}$. In this case, all edges of a given weight impose the same transmission risk, regardless of the particular animals involved. However, if load data are available, then the contact-driven edge-weights can be rescaled to also account for neighbour j's current pathogen load, l_j. Under this new load-weighted risk model, i's risk becomes a function of a new covariate, $\Sigma_{j \in I} e_{ij} l_j$. When outcomes and pathogen loads are known, these two hypotheses can be compared with standard model selection methods for logistic regression models whose outcomes uniquely depend on the first covariate vs. the second (Fig. 1). Individual-specific attributes hypothesized to alter susceptibility (e.g. antibody titres or nutritional condition) can also be incorporated as additional covariates, providing a means to directly compare how factors specific to the physiological condition of the recipient host individual and factors specific to that individual's set of infectious contacts shape transmission.

Several studies have already taken this approach without investigating the probability of transmission given contact *per se*. Grear, Luong & Hudson (2013) used a model conditioned on contacts to find optimal temporal lags for capturing transmission dynamics in two classes of macroparasite, and Godfrey *et al.* (2010) used a model accounting for contacts to examine host sex differences in transmission of three tuatara parasites. Nevertheless, studies that condition on measured contacts to better estimate other aspects of transmission remain rare.

APPLICATION: INFERRING TRANSMISSION RISK IN BIGHORN SHEEP

Bighorn sheep experience recurrent spillover and prolonged persistence of pathogens causing population-limiting pneumonia (Cassirer *et al.* 2013; Manlove *et al.* 2016). Disease events start with an all-age die-off typically killing between 15% and 100% of animals in affected herds. Following all-age die-offs, some adults continue chronically carrying *M. ovipneumoniae*, a key agent underlying disease (Besser *et al.* 2013). Chronically infected adults apparently act as reservoirs for transmission to naïve lambs, initiating lamb disease events that severely reduce lamb survival to weaning

in the years-to-decades following die-offs (Cassirer *et al.* 2013; Manlove *et al.* 2016). Currently, it is unclear whether all chronically infected animals impose equivalent transmission risks on lambs, and whether all lambs are equally vulnerable to death from pneumonia.

We apply our general approach to understand *M. ovi* transmission in bighorn sheep during summer lamb pneumonia outbreaks. Our analysis hinges on the assumption that infected animals with low odds of transmission given contact will have relatively little effect on disease dynamics, even if their contact rates with susceptible lambs are high. In contrast, highly infectious animals may influence disease dynamics even if their contact rates with susceptible lambs are low. We arrange the investigation around three research questions. First, are association and interaction patterns consistent across three different demographic classes of bighorn sheep? Second, does the probability of transmission given contact differ between animals in these same demographic states? Finally, how does an individual's infection status relate to its transmission propensity in the bighorn pneumonia system?

Materials and methods

STUDY POPULATIONS AND FIELD DATA COLLECTION

Health and behavioural data were collected from three bighorn sheep populations in southeastern Washington and northeastern Oregon during the summers of 2013–2015 over a total of 6 population-years (Table 1). The Asotin Creek, Black Butte and Mountain View populations were established through translocations conducted between 1977 and 1997 due to efforts from the Washington and Oregon Departments of Fish and Wildlife (WDFD and ODFW). The populations experienced all-age pneumonia epidemics between 1988 and 2012, followed by pneumonia outbreaks of varying severity in lambs. Each population consisted of 42–65 animals during the study period, and the study relies specifically on observational field data from 88 marked ewes and yearlings, monitored for 129 animal-years. Nasal swabs were obtained from most (82 of 129, accounting for 74% of all observations) of the marked animals in the winter preceding summer observations. Samples were analysed at the Washington Disease Diagnostic Laboratory, who measured per cent inhibition by an anti-*M. ovi* antibody using a competitive ELISA, and identified active current infections on a categorical basis (an animal was infected or not) using real-time *M. ovi*-specific PCR (Ziegler *et al.* 2014). We assume PCR status during the preceding winter was a reasonable proxy for an active infection throughout this manuscript. Animals greater than 1 year of age were fitted with colour-coded radiocollars, and individually marked with numbered and coloured ear tags. Animals captured under 1 year of age received only ear tags. Capture and handling were conducted according to protocols approved by the collaborating State agencies.

We compared contact patterns and probability of transmission given contact for individuals in three different demographic states. These states were "dams" (ewes with lambs), "dry ewes" (non-reproductive adult ewes that do not have lambs), "yearlings" (1-year olds). We tracked an additional fourth state, "lambs", though we assumed that lambs could not be chronically infected at the outset of the study, as they are typically born uninfected. Crews located radiocollared individuals approximately every 3 days from 1 May to 15 July and recorded group location, group age composition (number of lambs, ewes and yearlings) and health status of all group members. Number of relocations and sampling intensities for each population-year are reported in Table 1. Observational data collection was conducted under protocols approved by Penn State (IACUC #40292) and Montana State University (IACUC #2014-59).

"Association" data, or information on how frequently each pair of marked animals occurred in the same group, came from 4548 individual relocation events, and were derived using the social contact network methods described below.

Direct contact, or "interaction" data came from 3234 ten-minute group follows on 1131 unique spatiotemporal aggregations ("groups"), during which all direct touching events were timed and classified. Contacts were attributed to specific individuals whenever individual identities were known. Unmarked individuals involved in interactions were recorded by their demographic state (ewes with lambs, dry ewes that were never observed with lambs or lost their lambs prior to 1 June, yearlings, and lambs). Adult rams occurred in less than 10 of the groups included in this analysis, and therefore were excluded. Lambs could not be uniquely identified, so nursing and bedded contacts were assumed to involve dam–lamb pairs unless otherwise noted by the observer; other interactions involving lambs were attributed to "unknown" lambs.

Table 1. Study population sample sizes. In "Disease status", "I" indicates that a disease event occurred in lambs; "P" indicates that live pathogens were detected in some hosts in the population during that population-year; "S" indicates that some adult animals actively displayed symptoms in that population-year; "E" indicates that some collected lamb carcasses showed evidence of pathogen infection in that population-year; and "He" indicates that no signs of clinical disease were observed in lambs

Year	Population	Population size*	Ewes (*n*)	Yearlings (*n*)	Lambs (*n*)	Number recognizable adults and yearlings	Number relocations	Disease status
2013	Asotin Creek	64	32	5	11	12	554	P
2013	Black Butte	13†	13	0	4	4	124	I, P, S, E
2014	Asotin Creek	65	29	9	15	28	1136	He, P, S
2014	Black Butte	13†	13	0	11	13	718	I, P, S, E
2015	Asotin Creek	54	24	13	23	33	1442	He
2015	Mtn View	42	24	14	11	16	574	He, P

*Population estimates, ewe counts and yearling counts are based on counts in March, just prior to the birth pulse.
†The full Black Butte population consisted of 52 animals (30 ewes and 8 yearlings) in 2013, and 36 animals (26 ewes and 2 yearlings) in 2014, but the group of 13 adult ewes studied were completely spatially and socially closed during this study.

NETWORK CONSTRUCTION

Two kinds of networks were used here: an association network whose edges linked pairs of animals that occurred in the same group, and an interaction network whose edges linked pairs of animals that directly contacted one another. In the association network, edges were weighted according to each pair's social affinity index, $S_{i,j}$ (Whitehead 2008, p. 98). The social affinity index is a measure that adjusts for uneven ability to record animal identification (in our case, due to a subset of animals that were only marked with ear tags). When $m_{i,j}$ is the number of days in which individuals i and j were observed associating, $n_{i,j}$ is the number of days in which both i and j were observed but not associating, n_i is number of days with just i observed, and n_j is number of days with just j observed, then the social affinity index for individuals i and j is:

$$S_{i,j} = \frac{m_{i,j}}{\min\{(m_{i,j} + n_{i,j} + n_i), (m_{i,j} + n_{i,j} + n_j)\}}$$

Since lambs associate with their dams continuously until weaning in the fall (Festa-Bianchet 1988), lambs were assumed to be present in the same group, and therefore had the same social affinity indices, as their dams.

Ewe interactions did not approximate lamb interactions, and lambs were not uniquely identifiable, so we could not build the interaction network at the individual level. Instead, the network was aggregated such that nodes reflected demographic states, and edges reflected contact between members of each state. Estimated interaction rates between individuals of two demographic states, State 1 and State 2, were denoted as $\hat{\gamma}_{\text{State1}-\text{State2}}$. When visualizing the network, we separated ewes never observed with lambs, and ewes that had lambs, but lost them prior to 1 June into two distinct states ('Dry' and 'Lost' in Fig. 2b), but we aggregated them for covariate generation and model fitting. Rates were estimated as the total minutes of observed direct contacts between individuals of State 1 and State 2, divided by the total duration of minutes in which all potential pairs of animals of those states were observed. Bootstrap confidence intervals were generated by resampling focal follow events, calculating contact rates for each bootstrapped replicate, and extracting the 2·5th and 97·5th quantiles of the bootstrapped distribution of contact rates.

NETWORK ANALYSIS

Temporally static networks were used to estimate two centrality metrics: degree (the total number of other animals with which a given animal associates), and eigenvector centrality (connectedness metric for a given animal that up-weights connections to well-connected neighbours, and down-weights connections to poorly connected neighbours; Bonacich 1987). Dynamic networks were used to estimate the rate of individual degree accumulation, as well as temporal variation in each individual's short-term degree. In all cases, metrics were compared between three demographic classes: reproductive ewes, dry ewes and yearlings.

Several series of networks, one series for each population-year during each day of the field season, were built to model degree accumulation. Each day's network used all data acquired through that day. Degree (the number of identifiable animals with which each marked animal co-occurred) was calculated for each individual each day, and divided by the total number of marked animals in that population-year, creating a time series of the cumulative proportion of the population with which the individual associated. This meant degree was constrained to increase monotonically through time, from a minimum of associations with 0% of the marked animals in a given population-year to a maximum of associations with 100% of the marked animals in that population-year. This constraint allowed the usage of a logistic function with a common upper bound across all population-years, even though the number of marked animals varied from population to population. Logistic functions were fit using the nls function in R's stats package (R Core Team 2015).

A second series of networks, each extending over sequential 7-day intervals (e.g. the first network included data from days 1–7, the second network included data from days 2–8, etc.) was built to model temporal changes in number of associates. For each network, each individual's degree was calculated, creating a time series of degrees for each animal. The average degree of an individual i in reproductive state g in population-year j in time window t were arranged into a 1× (Timesteps-6) vector Y_{gijt}, which could decline through time during periods when animals were isolated or in smaller-than-average groups. Generalized additive models (GAMs) were fit to the time series of average degrees, and smoothing functions (f_g) were allowed to vary across the g reproductive states. Models included a fixed intercept adjustment

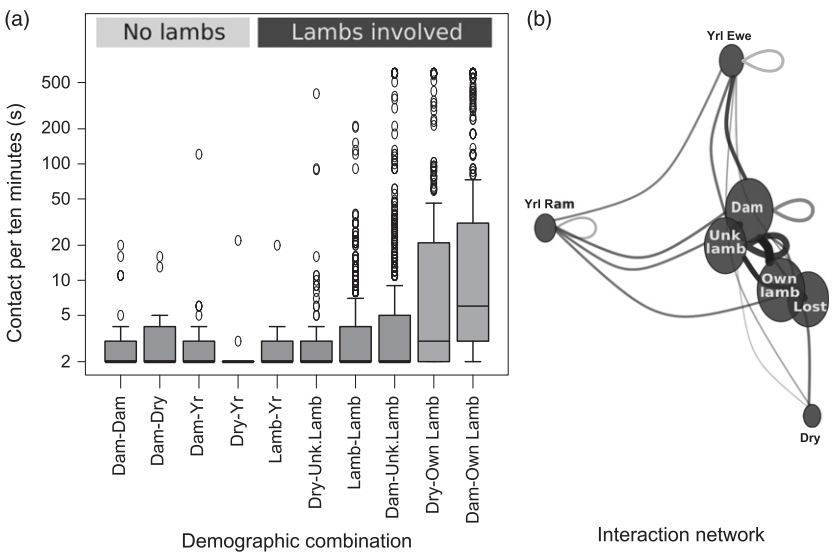

Fig. 2. Most direct contacts occur between lambs and adult ewes. (a) Interaction durations among demographic states where the bin shading scales with the number of contact events. "Dry" in this panel combines dry ewes and ewes that lost their lambs prior to 1 June, and "Yr" refers to yearlings. (b) Interaction network for bighorn nursery groups. Nodes are demographic classes and edge-weights are proportional to contact rates between the groups. Node size reflects number of contacts, and arrangement is based on a gravity model.

(α_j) for each of the j population-years. Formally, the average degree in time window t of individual i from reproductive state g living in population-year j was $y_{gijt} = \alpha_j + f_g(t) + \varepsilon_{ijt}$.

Since a fit to this model had a relatively symmetric and unimodal residual distribution (Fig. S1, Supporting Information), we assumed that $\varepsilon_{gijt} \sim N(0, \sigma^2)$. GAMs were fit using R's mgcv package (Wood 2011).

We evaluated whether association rate and interaction rate were reasonable proxies for one another by assessing the linear relationship between an animal's centrality within the association network (measured here with eigenvector centrality; Bonacich 1987) and its interaction rate, in a model that also contained intercept adjustments for population-year and demographic state. Interaction rate was our measure of interaction centrality, since that metric allowed us to normalize over unbalanced numbers of animal-specific focal follows.

LINKING NETWORK MEASURES TO LAMB SURVIVAL

Lamb survival until 1 October was the binary response variable in all transmission models, under the assumption that lambs that were infected died of disease prior to 1 October, and all other lambs survived. This structure casts lamb mortalities as sentinel events indicating pathogen transmission, an assumption partially justified by previous work in the Hells Canyon system that showed 88% of summer lamb mortality was attributable to disease (Cassirer *et al.* 2013). The analyses relied on lamb survival and covariate measures for 56 lambs born to 43 separate ewes over five population-years. Lambs born to Black Butte ewes in 2013 were excluded, since fewer than 50% of ewes were marked during that population-year, and none had been tested for *M. ovi*. Marked animals never observed with lambs did not contribute response values, but did contribute to the covariate sets.

Static association (together in same group) and interaction (direct contacts) network metrics provided a suite of covariates associated with transmission risk. Covariates were calculated for each lamb based on the set of individuals observed interacting or associating with that lamb (or that lamb's dam). We also included one individual-specific covariate hypothesized to alter lamb susceptibility, maternal per cent antibody inhibition. From the outset, we wanted to compare transmission risk posed by dry ewes and yearlings with transmission risk posed by reproductive ewes. To that end, some models allowed separate risks for contacts with reproductive ewes vs. contacts with dry ewes or yearlings. Covariates, along with details of their construction, are outlined in Table 2. Contacts with yearlings and dry ewes were combined in all transmission analyses, due to their similar interaction rates (Fig. 2). Bivariate plots of all covariate pairs are shown in Fig. S2.

Our objective was to model lamb survival, but limited knowledge of the incubation and infection periods, as well as the assumption that most transmission likely stems from newly infected lambs once the epidemic is underway, prevented us from using traditional Cox-like hazard models. Instead, we fit a suite of logistic regressions that captured all combinations of the hypotheses in Table 2. The association centrality, interaction intensity, and infection-weighted association and interaction hypotheses were all tested using variants on the same theme: that the risk imposed on a given individual by a certain peer group can be measured by summing over total contacts or interactions with members of that peer group (Table 2). Models with more conventional approaches to infection risk, for example, a model that treated an individual's risk as a function of the sum of that individual's edge-weights in the interaction or association network, were also included, as was a model that treated risk as a function of estimated population size (PopEst).

All models included a random effect for population-year. Models conditioned on contact were compared to models that overlooked contact, and instead used the number of reproductive ewes, dry ewes and yearlings in each population as covariates. Dam per cent antibody inhibition was also accounted for, so that results were directly comparable. AIC was used for model comparison throughout, which a 2-point difference in AIC regarded as substantial improvement in model fit (Burnham & Anderson 2003).

Results from the transmission models prompted us to conduct three follow-up analyses. The first was a detailed exploration of antibody patterns in adults as a function of age and population-year. The second was a Fisher's exact test comparing infection prevalence (as determined by *M. ovi* PCR status in the preceding winter) in yearlings ($n = 22$) to infection prevalence in adult females ($n = 60$). The third was a logistic regression of lamb survival status (0 or 1 for each lamb) as a function of two indicator variables: one indicating whether a lamb had any associations with infected reproductive ewes, and one indicating whether a lamb had any associations with infected yearlings or dry ewes.

Results

ARE ASSOCIATION AND INTERACTION PATTERNS CONSISTENT ACROSS DEMOGRAPHIC STATES?

Association networks based on shared group membership were generally well-connected, with all animals exhibiting similar connection numbers and strengths (Fig. S3). All animals accumulated associates at a relatively constant rate (Fig. 3a and Table S1; although demographic states showed statistically significant differences in patterns, qualitatively their degree accumulation trajectories were similar). While groups were large and stable prior to the mid-May birth pulse, all demographic classes showed diminished average degree during the lambing period. Average degree increased again about a week after the peak of the birth pulse, and remained high throughout summer (Fig. 3b and Table S2; demographic state differences are statistically significant, but qualitatively similar), consistent with formation of nursery groups.

Field crews recorded data on 1131 groups, and observed an average of 27·4 identifiable animals and 6·6 groups per 48-h period. Interaction patterns varied dramatically between demographic classes, with all the most common forms of interactions involving lambs (Fig. 2 and Table S3). The most common and longest-duration interactions were between lambs and their dams, and typically consisted of nursing or bedding together. Yearlings and adults of all demographic classes almost never directly contacted other post-juvenile animals. Interactions and associations were not significantly related to one another at the individual level for identifiable animals ($\beta < 0{\cdot}01$; standard error $<0{\cdot}01$; conditional R^2 from the regression of total

Table 2. Hypotheses, covariate definitions, and expected relationships. I_x denotes all of lamb i's edges connected to individuals of group X. $S_{i,j}$ is the social affinity index for individuals i and j, $\hat{\gamma}_{\text{State1}-\text{State2}}$ is the estimated of interaction minutes per day between individuals of demographic state 1 and demographic state 2 (reported in Table S3). Association and interaction metrics that are not group-specific sum over all associates, regardless of demographic state

Lamb survival predictors	Covariate	Calculation	Expectation
Associations with specific groups of animals, regardless of infection status	C_{ReproEwe}	$\log\left(1+\sum_{j\in I_{\text{ReproEwe}}} S_{i,j}\right)$	Lambs with many reproductive ewe associates have high P(death)
	C_{DryYrl}	$\log\left(1+\sum_{j\in I_{\text{DryYrl}}} S_{i,j}\right)$	Lambs with many dry ewe or yearling associates have high P(death)
Interactions with specific groups of animals, regardless of infection status	D_{ReproEwe}	$\log\left(1+\sum_{j\in I_{\text{ReproEwe}}} S_{i,j}\times\hat{\gamma}_{\text{ReproEwe}-\text{Lamb}}\right)$	Lambs with high interaction rates with reproductive ewes have high P(death)
	D_{DryYrl}	$\log\left(1+\sum_{j\in I_{\text{DryYrl}}} S_{i,j}\times\hat{\gamma}_{\text{DryYrl}-\text{Lamb}}\right)$	Lambs with high interaction rates with yearlings and dry ewes have high P(death)
Associations with specific groups of infected animals	PA_{ReproEwe}	$\log\left(1+\sum_{j\in I_{\text{PCR}+\text{ReproEwe}}} S_{i,j}\right)$	Many infected associate reproductive ewes increases P(death)
	PA_{DryYrl}	$\log\left(1+\sum_{j\in I_{\text{PCR}+\text{DryYrl}}} S_{i,j}\right)$	Many infected associate dry ewes and yearlings increases P(death)
Interactions with specific groups of infected animals	PI_{ReproEwe}	$\log\left(1+\sum_{j\in I_{\text{PCR}+\text{ReproEwe}}} S_{i,j}\times\hat{\gamma}_{\text{ReproEwe}-\text{Lamb}}\right)$	Many interactions with infected reproductive ewes increase P(death)
	PI_{DryYrl}	$\log\left(1+\sum_{j\in I_{\text{PCR}+\text{DryYrl}}} S_{i,j}\times\hat{\gamma}_{\text{DryYrl}-\text{Lamb}}\right)$	Many interactions with infected dry ewes or yearlings increases P(death)
Ewe's antibodies	M	Ewe's per cent antibody inhibition (measured in preceding winter)	Lambs born to seropositive ewes have lower P(death)

interactions on association eigencentrality = 23%; Fig. S4), suggesting individuals with high association centralities (those regularly observed with many other individuals within their populations) were not necessarily the same individuals who engaged in the most, or the longest, direct contacts. This is not overly surprising, as individuals with weak social ties may be more likely to switch groups, but less likely to actually touch other animals within those groups.

DOES THE PROBABILITY OF TRANSMISSION GIVEN CONTACT DIFFER BETWEEN DEMOGRAPHIC STATES?

Transmission models were fit to binary survival data from the 56 lambs documented during our study period for which dam *M. ovi* PCR and antibody inhibition data and contact data were available. Of these lambs, 17 died and 39 survived, forming the basis for our logistic regression models of transmission.

The best-performing transmission model (AIC weight = 0·59) included interaction-weighted, demographic class-specific contacts with infected animals, as well as dam's per cent antibody inhibition (Fig. 4c, AIC weights relative to other models shown in Table 3, and model coefficient estimates shown in Table S5). A second model that included group-specific associations with infected animals and dam's per cent antibody inhibition was competitive with the top model (AIC weight = 0·39; Table 3). Both high-performing models supported higher transmission coefficients for infected dams than for infected dry ewes and yearlings, suggesting that the probability of transmission given contact varied with demographic class. Models that overlooked neighbour infection status, or accounted for connectedness (either through associations or interactions) without adjusting for differences between demographic classes, and models that did not include dam's antibody values received little-to-no support (Table 3). There were not significant differences

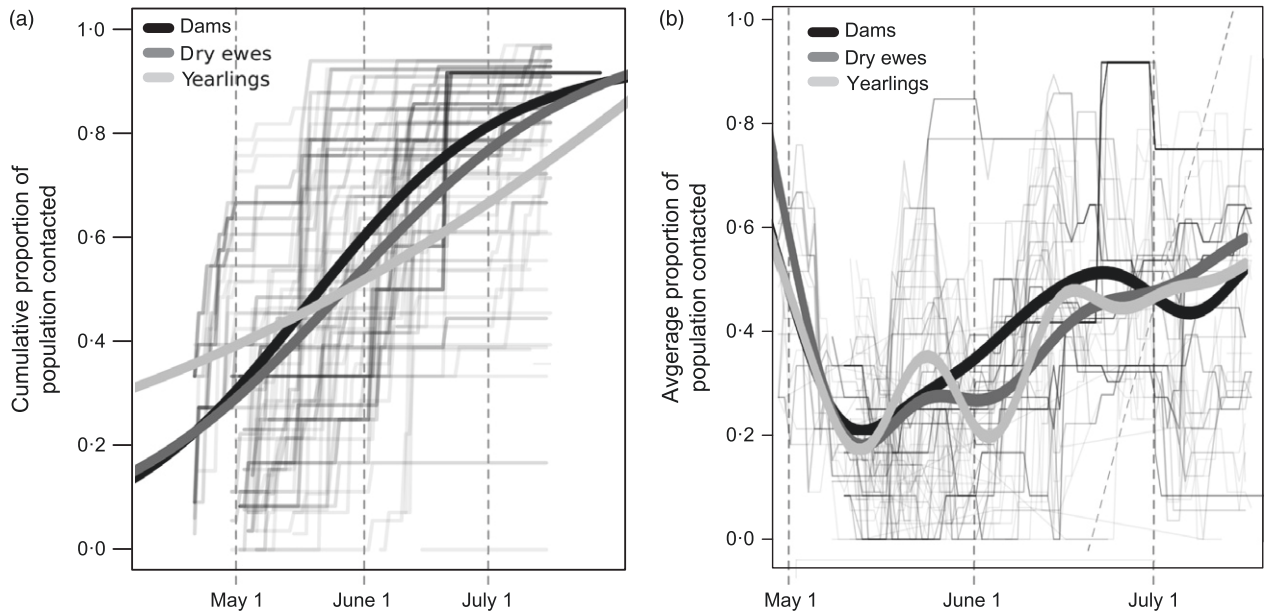

Fig. 3. Animals acquire new associates at qualitatively similar rates, regardless of their demographic status. (a) Cumulative proportion of the population each individual contacted (i.e. node degree/number of marked animals) through time for all animals in our study (thin grey lines), and fits from logistic functions for reproductive ewes ("dams"), non-reproductive "dry" ewes, and ewes who lost their lambs prior to 1 June and yearlings. (b) Average proportion of the population contacted during 7-day moving windows during the study.

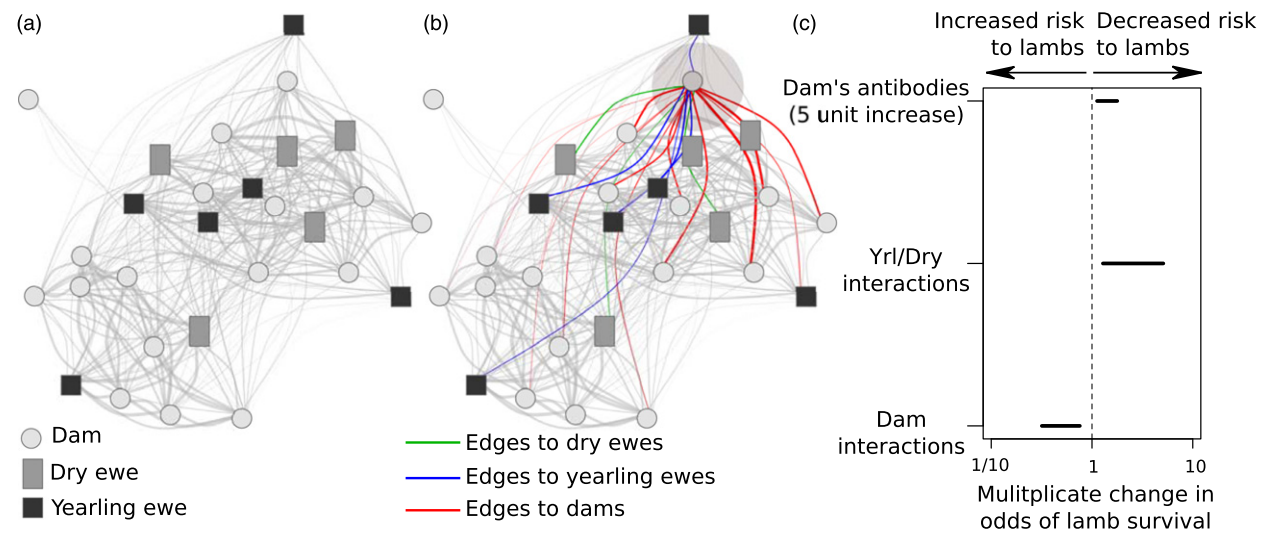

Fig. 4. Network-derived metrics suggest interactions with infected dams decrease odds of lamb survival. (a) Social contact network from the Asotin Creek population in 2014. (b) The same network, with edges categorized into demographic classes hypothesized to exert different probabilities of transmission given contact on their neighbours for a single animal, whose node is circled in grey. (c) Transmission coefficient estimates from the top lamb survival model. "Dam" interactions refer to direct contacts with reproductive ewes. [Colour figure can be viewed at wileyonlinelibrary.com]

in per cent antibody inhibition among adult females across all population-years included in this analysis ($F = 2{\cdot}07$, $P = 0{\cdot}10$).

In the top model, increased contact with PCR-positive yearlings and dry ewes was associated with a significant increase in lamb survival ($\beta = 0{\cdot}94$; SE(β) = 0·35; $P < 0{\cdot}01$; Table S5), whereas increased contact with PCR-positive dams was associated with decreased survival ($\beta = -0{\cdot}71$; SE(β) = 0·22; $P = 0{\cdot}01$; Table S5). The exponentiated difference between these coefficients describes the multiplicative change in lamb survival odds when a contact with an infected dry ewe or yearling is switched for an equivalent contact with an infected dam. In this case, that quantity is $\exp(-0.71-0.94) = 0{\cdot}19$, suggesting contacts with infected dry ewes or yearlings subject lambs to one-fifth the survival risk of comparable contacts with infected dams. Higher

Table 3. Hypotheses, models and AIC diagnostics

Lamb survival predictors	Model	AIC	Delta-AIC	AIC weight
Interactions with specific groups of infected animals Dam's antibodies	$\beta_0 + \beta_1 \mathrm{PI}_{\mathrm{ReproEwe}} + \beta_2 \mathrm{PI}_{\mathrm{DryYrl}} + \beta_3 M$	48·41	0	0·59
Associations with specific groups of infected animals Dam's antibodies	$\beta_0 + \beta_1 \mathrm{PA}_{\mathrm{ReproEwe}} + \beta_2 \mathrm{PA}_{\mathrm{DryYrl}} + \beta_3 M$	49·25	0·84	0·39
Total population size	$\beta_0 + \beta_1 \mathrm{PopEst}$	55·46	7·06	0·02
Interactions with specific groups of animals regardless of infection status Dam's antibodies	$\beta_0 + \beta_1 \mathrm{D}_{\mathrm{ReproEwe}} + \beta_2 \mathrm{D}_{\mathrm{DryYrl}} + \beta_3 M$	57·13	8·72	0·01
Associations with specific groups of infected animals	$\beta_0 + \beta_1 \mathrm{PA}_{\mathrm{ReproEwe}} + \beta_2 \mathrm{PA}_{\mathrm{DryYrl}}$	60·37	11·96	No support
Interactions with specific groups of infected animals	$\beta_0 + \beta_1 \mathrm{PI}_{\mathrm{ReproEwe}} + \beta_2 \mathrm{PI}_{\mathrm{DryYrl}}$	60·43	12·02	No support
Associations with specific groups of animals regardless of infection status Dam's antibodies	$\beta_0 + \beta_1 \mathrm{C}_{\mathrm{ReproEwe}} + \beta_2 \mathrm{C}_{\mathrm{DryYrl}} + \beta_3 M$	63·51	15·1	No support
Interactions with specific groups of animals regardless of infection status	$\beta_0 + \beta_1 \mathrm{D}_{\mathrm{ReproEwe}} + \beta_2 \mathrm{D}_{\mathrm{DryYrl}}$	64·47	16·06	No support
Dam's antibodies	$\beta_0 + \beta_1 M$	67·24	18·83	No support
Constant risk	β_0	70·75	22·34	No support
Total associations with infected animals, regardless of demographic state	$\beta_0 + \beta_1 \sum(\mathrm{PA}_{\mathrm{ReproEwe}} + \mathrm{PA}_{\mathrm{DryYrl}})$	72·25	23·84	No support
Associations with specific groups of animals regardless of infection status	$\beta_0 + \beta_1 \mathrm{C}_{\mathrm{ReproEwe}} + \beta_2 \mathrm{C}_{\mathrm{DryYrl}}$	71·81	23·4	No support
Total associations with all animals, regardless of infection status or demographic state	$\beta_0 + \beta_1 \sum(\mathrm{C}_{\mathrm{ReproEwe}} + \mathrm{C}_{\mathrm{DryYrl}})$	77·11	28·69	No support
Total interactions with all animals, regardless of infection status or demographic state	$\beta_0 + \beta_1 \sum(\mathrm{D}_{\mathrm{ReproEwe}} + \mathrm{D}_{\mathrm{DryYrl}})$	77·91	29·49	No support

antibody values in a lamb's dam was associated with a small but significant increase in the odds the lamb survived ($\beta = 0{\cdot}07$; SE(β) = 0·02; $P < 0{\cdot}01$; Table S5).

Ewes that lost lambs had lower median per cent antibody inhibition than ewes whose lambs survived (Wilcoxon rank-sum $W = 23$; $P = 0{\cdot}08$; median antibody inhibition for ewes whose lambs survived was 68%; median antibody inhibition among ewes whose lambs died was 54%). This effect is somewhat confounded with ewe age, since younger ewes were born after the major disease transmission event at Asotin Creek in 2011, and also frequently lost their lambs (Fig. 5). The apparent benefit of high dam antibody

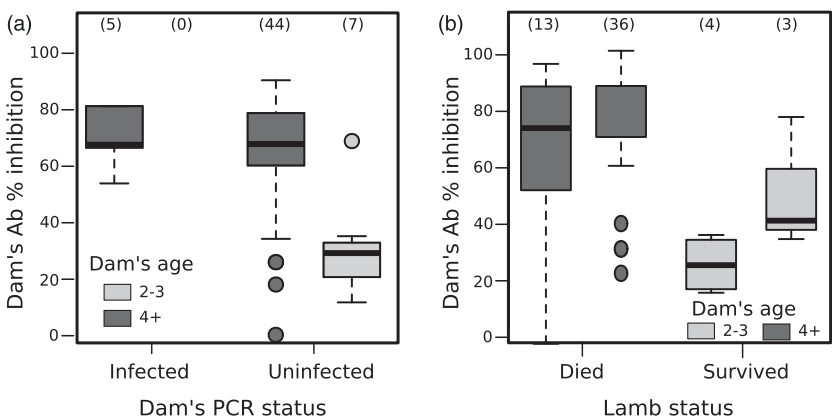

Fig. 5. The apparent effect of dam's antibody per cent inhibition on lamb survival is confounded with dam age. Sample sizes are shown in parentheses above each box. (a) Percent antibody (Ab) inhibition by dam's infection status for ewes aged 4 + (dark grey) and 2–3 (light grey). The younger dams studied here had low antibody values, and none had active infections. (b) Percent antibody (Ab) inhibition by lamb survival status and ewe age. Bars extend across the range of observations, unless points were identified as outliers, in which case they are plotted individually. Per cent antibody inhibition had no detectable relationship with lamb survival for lambs born to older dams (Wilcoxon rank-sum P-value = 0·38).

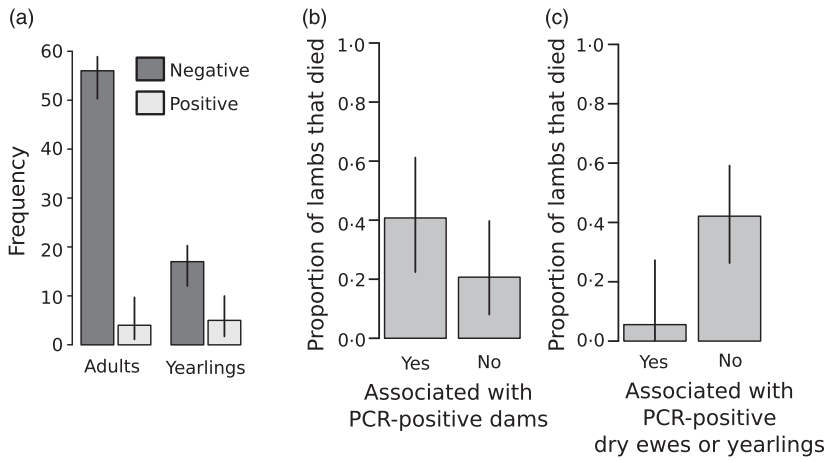

Fig. 6. The relation between lamb survival and association with PCR-positive animals varied between demographic classes. (a) Infection status in winters preceding our behavioural and lamb survival studies for adults and yearlings. (b) Proportion of lambs that died by association status with infected reproductive ewes ("dams"). (c) Proportion of lambs that died by association status with infected yearlings or dry ewes. Bars represent 95% binomial confidence intervals in all panels.

inhibition values might actually be due to independent relations between both antibody values and lamb survival, and the dam's age. Since the six lambs born to 2-year-old ewes generally had low survival, we refit the entire model suite using a dataset omitting those animals. Model rankings were nearly identical (Table S4), as were coefficient estimates for the best-performing model.

HOW DOES INFECTION STATUS RELATE TO TRANSMISSION RISK?

Yearlings had marginally higher *M. ovi* prevalence than adults (22% of yearlings were PCR-positive for *M. ovi* vs. 7% of adults; Fisher's exact test $P = 0{\cdot}05$; Fig. 6a). However, lambs that never associated with infected reproductive ewes had significantly higher survival rates than lambs that associated with at least one infected reproductive ewe ($\beta = 1{\cdot}6$ SE(β) = 0·7; $P = 0{\cdot}02$; Fig. 6b). In contrast, lambs that never associated with infected dry ewes or yearlings had significantly worse survival than lambs that associated with at least one infected dry ewe or yearling ($\beta = -2{\cdot}99$; SE(β) = 1·13; $P = 0{\cdot}01$; Fig. 6c).

Discussion

We proposed a simple logistic regression approach to infer whether classes of individuals differ in odds of transmission given contact by relating contact structure and within-host attributes to measured disease outcomes. When we applied the approach to *M. ovipneumoniae* transmission in free-ranging bighorn sheep, we found that association patterns (presence in the same group) did not correspond to interaction (direct contact) patterns. Networks incorporating both interactions and associations, along with dam per cent antibody inhibition, provided the best explanation for observed epidemic patterns, and incorporating individual-specific infection status improved model fits further still. The competitive performance of a model that overlooked interactions and focused exclusively on associations suggests that associations (animal presence in the same group) may perhaps be more important than interactions (direct touching) for transmission in this system. Our results indicate that both the particular composition of an individual's set of infectious contacts (namely, the distribution of those contacts across demographic classes), and the particular vulnerabilities of the focal individual (measured in terms of dam's per cent antibody inhibition to an important causal agent), shape pathogen transmission in the bighorn sheep pneumonia system.

Our approach extends existing epidemiological network methods, which traditionally focused on network topology as the primary determinant of disease transmission patterns (reviewed in White, Forester & Craft 2015). Contact heterogeneity can certainly produce "superspreader" individuals who disproportionately shape population-level epidemiology, but superspreaders (and supershedders) can also arise because of disproportionately high probability of transmission given contact. Evidence of supershedder and superspreader groups whose roles cannot be explained by behaviour alone has been reported in West Nile Virus (Kilpatrick *et al.* 2006), chytridiomycosis (Kilpatrick, Briggs & Daszak 2010) and gastro-intestinal parasites of small mammals (Streicker, Fenton & Pedersen 2013). However, empirically comparing the probabilities of transmission given contact in free-ranging systems remains a major impediment to incorporating within-host heterogeneities into a broader understanding of population-level disease transmission. Here, we took a first step towards bridging that gap by using measured networks and known disease outcomes to infer probability of transmission given contact, without measuring that probability directly.

In bighorn sheep, degree accumulation and association strengths were similar for reproductive ewes, dry ewes, and yearlings during lamb rearing (Fig. 3), but direct contact rates with lambs varied dramatically between demographic classes (Fig. 2). Qualitatively, we observed that animals without lambs were often spatially peripheral to nursery groups, and rarely interacted directly with other group members. Although a simple evaluation of prevalence by reproductive status suggested yearlings might act

as important local pathogen reservoirs due to their relatively high rates of infection (Fig. 6a), our analyses suggested that in fact yearlings and dry ewes rarely transmit pathogens to lambs. We estimated that a contact with an infected dam has approximately five times the odds of producing a lamb mortality event than an identical contact with an infected dry ewe or yearling.

Average number of associates declined during the birth pulse, and then rapidly increased (Fig. 3b), corresponding to "crèching", the formation of ewe–lamb nursery groups which structure disease mortalities in this system (Manlove *et al.* 2014). From a disease transmission standpoint, crèching elevates an animal's number of potential infectious contacts. Whether total number of associates is the best measure for rate of acquiring new potentially infectious contacts likely depends on the intensity of contact necessary for transmission, as well as the interaction rates, pathogen loads and symptoms of the particular animals involved. While each of these mechanisms clearly contributes to disease dynamics, our data are insufficient to differentiate among them at this time.

A ewe's per cent antibody inhibition value was associated with small but significant increases in the odds that her lamb survived, but we hesitate to overemphasize this relationship for several reasons. First, a model consisting of only dam's antibody value was not competitive with models that incorporated contact (Table 3); the effect was only evident in models accounting for contact structure. This does not necessarily preclude a dam antibody effect, but it does suggest that antibody values cannot overcome – and may confounded with – contact structure. Second, antibody inhibition measures were based on field samples collected 2–6 months prior to lamb birth, and are therefore likely subject to both process error from antibody waning in the dam, and measurement error associated with the sampling protocol and subsequent cELISAs. Finally, we saw similar antibody inhibition levels across all older dams, regardless of their current infection status (Fig. 5a), suggesting that immune response is not strongly associated with pathogen clearance (in which case we would expect to see higher per cent inhibition among uninfected animals). Despite these reservations, we nevertheless include the antibody result here, as we believe it merits future exploration.

Our analysis relies on a few additional assumptions that could shape the results. First, we used lamb survival as a proxy for transmission and infection, assuming all infected lambs died and all uninfected lambs survived. In reality, we know that some infected lambs survive, and that some lambs die of other causes. However, previous studies found that most (88%) of pre-weaning lamb mortality in these populations is attributable to disease, and that median summer lamb survival in the absence of disease is in excess of 80% (Cassirer *et al.* 2013). Furthermore, although misclassification error undoubtedly adds noise to this analysis, we see no reason to suspect that noise to systematically bias our findings (but see Vander Wal *et al.* 2015, who found an association between individual fitness and social network centrality in a different bighorn sheep system). Additionally, some of our data are drawn from a year with a novel M. ovi strain introduction event (described in Cassirer *et al.* 2017), which may have resulted in particularly severe disease and marginally altered behavioral signals.

Second, this analysis is likely subject to measurement error and confounding in the covariates, since our predictor values derive from field observations of association. While this form of error is present yet unaccounted for in many ecological studies, recent work suggests it may factor disproportionately into inappropriate detection of effects (Westfall & Yarkoni 2016). In this system, we have no clear means of validating the reliability of our association indices (that is, there is no "gold standard" to which we can compare our field-estimated association indices). The role of measurement error, both in social network analysis and in ecological studies more generally, requires further consideration in future work.

An additional factor that might confound our results is the assumption that direct contact patterns during focal follows reflected direct contact patterns at other times of day. Importantly, we have no observations during night. We assume that bighorn sheep decrease their activity levels, and spend most of their time bedded in groups. Since lambs probably bed preferentially with their dams and may nurse at night, the data presented here likely underestimate direct dam–lamb contact rates. This might explain the much higher force of infection from infected dams than from infected yearlings and non-reproductive ewes. However, we would expect that force of infection to apply disproportionately to lambs with infected dams (as opposed to lambs that were associated with infected ewes, but that were born to uninfected ewes), and that effect did not emerge in our analyses.

Finally, we assumed that contact rates were homogeneous within demographic states. This assumption was made partially out of necessity, since we could not uniquely identify lambs. For the demographic states in which animals were uniquely identifiable, rare contacts paired with unbalanced observation times complicated formal assessments of within-group heterogeneities in interaction patterns. Therefore, while within-group heterogeneities in interaction rates likely exist, we overlook them here.

Despite these caveats, our results strongly suggest that pathogen transmission risk is not constant across all infected hosts, even after accounting for differences in association patterns and interaction rates. We saw major differences in transmission probabilities between demographic states *after* accounting for differences in contact intensity, with yearlings and dry ewes apparently imposing a much lower force of infection on lambs than the force imposed by infected dams. Our results suggest that perhaps test-removal strategies aimed at reducing *M. ovi* prevalence should primarily target infected adult ewes. Incorporating age-structured removals as one potential

management action in a broad-scale experimental adaptive management framework could clarify this effect.

This analysis is an early effort to leverage empirically measured contacts in order to test hypotheses about probability of transmission given contact. We anticipate that this line of inquiry will increase in importance as the set of measured animal contact networks continues to grow, and research efforts shift to the remaining variation in transmission not attributable to contacts alone. Key next steps include evaluating how measurement error might drive these results, and incorporating additional aspects of individual heterogeneity. Inferring probability of transmission given contact using social network analyses may be more plausible for many wildlife systems than conducting the analogous infection and transmission trials in captivity. As such, network-based inferences like the ones presented here could fill an inferential gap with important implications for wildlife disease management.

Authors' contributions

All authors designed the project, and K.R.M. and E.F.C. collected data. K.R.M. developed the inferential framework with input from P.C.C., and drafted the manuscript. All authors contributed to manuscript revisions.

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Data accessibility

Data for all regression models are available from the Dryad Digital Repository https://doi.org/10.5061/dryad.q7v72 (Manlove *et al*. 2017).

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Supporting Information

Details of electronic Supporting Information are provided below.

Fig. S1. Residual distribution from the generalized additive model (GAM) of node strength through time.

Fig. S2. Pairwise correlations between predictors in the lamb survival model.

Fig. S3. Node strength and degree for each population-year.

Fig. S4. Relationship between interaction rate and eigencentrality.

Table S1. Fits to logistic growth curve in degree accumulation for dams, dry ewes and yearlings.

Table S2. Generalized additive model fits for moving average of degree in each demographic groups.

Table S3. Contact rate estimates for dyads of various demographic groups.

Table S4. Models and information criteria for models fit without lambs born to 2-year-old dams.

Table S5. Coefficient estimates from the top model in a fit that excludes 2-year-old ewes.