MOLECULAR ECOLOGY

Molecular Ecology (2012) **21**, 1271–1282 doi: 10.1111/j.1365-294X.2011.05431.x

Influence of landscape and social interactions on transmission of disease in a social cervid

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Abstract

The mechanisms of pathogen transmission are often social behaviours. These occur at local scales and are affected by landscape-scale population structure. Host populations frequently exist in patchy and isolated environments that create a continuum of genetic and social familiarity. Such variability has an important multispatial effect on pathogen spread. We assessed elk dispersal (i.e. likelihood of interdeme pathogen transmission) through spatially explicit genetic analyses. At a landscape scale, the elk population was composed of one cluster within a southeast-to-northwest cline spanning three spatially discrete subpopulations of elk across two protected areas in Manitoba (Canada). Genetic data are consistent with spatial variability in apparent prevalence of bovine tuberculosis (TB) in elk. Given the existing population structure, between-subpopulation spread of disease because of elk dispersal is unlikely. Furthermore, to better understand the risk of spread and distribution of the TB, we used a combination of close-contact logging biotelemetry and genetic data, which highlights how social intercourse may affect pathogen transmission. Our results indicate that close-contact interaction rate and duration did not covary with genetic relatedness. Thus, direct elk-to-elk transmission of disease is unlikely to be constrained to related individuals. That social intercourse in elk is not limited to familial groups provides some evidence pathogen transmission may be density-dependent. We show that the combination of landscape-scale genetics, relatedness and local-scale social behaviours is a promising approach to understand and predict landscape-level pathogen transmission within our system and within all social ungulate systems affected by transmissible diseases.

Keywords: bovine tuberculosis, *Cervus canadensis manitobensis*, dyadic interactions, elk, landscape genetics, pathogen transmission, population structure, relatedness, sociality

Received 21 April 2011; revision received 15 November 2011; accepted 15 November 2011

Introduction

In disease ecology, understanding how the social systems of individual hosts could influence infectious contacts and pathogen transmission is critical (Altizer *et al.* 2003; Tompkins *et al.* 2011). Social relationships in space and time influence the likelihood and duration of contacts between hosts. Behaviours that influence close-contact interaction rates within social groups are known to be important factors explaining the local spread of a wide range of infectious diseases in natural populations (Joly *et al.* 2006; Blanchong *et al.* 2007, 2008; Ramsey 2007; Woodroffe *et al.* 2009; Wendland *et al.* 2010). In familial (e.g. matrilineal) groups, where the individual composition and size remain relatively constant, the number of contacts between infectious and susceptible individuals is independent of fluctuation in population density (de Jong *et al.* 1995; for examples see Miller *et al.* 2000; Gross & Miller 2001; Grear *et al.* 2010). As a result, in these types of social systems contact rates are constant and transmission is assumed to occur predominantly within kinship groups, potentially limiting a pathogen's spread. This form of transmission is referred to as frequency-dependent. Alternatively, in contrast with frequency-dependent transmission is

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density-dependent transmission. This form of transmission is expected in fission–fusion societies where group and host density affect some aspect of contact rate, which results in a higher risk of disease spread between local groups (Begon *et al.* 2002).

However, the spread of infectious diseases occurs over multiple spatial scales (Cross *et al.* 2005). In many wildlife species, different social groups exist as assemblages of spatially delimited groups inhabiting a landscape of patchily distributed habitats [e.g. red deer, *Cervus elaphus* (Coulson *et al.* 1997), wild boar, *Sus scrofa* (Gabor *et al.* 1999) and blackbuck antelope, *Antilope cervicapra* (Isvaran 2007)]. Thus, the transmission and prevalence of infectious diseases depends not only on the movements within social groups but also on connectivity among host groups and long-distance dispersal across the landscape (e.g. Blanchong *et al.* 2007, 2008; Pope *et al.* 2007; Cross *et al.* 2009; Cullingham *et al.* 2009, 2010; Beik & Real 2010; Grear *et al.* 2010).

Despite the crucial importance of integrating within group social interactions and population structure in the study and management of infectious diseases, few studies have simultaneously considered these two factors. Here we integrate individual and landscape-level approaches (Fenton *et al.* 2002; Blanchong *et al.* 2008; Cullingham *et al.* 2010, 2011) to assess how spatial structure in the host population and complex social relationships among individuals may affect the risk of disease spread and prevalence. Specifically, we focus on how the social and population structure of elk (*Cervus canadensis manitobensis*) could influence the prevalence and horizontal spread of bovine tuberculosis (*Mycobacterium bovis*) in a fragmented landscape.

Bovine tuberculosis is a multihost pathogen affecting a wide range of species worldwide (Daszak *et al.* 2000). However, in North America (north of 32° latitude), tuberculosis is primarily associated with three geographically separated populations of wild, social ungulates. In the northern boreal plains (Alberta-Northwest Territory, Canada), bison (*Bison bison*) is the only confirmed reservoir for tuberculosis in the wild (Wobeser 2009). Although white-tailed deer (*Odocoileus virginianus*) also host TB in the region, elk are suspected to be the primary wild reservoir in the prairie parkland of Manitoba, Canada (Lees 2004; Nishi *et al.* 2006), whereas in the temperate forest biome of Michigan, USA, tuberculosis is mostly found in white-tailed deer (Schmitt *et al.* 1997). Of the wild cervids with tuberculosis, elk has the most complex social structure (de Vos *et al.* 1967; Geist 1974).

The elk population of The Riding Mountain Region (RMR, Manitoba) therefore represents a unique opportunity for studying pathogen transmission from local social scales of behaviour to large-scale landscape phenomena. After the elk population collapse associated with post-European colonization (O'Gara & Dundas 2002), the RMR population has remained relatively isolated. Currently, the RMR is a discontinuous population that inhabits a fragmented landscape of protected areas [Riding Mountain National Park (RMNP), and Duck Mountains Provincial Park and Forest (DMPP&F)]. From 1991 to 2009, clusters of tuberculosis were detected in the population (Nishi *et al.* 2006; Brook & McLachlan 2009). The observed variance in apparent prevalence across the region [most of the infected individuals are found in western RMNP, see discussion (Shury & Bergeson 2011)] implies that biotic and/or abiotic factors affect disease spread. At a landscape scale, we investigated whether the spatial distribution of tuberculosis apparent prevalence correlates with the presence of natural and/or anthropogenic barriers that may limit regional dispersal. Our results unveiled that the apparent distribution of tuberculosis in the region coincides with that of a relatively isolated subpopulation of elk. In addition, we assessed how elk social structure may affect the spread of the disease at a local scale (i.e. within subpopulations). Specifically, we investigated whether social structure resembles that of familial groups by studying how the frequency and duration of social interactions were affected by the degree of genetic relatedness. We revealed a lack of stable familial groups. Integrating information about group social interactions and population structure has allowed us to better understand apparent prevalence of tuberculosis in the region and discuss the potential for frequency- vs. density-dependent disease transmission in this system (see Grear *et al.* 2010, Cullingham *et al.* 2011).

Materials and methods

Study area

The Riding Mountain Region of southwestern Manitoba, Canada comprises Riding Mountain National Park (RMNP; 3000 km^2) and the Duck Mountain Provincial Park and Forest (DMPP&F; 3800 km^2) (Fig. 1). The region lies in the transition from the Prairie ecoregion to the northern Boreal Plains ecoregion (Bailey 1968). The eastern portions of RMNP rise an abrupt 475 m from the Manitoba lowlands to the Manitoba escarpment, resulting in variation in vegetation (Caners & Kenkel 2003) and local climate. Both reserves are surrounded by agricultural land, which largely precludes movement of elk in and out of the region (Brook 2008; Vander Wal 2011). The RMNP and DMPP&F are connected by the remnants of a once extensive corridor, which has been fragmented over the last five decades by agricultural expansion (Walker 2001). A major highway (Hwy 10), a secondary road (Hwy 19) and

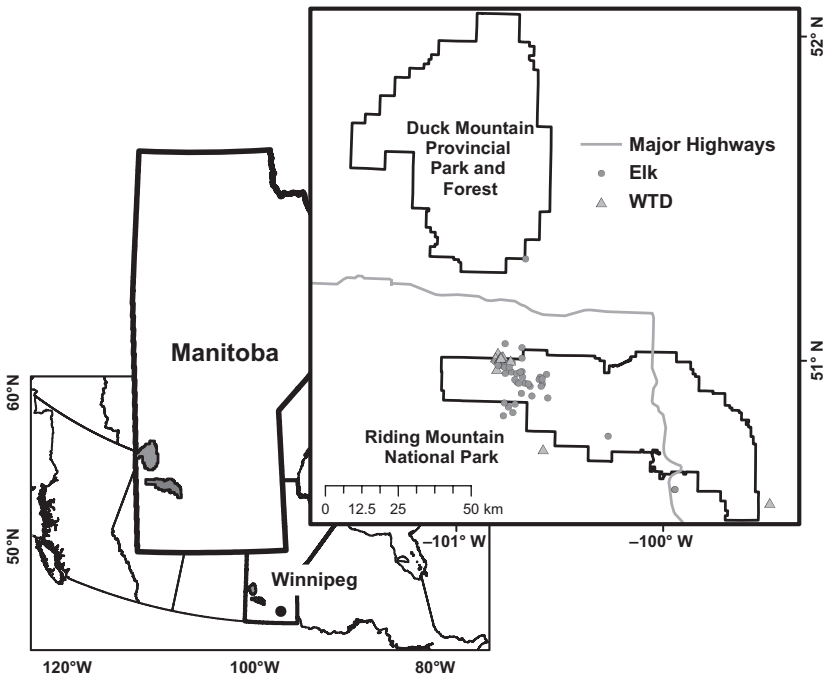

Fig. 1 Riding Mountain National Park (RMNP) and Duck Mountain Provincial Park and Forest (DMPP&F), Manitoba, Canada with spatial locations of bovine tuberculosis positive elk (n = 42) and white-tailed deer (WTD; n = 11) from 1991 to 2010 illustrating the grouped distribution of disease in the Riding Mountain Region. Only three cases of tuberculosis in wildlife have occurred outside of the western RMNP: one elk that was assigned to the DMPP&F, one elk in central RMNP and one WTD in the east end of RMNP.

electrical transmission lines also cross RMNP. Because of the built infrastructure, agriculture development and natural physiography, the elk population is spatially structured (Brook 2008; Vander Wal 2011). At present, the occurrence of TB is not ubiquitous in the regional elk population. Although the apparent prevalence remains relatively high in the west side of RMNP (4.0%; i.e. 29/723), the number of diseased animals in the east side of the park is much lower (0.0%; i.e. 0/179), and in DMPP&F (<1%; i.e. 1/116 Fig. 1; see Rousseau & Bergeson 2005 and Shury & Bergeson 2011 for a description disease testing protocol, and Rohonczy *et al.* 1996; Surujballi *et al.* 2009 for TB assay descriptions used therein). The presence of TB in and around RMNP has resulted in significant economic repercussions (Nishi *et al.* 2006; Brook & McLachlan 2009). [Correction added after online publication 8 February 2012: in the preceding paragraph the prevalence in western RMNP and the east side of RMNP was corrected.]

Sampling

We captured free-ranging elk (n = 312) from 2005 to 2008 using a net-gun fired from a helicopter (Cattet *et al.* 2004). Location of capture was recorded and available for n = 172 individuals. This latter subset was used for all geo-referenced analyses. Tissue and hair samples were obtained from the ear and frozen until processed for genotype analysis. In 2007 and 2008, we captured and fit 49 (23 females and 26 males) and 55 (35 females and 20 males) adult elk, respectively, with Sirtrack Proximity Logger radio-collars (Sirtrack Ltd., Havelock North, New Zealand, see Vander Wal (2011) and Vander Wal *et al. in press* for examples). Animals were collared for 1 year, and proximity collars operated continuously during that time. Our proximity loggers were programmed to activate and collect data whenever a collared elk came within 1.4 m of another collar [initiation distance 1.42 ± 0.22 m; separation distance 1.98 ± 0.32 m ($\bar{x}$ ± 95%CI) (Goodman 2007)]. For a large bodied animal, this distance was assumed to reflect direct interactions. Collars deactivated recording an encounter each time a pair separated for >30 s. These collars recorded a count and duration of the number of pairwise interactions (*sensu* Whitehead & Dufault 1999) between animals wearing collars. Our research followed Animal Care Protocol #20060067 of the University of Saskatchewan, following guidelines of the Canadian Council on Animal Care.

Genotyping

We isolated genomic DNA from ear plug samples using DNAdvance™ (Agencourt®; Agencourt Bioscience Corporation) following the manufacturer's instructions. Amplifications were carried out using Qiagen type-it microsatellite kit in nine different multiplex reactions (detailed descriptions of multiplex reactions, PCR primers and conditions are available in Table S1, Supporting information). We checked for genotyping errors using MICROSATELLITE TOOLKIT version 3.1 (Park 2001) and estimated null alleles and large allele dropout in MICRO-CHECKER version 2.2.3 (Van Oosterhout *et al.* 2004). No

significant pairwise linkage disequilibrium was detected. Potential null alleles were detected in only one locus. Overall, loci conformed to HW proportions for each locus, and only four loci (T26, T126, RBP3 and BM848) show significant deviations.

Spatial Bayesian clustering

To study the population genetic structure at a regional scale and the location of possible genetic boundaries, we first examined the population genetic structure using the Bayesian clustering method implemented in GENELAND version 3.2.1 (Guillot *et al.* 2005). This method uses geo-referenced individual (n = 172) multilocus genotypes for the inference of the number (K) and spatial distribution of clusters (subpopulations) in Hardy–Weinberg and Linkage Equilibrium. Given the recent history of the sampled population(s), we expected low genetic differentiation, if any, among clusters. Therefore, we assumed a Dirichlet (i.e. correlated frequency) model to determine the number and composition of genetically differentiated subpopulations (See Appendix S1, Supporting information for analyses details). A potential problem with this approach is that the sampling schemes and deviations from random mating not related to barriers to gene flow can have a strong influence on the detection and interpretation of genetic structure. More specifically, spatial Bayesian clustering can result in the overestimation of genetic structure for data sets characterized by continuously distributed individuals and spatially autocorrelated allele frequencies (Schwartz & McKelvey 2009; Frantz *et al.* 2010). Thus, we complement the Bayesian approach with spatial multivariate analysis to detect any potential continuous gradient in the genetic pattern.

Spatial multivariate analysis

Spatial principal component analysis (sPCA, Jombart 2008) makes no assumptions regarding Hardy–Weinberg equilibrium. Instead, sPCA defines eigenvalues that optimize the product of the genetic variance and Moran's I (Moran 1948, 1950), summarizing spatial patterns of genetic structure. These patterns are then separated into positive (i.e. global) and negative (i.e. local). Global patterns identify clines in allele frequencies and genetically distinguishable groups, whereas local ones detect differentiation between neighbouring individuals (Jombart 2008). We performed all calculations in R 2.11.1 (R Development Core Team 2010) using the ADEGENET (Jombart 2008) and ADE4 (Dray & Dufour 2007) packages (see Appendix S1, Supporting information for a detailed description of the sPCA analyses).

Social interactions and relatedness

Here we describe the social structure of elk focusing on two aspects of social behaviour that are potentially relevant to horizontal disease transmission: frequency and duration of close-contact dyadic interactions (see Begon *et al.* 2002). Interaction frequency (i.e. number of interactions per year) was partitioned into four categories: 0, ≥1, ≥10 and ≥100 dyadic interactions. We choose these categories because they reflect the differing degrees of social familiarity (Vander Wal 2011). Animals that did not interact within 1.4 m of one another were not considered socially familiar. As the threshold of interactions increased from 1 to 100, we assumed that animals were (i) increasingly socially familiar and (ii) more closely associated. Similar to many social cervids, elk segregate sexually outside of the breeding season (see Main & Coblentz 1990; Ruckstuhl & Neuhaus 2002; Bowyer 2004 for reviews). Thus, we divided dyadic interaction rates and duration by sex-specific dyad: female–female, male–male and female–male.

We estimated pedigree relationships between male–male, male–female and female–female dyads using Lynch and Ritland (R_{LR}) (Lynch & Ritland 1999) as implemented in IDENTIX (Belkhir *et al.* 2002). We chose this index because it shows the best performance in natural populations of outbred vertebrates (Csillery *et al.* 2006), and because it performs well in substructured populations (Casteele *et al.* 2001; Csillery *et al.* 2006; Oliehoek *et al.* 2006). The distribution of R_{LR} values among all individuals for which we had genetic data (n = 312) was evaluated, without respect to the interaction rates to determine the overall degree of pairwise relatedness of elk in the entire studied area as well as within the east and west areas of RMNP, the two spatially distinct groups for which we had interaction data (n = 104). We bootstrapped (n = 1000 iterations) relatedness of (i) the population, (ii) animals that did not interact within a cluster and (iii) animals that interacted ≥1, ≥10 and ≥100 times, the samples permitting. Furthermore, owing to assumed pseudo-replication present in all pairwise data, we used randomized general linear models (GLMs) to test the relationship between pairwise relatedness and median duration of dyadic interactions. Randomized GLMs compare the observed linear relationship in the data against a distribution of values derived from repeated GLMs of randomizations of the data (see Manly 1998 for further explanation). Median interaction duration per dyad was transformed ($\log_{10}$) to improve normality. Bootstrap and linear model analyses were implemented in R 2.11.1 (R Development Core Team 2010).

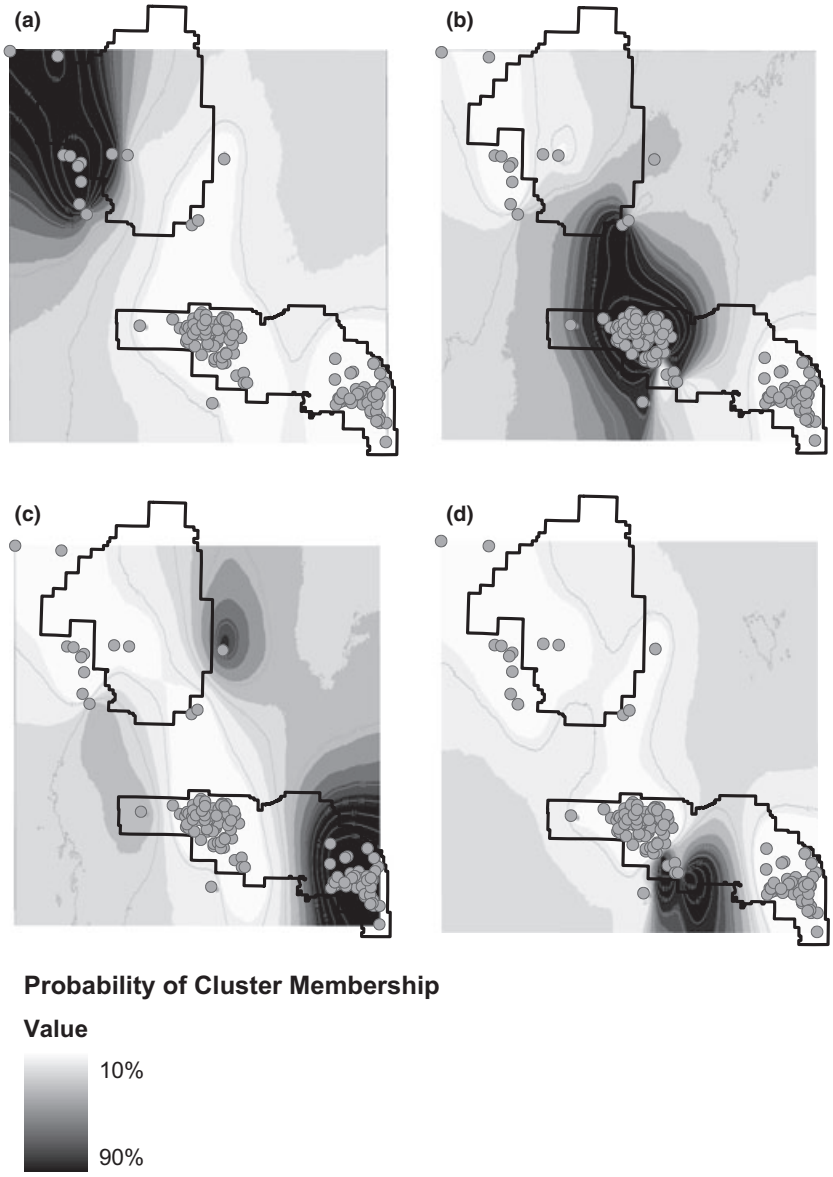

Fig. 2 Spatial Bayesian clustering of posterior probabilities of population membership and genetic discontinuities from the spatial model in GENELAND for the sampled elk. Contour lines indicate the predicted spatial position of genetic discontinuities. Black areas indicate a high probability of group membership (≥90%). Four genetic clusters were identified, each depicted in one of the maps: (a) subpopulation in Duck Mountain Provincial Park and Forest, (b) subpopulation in west Riding Mountain National Park, (c) subpopulation in east Riding Mountain National Park and (d) small subpopulation in south-central Riding Mountain National Park.

Results

Spatial Bayesian clustering

GENELAND analyses consistently identified four population clusters (*K*; Fig. 2). Individual animals had a probability of 0.8–0.9 of belonging to the assigned cluster (Fig. 2), providing strong support for the clustering result. The clusters defined by the posterior probabilities were named as follows: (i) the DMPP&F individuals, (ii) west RMNP individuals, (iii) east RMNP individuals and (iv) small group of individuals south-central RMNP. Mean F_{ST} calculated by GENELAND range between 0.0423 and 0.0845. AMOVA analysis revealed that almost all genetic variation resides within clusters, and that only a small (3%) but significant ($P < 0.0001$) amount of the genetic variation is related to differences among the inferred clusters.

Spatial multivariate analyses

Spatial principal component analysis (sPCA) scores can detect clines and spatial groups (global structures) as well as strong genetic differences between neighbours (local structures—see Jombart 2008). As expected, results from the GENELAND Monte Carlo tests revealed global structure ($P = 0.0013$). We did not find significant local structures ($P = 0.273$). The first two eigenvalues were large compared with the others and therefore were retained (Fig. 3). Individual scores on these two axes are shown on Fig. 3b. The first scores revealed a pattern consistent with the Bayesian cluster-

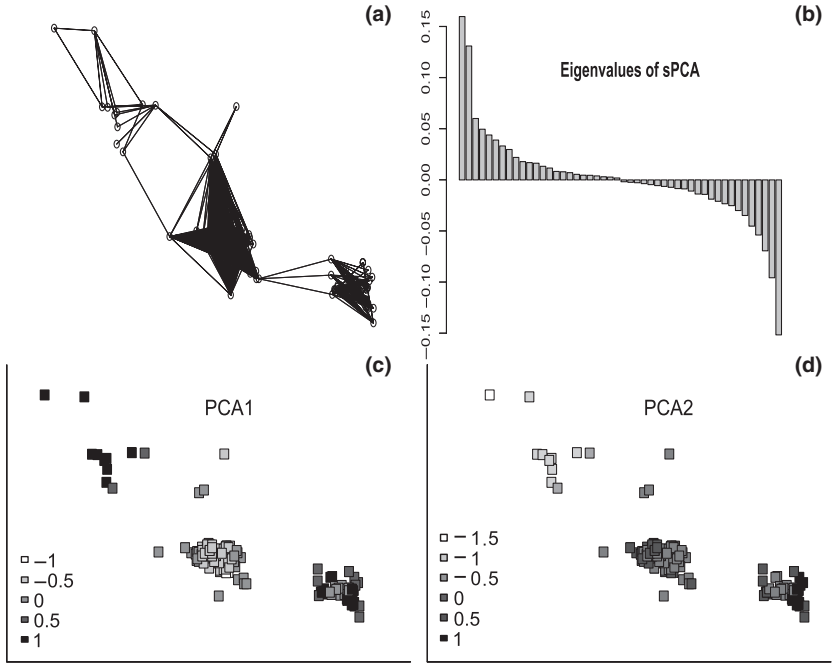

Fig. 3 Analysis of global eigenvalue scores for the spatial principal component analyses (sPCA) performed on the genotyped elk (at 30 microsatellites) from Riding Mountain National Park (2005–2008). All plots are positioned according to their spatial coordinates. (a) Connection network (produced by minimum neighbour distance) used in the analysis. (b) Global and local eigenvalues of the analysis. In (c) and (d), large black squares correspond to high positive autocorrelation scores, whereas large white squares correspond to high negative scores. First axis PCA scores (c) identified the west subpopulation cluster. The second axis PCA scores (d) illustrate gradual variation in autocorrelation scores, which represents clinical, isolation-by-distance genetic variation.

ing and clearly differentiated the west RMNP individuals from the rest (Fig. 3c). This pattern was associated with a strong spatial structure ($I = 0.616$). The second scores also show strong spatial structure ($I = 0.542$). DMPP&F individuals have high negative scores on the second axis, whereas RMNP individuals, especially those in the east, tend to have positive scores. These second scores do not show sharp boundaries between patches but a progressive change from the NW to the SE, suggesting that this global structure may be a cline (Fig. 3d).

Relatedness and social interactions

Population-scale mean pairwise relatedness was not consistently different from each class of interacting animal [≥1 or ≥10 (Fig. 4) or ≥100] in either east or west RMNP clusters. In four cases (Fig. 4d,f,k,l), however, marked noninteracting elk were more closely related to one another than they were to the population-scale mean. These groups were not more related than interacting animals within the same cluster. This result became clearer when we compared sampled individuals with population cluster (derived from the landscape analyses above), rather than the total population (Fig. 4). In all cases and within every class of interaction (including noninteraction), individuals were not more related to one another than they were to the mean relatedness of the population cluster (Fig. 4, $P > 0.05$). For males from the west cluster sampled in 2007 (Fig. 4e), interacting individuals were less related than the population mean, the population cluster mean and less related than noninteracting males. This result was not replicated in 2008 because of low sample size (Fig. 4g), nor was it apparent in males from the eastern cluster (Fig. 4f,h). Similarly, no linear relationship is apparent between pairwise relatedness and interaction duration of female–female dyads (Fig. 5a): $P = 0.95$ ($t = 0.07$, randomized t distribution 95%CI 1.98−−1.89; $n = 222$); male–male dyads (Fig. 5b) $P = 0.15$ ($t = 0.81$, randomized t distribution 95%CI 2.11−−2.00; $n = 34$); female–male dyads (Fig. 5c) $P = 0.23$ ($t = -1.47$, randomized t distribution 95%CI 1.99−−1.98; $n = 30$).

Discussion

The spread of infectious diseases in natural populations is innately a spatial process facilitated by social intercourse among hosts. Here we have combined landscape genetics and relatedness studies to assess the dispersal patterns and social structure of the prairie-parkland population of elk (Manitoba, Canada), one of the three

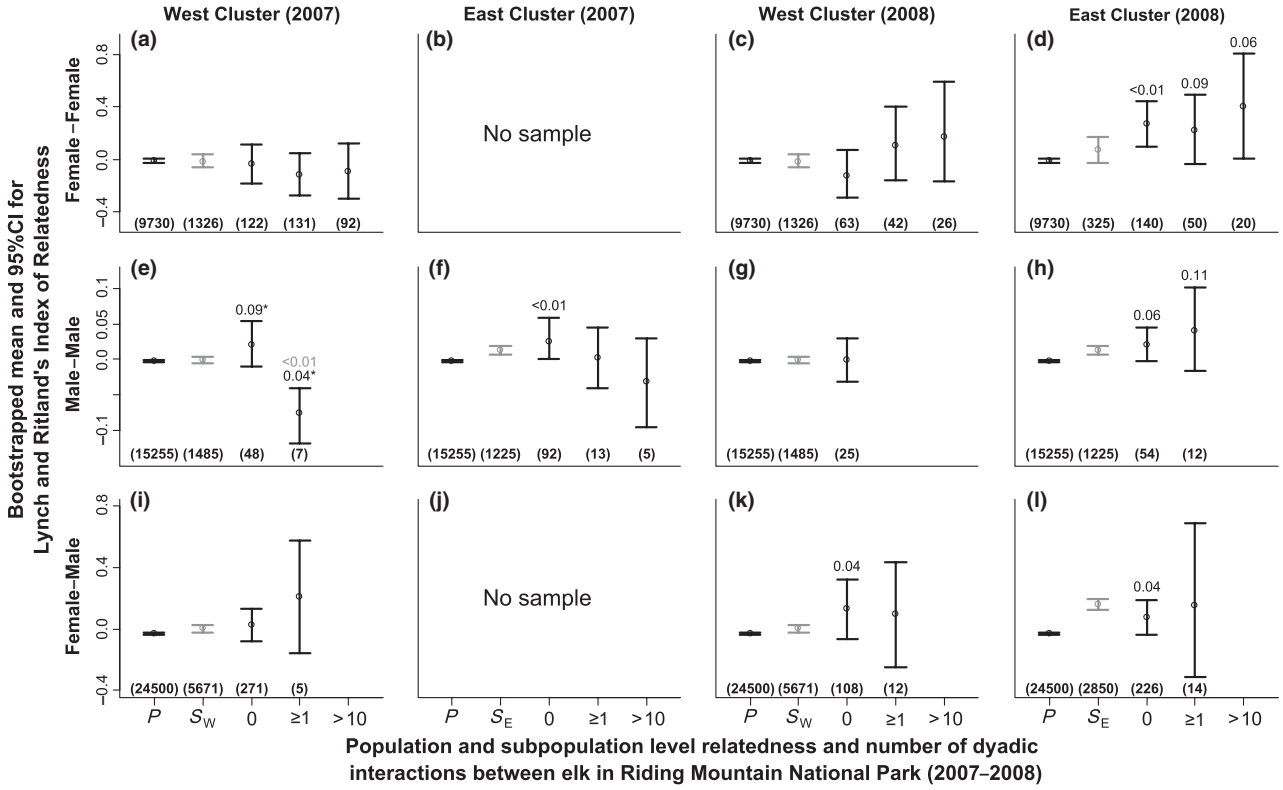

Fig. 4 Bootstrapped sex-specific pairwise relatedness by dyadic interaction frequency (i.e. degree of social association) dissected by subpopulation cluster [in east and west Riding Mountain National Park (RMNP, MB, Canada)] and sample year (2007 and 2008) for 104 elk in RMNP. All test statistics with $P < 0.15$ comparing noninteracting (0) and interacting (≥1 and ≥10) dyads with the pairwise population relatedness (P) appear in black and with pairwise subpopulation relatedness [east (S_E) and west (S_W)] appear in gray. *P*-values in west cluster male–male dyad (e) comparison between noninteracting dyads and interacting dyads within the subpopulation (indicated with *).

main reservoirs of the bovine tuberculosis in North America.

At a landscape scale, spatially explicit genetic analyses of the host show reduced gene flow at the regional level. Both Bayesian (GENELAND) and multivariate analyses (sPCA) identified a genetic cluster geographically congruent with the group of elk present in western RMNP. Telemetry data support this structure. Between 2004 and 2009, only three of 400 elk (all juvenile males) actively tracked with biotelemetry in and around RMNP moved across the 30 km separating the northwest part of RMNP and the southern border of the Duck Mountain Provincial park (Brook 2008; Vander Wal 2011; Parks Canada unpublished data). Although narrow habitat corridors that appear to connect RMNP and DMPP&F are still present in the agricultural matrix surrounding both parks, these corridors have been severely fragmented over the last five decades (Walker 2001) and are bisected by many roads. Although elk are highly vagile (Kie *et al.* 2005), our results suggest that in current circumstances, movements are now restricted, and dispersers between the two parks rarely interbreed. The genetic discontinuity between the east and the west areas of RMNP is also supported by telemetry data. None of the more than 400 elk monitored in RMNP has moved between the east and west clusters (Brook 2008; Vander Wal 2011; Parks Canada unpublished data). This is likely influenced by reduced dispersal across highway 10, which appears to act as a barrier to movement. Primary roads, alone or in conjunction with other landscape features, have been identified as dispersal barriers for elk and other large and highly mobile herbivores (Woods *et al.* 1996; Epps *et al.* 2005; Pérez-Espona *et al.* 2008).

The Bayesian analysis identified at least two other distinct genetic clusters, DMPP&F and east RMNP. However, our multivariate analysis did not support this subdivision. Instead, it showed the existence of a continuous cline from the NW to the SE. Accordingly, these two clusters are likely the consequence of uneven sampling along a genetic cline (see Schwartz & McKelvey 2009). Two nonmutually exclusive biological phenomena could explain this cline, mating with proximal individuals and the existence of a transitional

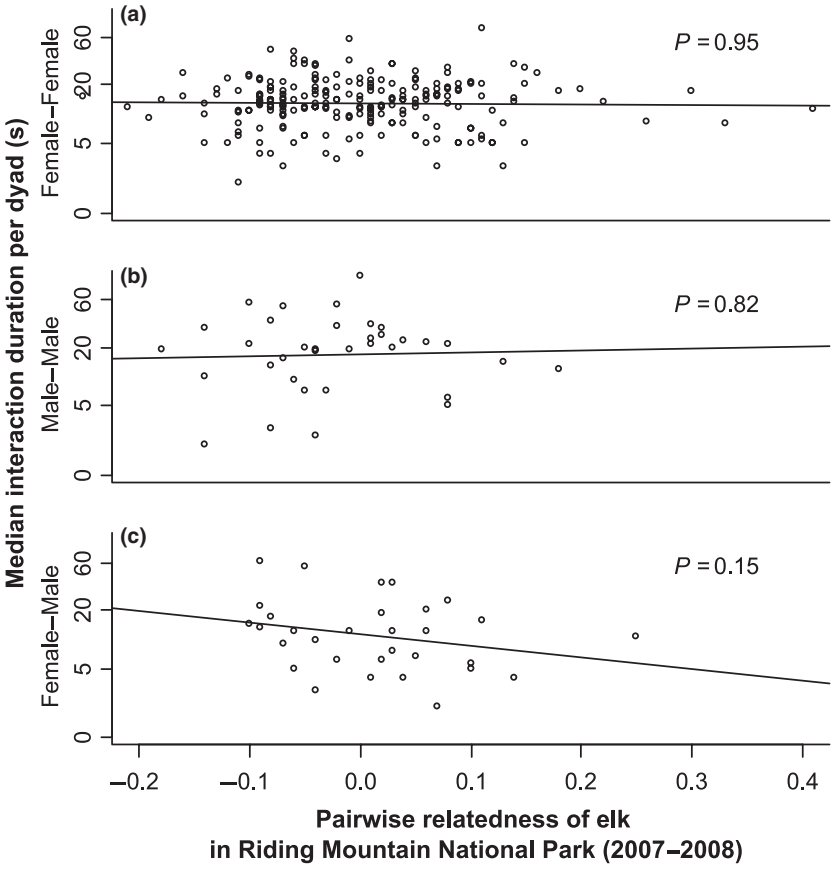

Fig. 5 Relationships between interaction duration and pairwise relatedness—Lynch and Ritland (R_{LR})—in Riding Mountain National Park (2007–2008).

zone between historically divergent subpopulations of elk (Turner 1906; Green 1933; Banfield 1949). Thus, the interplay between historical and contemporary factors (Brook 2009) and the superimposition of anthropogenic barriers upon a genetic cline seem to explain existence of a largely isolated subpopulation of elk in west RMNP.

Understanding landscape permeability and the selectiveness of host dispersal barriers can be used to understand spatial heterogeneity in pathogen prevalence and the risk of spread (e.g. Blanchong *et al.* 2008 Cullingham *et al.* 2010, 2011). During the last decade, clusters of tuberculosis have been detected in the studied population, and almost all cases have been restricted to west RMNP (Nishi *et al.* 2006; Brook & McLachlan 2009). Our results show that natural landscape features combined with anthropogenic disturbances are important factors affecting elk movements and that tuberculosis is almost endemic to this semi-isolated subpopulation At a fine-grained spatial scale, elk have been shown to avoid roads and highways (Lyons 1983; Woods *et al.* 1996; Anderson *et al.* 2005; Harju *et al.* 2011), although in montane coniferous forest of Arizona, roads seem to be relatively permeable (Dodd *et al.* 2007). In contrast, we found infrequent exchange among subpopulations of elk, which strongly suggests that primary roads in our study area are mostly impermeable barriers that have a considerable effect on elk movement and gene flow. Consequently, connectivity and communication between infected and uninfected subpopulations are low. This suggests a low potential for long-distance disease spread through the movement and dispersal of infected individuals.

The introduction of disease into a spatially structured population with low connectivity can have a significant effect on the persistence of subpopulations (Hess 1994, 1996) and in some cases [e.g. devil facial tumour (McCallum 2008)] can cause the local extirpation or extinction of the host (Best *et al.* 2011). To date, no population-level health or demographic effects of TB infection have been detected for RMR elk. Strong population structure may accord some population-scale protection from diseases that cause host mortality at a timescale smaller than host dispersal among subpopulations (Cross *et al.* 2005). If an acute disease is introduced into a structured population, some subpopulations may be extirpated (Hess 1996), and consequently genetic variation may be lost (McCauley 1991). However, in RMR elk, the majority of genetic variation is maintained in the neighbouring subpopulations from which coloniza-

tion is most likely to occur naturally or via human intervention, e.g. translocation.

At a local scale (i.e. within subpopulations), close-contact interactions are a critical parameter affecting pathogen transmission and ultimately disease prevalence. Thus, social behaviours that influence the process of disease transmission are key factors explaining the distribution and spread of diseases (Altizer *et al.* 2003). Previous studies of white-tailed deer suggest that, in social cervids, contacts within family groups are a key factor explaining the transmission of infectious diseases (Farnsworth *et al.* 2006; Joly *et al.* 2006; Blanchong *et al.* 2007; Grear *et al.* 2010). In white-tailed deer systems, individuals form small, stable matrilineal groups (Mathews & Porter 1993; Miller *et al.* 2011). Accordingly, at small spatial scales (3–4 km), disease transmission is likely to be socially constrained and driven by interactions among relatives. In contrast, our data clearly show that social interactions in elk are not necessarily based on the genealogical relationship between individuals and that close-contact interaction rate and duration [which may be indicative of different behaviours and different behaviours may influence the probability of successful transmission (e.g. Drewe *et al.* 2011)] do not covary with genetic relatedness. Elk form relatively large groups of several mixing matrilineal subgroups (Geist 1982), and at small scales, these groups seem to mix freely (Millspaugh *et al.* 2004). This results in a very different type of social unit that has important ecological and behavioural implications.

In matrilineal systems, such as the white-tailed deer, transmission is most likely to be frequency-dependent (Miller *et al.* 2000; Gross & Miller 2001; Wasserberg *et al.* 2009). In this type of model, highly virulent pathogens (i.e. those causing high mortality rates) may lead to local population extinctions. Although frequency-dependent transmission is not universally assumed to be true for white-tailed deer (see Schauber & Woolf 2003; Smith *et al.* 2009; Cross *et al.* 2010), it still remains a chosen model for transmission (Grear *et al.* 2010). That we failed to detect similar structuring among elk social units provides some evidence that highly virulent pathogens in elk might display density-dependent transmission and thus have critical community sizes (Bartlett 1957) and thresholds for pathogen establishment or fade-out (Lloyd-Smith *et al.* 2005). However, our analysis of relatedness within social groups does not preclude the possibility that elk may form nonkin, but stable social groups that may affect whether transmission is frequency- or density-dependent.

The spread of communicable diseases is a multispatial process (Cross *et al.* 2005, 2009; Heisey *et al.* 2010). We provide small-scale mechanistic (i.e. interaction rates and durations) evidence for sex-specific admixture of social interactions among elk, which suggests ease of pathogen spread at local scales. However, this is contrasted to landscape-scale evidence for population structure (i.e. clustering), which creates barriers to the spread of disease among demes. Both scales reinforce the importance of a multispatial approach to avoid erroneous conclusions regarding relatedness and social behaviours upon which pathogen transmission is predicated and the likelihood of interdeme transmission within a regional population. The combination of landscape-scale genetics, pairwise relatedness and local-scale social behaviours is a promising approach to understanding and predicting landscape-level pathogen transmission in social systems affected by transmissible diseases.

Acknowledgements

Funding and logistic support was provided primarily by Parks Canada Agency (Riding Mountain National Park of Canada), the Natural Science and Engineering Research Council of Canada and PrioNet Canada. Simpson and B. Simpson adroitly flew elk relocation flights, and C. Wilson, T. Vandenbrink and T. Shury efficiently and safely handled elk. R. Grzela, S. McKay and R. Robinson are acknowledged for assistance recovering collars. I. Potts and Y. Plante are acknowledged for microsatellite analysis. We thank K. Kingdon, D. Bergeson and T. Sallows at Parks Canada, and R.K. Brook, F. Messier, P.D. McLoughlin, M. Musiani and A. McDevitt for their advice and guidance.

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Data accessibility

Microsatellite data uploaded as Supporting Information.

Supporting information

Additional supporting information may be found in the online version of this article.

Table S1 Information on microsatellites used for genotyping.

Table S2 Genotyped elk microsatellite panel.

Table S3 Individual elk capture locations.

Appendix S1 Additional methods for Geneland and sPCA analyses.

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