Shared resources and disease dynamics in spatially structured populations

Charles L. Nunn a,b,c,*, Peter H. Thrall d, Peter M. Kappeler e

a Department of Evolutionary Anthropology, Duke University, Durham, NC 27708, United States
b Duke Global Health Institute, Duke University, Durham, NC 27710, United States
c Department of Human Evolutionary Biology, Harvard University, Cambridge, MA 02138, United States
d CSIRO Plant Industry, GPO Box 1600, Canberra, ACT 2601, Australia
e Behavioral Ecology and Sociobiology Unit, German Primate Center, 37077 Göttingen, Germany

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ABSTRACT

Infectious agents are likely to spread among animals that live together, yet we know remarkably little about how infectious agents move among social units. Sharing of resources – such as shared waterholes during a dry season – may provide an efficient route for the transmission of infectious agents among different social groups, and thus could represent an overlooked factor in understanding disease risks in spatially structured populations. We developed a spatially explicit individual-based model to investigate a situation in which multiple individuals of a single species converge at shared resources during periods of resource scarcity (i.e., “lean seasons”). We simulated the transmission of a fecally transmitted infectious agent in a spatially explicit meta-population of 81 social groups distributed on a square lattice. Time steps in the simulation corresponded to “days,” and we simulated disease dynamics over 10 yearly cycles of normal and lean seasons. The duration of the lean season varied across 1000 independent simulation runs, as did 12 other parameters sampled from a Latin hypercube distribution. Seasonal sharing of resources had marked effects on disease dynamics, with increasing prevalence of the infectious agent as lean season duration increased (and thus, duration of resource sharing also increased). Infection patterns exhibited three phases: an initial intermediate prevalence on the normal season home range, a rapid increase in prevalence around the shared resource during the lean season, and then a rapid decline in prevalence upon returning to the normal season range. These findings suggest that seasonal migration increases disease risk when animals congregate around resources, but enables them to escape soil-borne infectious agents upon returning to their original home ranges. Thus, seasonal sharing of resources has both negative and positive effects on disease risk.

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1. Introduction

Infectious agents are ubiquitous in wild animals and can strongly impact their population dynamics. For example, 20 different species of helminths are found among feral Soay sheep on the island of St. Kilda, Scotland (Gulland, 1992), more than 80 species of macroparasites have been reported in zebras in southern Africa (Roberts et al., 2002), and nematodes drive cyclical population dynamics in grouse (Hudson et al., 1998). Importantly, many animals live in social groups, and within a group, individuals interact most commonly and directly with their group-mates while mating, grooming, and raising offspring, and indirectly when they share shelters, sleeping sites and resources (Krause and Ruxton, 2002). In general, predicting the spread of infections among individuals within groups is likely to be relatively straightforward based on patterns of social and mating behavior, rates of interaction, and sharing of habitats and resources.

In contrast to the spread of disease within groups, movement of infections between groups will be more challenging for parasites that lack other means of movement, such as mobile vectors. Intergroup disease transmission might occur when parasites “hitch a ride” in a dispersing host individual that successfully immigrates to a new social group prior to clearing the infection. An infectious stage of the parasite might also be deposited in an area of range overlap between groups, for example, through fecal contamination in overlap zones. Between-group transmission might also occur through overlap at food sites (Walsh et al., 2007), at sleeping or resting sites, or during aggressive social contacts during intergroup territorial encounters (Nunn and Altizer, 2006). In addition,
a “bridge” or reservoir host species may move a generalist parasite between two groups of another species, for example if the bridge species has a larger home range that overlaps the ranges of individuals from another species, or when multiple species converge on a single resource. Finally, it is reasonable to expect that seasonal variation in any of these behaviors will influence disease dynamics over time and space, including the spread of infectious diseases between groups, because the life cycles of parasites are adapted to seasonal changes in environmental conditions (Altizer et al., 2006).

Previous authors have modeled this situation of spatially distributed groups in an ecological context. For example, Hess (1996a,b) developed an island metapopulation model that investigated both within and among-patch dynamics of a contagious infectious agent. He found that in the absence of host resistance, increased connectivity among patches (i.e., groups) led to a greater likelihood for epidemic spread of a contagious disease. Several other models have shown that specific patterns of sociality reduce parasite risk, for example when large groups are divided into smaller clusters of locally interacting individuals. This suggests that in highly structured populations with limited dispersal, infectious diseases are less likely to become established because infectious agents are effectively contained within subgroups (Salathe and Jones, 2010; Thrall et al., 2000; Wawe and Jog, 1997; Wilson et al., 2003). In another recent model of sociality and infectious disease dynamics, Bonnell et al. (2010) developed an agent based model to investigate how the distribution of food resources influences movement and resource sharing by social primates, and how this affects movement of infectious agents through the simulated populations.

Here, we add to the understanding of disease dynamics in spatially structured populations by developing a model that focuses on seasonal shifts in ranges that are associated with sharing of a resource, such as water or food. Shared resources can impact the spread of infectious agents in at least three ways. First, aggregations of animals around resources increase direct and indirect contact rates among individuals, both within and among social groups (Aiyeni, 1975; Cooper et al., 2010; Valeix et al., 2007; Vicente et al., 2007). Second, intensified use of resources increases the local density of infectious stages of parasites in the soil (Vicente et al., 2006). Lastly, when animals leave their home ranges in search of resources, they may be exposed to infectious agents not found in their home ranges, including areas used intensively by neighboring groups (Nunn and Dokey, 2006; Stoner, 1996). Conversely, when animals return to their normal ranges, infectious stages of parasites may have cleared from the soil through parasite mortality, and disease risk is further lowered if infected hosts are less able to survive migration back to the range (Altizer et al., 2011; Hausfater and Meade, 1982; Loehr, 1995). Thus, seasonality is likely to have major impacts on the dynamics of infectious organisms, with both positive and negative effects.

In our model, the infectious agent is fecally transmitted and builds up in the environment according to habitat use. The density of infectious material is expected to increase in the environment when group ranges overlap around shared resources. We do not assume that the agent is specifically transmitted in water, food, or in soil, but only that it is gastrointestinal and acquired through ingestion of food or water, or through contact with skin.

Remarkably little is known about the role of shared water sources in wildlife disease ecology. Water sources are expected to impact movement patterns of terrestrial vertebrates (Redfearn et al., 2003; Scholz and Kappeler, 2004; Smit, 2011; Smit et al., 2007). Foot-and-mouth disease has been proposed to spread among buffalo and impala through shared drinking resources (Bastos et al., 2000), and waterholes are important in the spread of other parasites and pathogens within species (Vicente et al., 2006, 2007). Much of the effort aimed at investigating the role of waterholes as sources of disease has focused on whether water resources (and their surroundings) provide a means for infections to move between livestock and wildlife in human-altered habitats (e.g., Bengis et al., 2002), rather than how such situations might alter natural patterns of infection in wild animal populations. In addition, waterholes are interesting because experimental approaches are possible in future extensions of this research – for example, by providing artificial waterholes, using barriers to limit access to particular species, or experimentally reducing levels of infection in water sources (e.g., Hunt et al., 2007). In terms of food resources, Walsh et al. (2007) describe how groups of apes share fruit, including discarded fruit, with potential for disease transmission among social units.

In the research described here, we extended an agent-based model of disease dynamics (Nunn et al., 2011) to investigate how seasonality of resource use and ranging patterns influences disease dynamics. By building on the Nunn et al. (2011) modeling framework, we are able to compare results with and without seasonality, and to assess the role of seasonal ranging in relation to other variables in the previous model. We expected that a longer lean season would increase exposure to infectious agents that accumulate in the soil, resulting in higher population prevalence, a greater number of individuals lost due to disease, and more rapid penetration of the infectious agent into the majority of social groups. We also expected that host behavioral changes related to seasonality – such as ranging patterns – would have strong effects on seasonal variation in infection-related measures, such as prevalence. By systematically varying key environmental, behavioral and disease transmission parameters in the model, we compared the relative importance of seasonal variation with more traditional epidemiological factors, such as disease related mortality and transmission probability. Overall, the model presented here extends the previous model in new directions with additional parameters, while also investigating the previously investigated parameters (Nunn et al., 2011) in the context of seasonality.

2. Materials and methods

2.1. Simulation structure

The model was designed to investigate behavioral, ecological and epidemiological parameters that could potentially influence the spread of an infectious organism in a spatially structured population, specifically in the context of seasonal variation in resources that influence ranging patterns and inter-mingling of social groups. A fecally transmitted infectious organism was introduced at one edge of the habitat. The simulation then tracked the spatial movements of groups of individuals relative to a “core area” of the home range, and the dispersal of individuals among different social groups. Each simulation run consisted of 10 yearly cycles of seasonal change. Groups converged around shared resources during a defined period of resource shortage, which varied in length across simulation runs.

We focus on the aspects of the model involving seasonality and shared resources, but we repeat some model description for clarity and completeness (Nunn et al., 2008, 2011). Model dynamics proceeded in discrete time steps, which represented single days in the lives of host individuals. In each time step several dispersal, infection and demographic processes took place sequentially: (1) ranging and possible infection of hosts through exposure to feces in the ranging matrix, (2) deaths due to the intrinsic mortality rate and disease-related mortality, (3) stochastic dispersal of individuals to neighboring groups, and (4) stochastic births of individuals in groups to replace individuals lost to disease or other factors in those groups over time. These are described below. In addition, each year was divided into “normal” and “lean” seasons, with resources
shared among groups during the lean season. The duration of the lean season \( p \) was measured in days and was situated at the end of a yearly cycle; thus, years started with a normal season that shifted to lean season conditions at day 365 – \( p \).

Simulations took place on a \( 9 \times 9 \) square lattice (social group lattice), within which smaller square lattices of cells (range lattice) were designated that reflect a home range \( (10 \times 10 \) cells) for each group on the social group lattice. Collectively, the area of the range lattice that includes social groups for the focal host species was referred to as the reserve (Fig. 1). Within the reserve were nine equally spaced resources (Fig. 1), which were seasonally available and used by the nine most physically proximal groups during the lean season (see below). Within each home range a core area was further defined as the number of cells away from the edge of the home range that the animals preferred to use (Fig. 2). A smaller core area resulted in less home range overlap among groups. When this parameter equaled zero, the core area and the home range coincided \( (10 \times 10, \text{i.e.} 100 \) cells), when the parameter equaled 1, the core area represented the inner 64 cells \( (8 \times 8, \text{with a one unit separation between core and home range boundaries}) \), when the parameter equaled 2, the core area represented the inner 36 cells \( (6 \times 6, \text{shown in Fig. 2}) \), and so on.

Around the reserve we added a further 10 cells of potential ranging by the groups. Infectious material was introduced continuously along the upper edge of this area (Fig. 1) at a rate of 10 infected fecal piles scattered randomly in each time step of the simulation. This was implemented to assess spatial heterogeneity in infection patterns across the population relative to the source of infections. Feces containing infectious stages of parasites accumulated in range grid cells and, following a soil incubation period on the ground, were potentially infectious during a soil infectious period to individual hosts in the focal population that were located in a grid cell with infectious material. We thus took into account that parasites exhibit an incubation period, an infectious period, and death rate of infectious stages in the soil. Many examples of life cycles with parasite mortality in the environment are found in parasites of wildlife and humans, including nematodes that infect baboons in Africa (Hausfater and Meade, 1982), Baylisascaris procyonis that infects raccoons (Procyon lotor) and other mammals (Kazacos, 2001), Giardia in a wide range of mammals (Olson et al., 2001), and hookworm (Necator and Ancylostoma) in humans (Hotez et al., 2004).

Individual hosts were associated with one of the 81 groups in the social structure lattice. Each group had a location in the range lattice that was typically, but not always, in the designated home range of that particular group. Individuals were further characterized by their infection status, including number of days in a defined host incubation period (i.e., exposed but not yet infectious) and, following host incubation, number of days in a host infectious period. During the host infectious period, fecal material was deposited in

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**Fig. 1.** Social group, ranging and shared resources. Social groups were arranged on the landscape and identified by the social group lattice, which was a \( 9 \times 9 \) lattice in all simulations presented here \( (n = 81 \) social groups). Social groups ranged within the range lattice, which was a \( 10 \times 10 \) lattice for each of the 81 social groups and contained a core area (see Fig. 2). The \( 81 \times 10 \times 10 \) ranging lattices were linked and constituted the reserve. Around this reserve, we placed a further 10 cells, producing a total potential ranging area of \( 110 \times 110 \). Along the top edge of the buffer and reserve, infectious material was introduced to the system; it penetrated the reserve within 5 cells, thus overlapping with the uppermost social groups. Nine shared resources were spaced evenly across the social group matrix. Nine groups congregated on each of the most proximate resources to their assigned (normal season) ranging lattice.

**Fig. 2.** Core area and fecal contamination. A core area was centered in each \( 10 \times 10 \) ranging area per group. This core was identified as a certain number of cells in from the range. In this case, the core area is 2 cells from the edge, giving a \( 6 \times 6 \) core area. Groups ranged with a random walk within the core, and exhibited a tendency to move toward the core when outside of it, where the bias toward the core is a function of how far the group was currently away from the core. This figure further shows the build-up of infectious material (fecal contamination) within and outside the group’s core area, indicated by black circles. An individual cell in the range matrix could have zero, one or multiple feces that harbor infection, and risk of infection increased with increasing fecal contamination.
the environment and went through the incubation and infectious period described above.

Infection occurred with transmission probability $\beta$ for each infectious pile in the ranging grid cell. We assumed that after clearing the infection, individuals had no immunity to the infectious agent and thus were susceptible to re-infection (i.e., a susceptible-exposed-infected-susceptible, or SEIS, model). While infected, however, individuals could not become infected with another parasite; thus, the individual had to move through the incubation and infectious periods to be re-infected.

Groups of individuals were formed based on user-specified values for group size by drawing random numbers from a Poisson distribution. All groups had at least two individuals, and all individuals in the population were initially uninfected. Deaths, births and dispersal of individuals will tend to cause the initial social group structure to drift over a simulation run, especially when simulations are run for many time steps. To help maintain initial demographic conditions, we retained a matrix of the initial numbers of males and females in each group. This initiating matrix was used to stochastically adjust probabilities associated with demographic parameters (birth and dispersal) to help maintain initial conditions for each group throughout a simulation run, as described below. It is worth noting, however, that disease related mortality resulted in reductions in overall population size, and thus influenced seasonal population dynamics.

The largest temporal unit of the simulation was a year, and a proportion $p$ of that yearly cycle was designated as a lean season, which was characterized by the breakdown of the groups’ home range boundaries and their movement toward its shared resource (see Fig. 1). Thus, during the lean season, the groups’ ranges overlapped to a greater extent around shared resources, but two groups did not co-occur in the same lattice cell in a given time step. The simulation ran across ten yearly cycles.

2.2. Ranging behavior and model dynamics

During the normal season, individuals moved in their own home ranges (i.e., the ranging matrix) together with other members of their social group. Groups ranged in a random walk within their core areas on the range lattice (Fig. 2), and individual members of a group stayed together in the same ranging matrix cell (i.e., groups were cohesive). Core areas were centered inside a group’s designated home range, and thus did not overlap with other groups’ core areas. Groups could range outside of their core areas, including into other groups’ home ranges and core areas, but they did so via a “rubber-band” process that tended to pull them back toward the core area (and thus is not a random walk outside the core area). More specifically, in a given time step, a random draw determined whether a group moved horizontally or vertically. For example, assuming a vertical movement was drawn, a group within its core area had an equal probability of moving either up or down (determined by drawing a second random number). Outside the core area, however, this decision to move up or down was biased by the vertical distance from the edge of the core area. To implement the stochastic “rubber-band” process, we did the following: to the base probability of 1/2 for moving up or down, we added one to both numerator and denominator for each cell away from the core for the probability of moving back to the core. Thus, if the group was one step “above” its core area, the probability of moving down on the next time step was 2/3, if two steps the probability was 3/4, and so on, asymptotically to a probability of 1. The same procedure was used for movements in the horizontal direction. Hence, the probability of movement back toward the core area increased with distance outside the core area.

During the lean season, social groups congregated around nine lean season ranges that were evenly dispersed across the lattice, with nine groups ranging around each of the nine resources closest to their normal season ranges. The home ranges and core areas were equivalent in size to those on the normal season range, but with nine groups congregating on one of nine single ranges. Movement followed the same ranging process described above, with the rubber-band process centered on the waterholes. Once the lean season was over, the groups moved back to its normal season range, with the tendency to move back governed by the rubber-band process.

Two further constraints were placed on ranging behavior. First, groups were unable to move off the total matrix, which included the social group matrix plus the buffer zone equivalent to one home range diameter surrounding the reserve (see Fig. 1). Second, groups could not occupy a grid cell already occupied by another group in that step. When movement brought a group to a boundary or an already occupied cell, the stochastic selection of movement direction was repeated, and if a suitable range cell was not located after 10 tries, the social group remained where it was for that time step. Thus, while ranges overlapped during the lean season, the groups did not intermingle on a single grid cell; interactions between groups were thus of an indirect nature.

The core area affected the probability of overlap with other groups. Specifically, a larger core area meant that groups tended to range closer to the boundary of their home ranges before the rubber band process biased movement back to the group’s core area within its home range. In such cases, a given group could cross over into a neighboring group’s range or outside the reserve (Fig. 1). Conversely, a smaller core area (which was centered in the group’s range) meant that groups were less likely to range outside of their home ranges, resulting in decreased home range overlap during the normal season (but possibly more indirect contact during the lean season as groups congregated on a smaller core area).

Range use intensity was also varied systematically. In primates and other mammals, researchers have used a measure known as the defensibility index ($D$-index) to measure range use intensity (Mitani and Rodman, 1979). The $D$-index measures the intensity of range use by examining day journey length relative to home range size. Here, all groups had the same home range size; hence, the $D$-index was varied by simply changing the day range. We therefore refer to day range intensity simply as day range ($D$).

Following each movement to a new cell, infectious individuals defecated with probability $d$. The location of fecal piles was recorded on the range lattice based on the location of the group, and following the soil incubation period, they became infectious to individuals occupying that cell in future time steps. Uninfected individuals in the range cell were exposed to infectious fecal material and became infected with probability $\beta$ per fecal pile in the cell.

Individuals also experienced disease-related and background mortality. Each individual experienced a baseline probability of death ($m_0$), and infected individuals had an additional source of mortality due to disease ($m_i$), where $m_i$ was a simple multiplier of $m_0$ (range of values is given in Table 1). Infected individuals that died were removed from the simulation and could no longer infect other individuals.

Dispersal of individuals to neighboring groups occurred with probability $i$. Individuals always moved to adjacent neighboring ranges (on flat side or corners of current range). The new group was selected randomly, and thus not determined based on group size. Dispersal was completed in one time step.

Lastly, births occurred for groups with at least one individual present. We assumed that the population was at carrying capacity when the simulation was initiated. Thus, we recorded the initial population size and also the initial sizes of each group, and assigned a higher probability of birth if the current population size was less than the initial population size (and conversely, a lower probability if the current population was larger than its initial size).
2.4. Statistical analyses of model output

First, we used general linear models to investigate how parameters from the Latin hypercube sample influenced average prevalence, population decline due to disease (based on all groups combined), and number of groups infected. Because significance levels are sensitive to sample size and here we are interested in relative effects, we avoided interpreting the findings based on frequentist statistical tests of null hypotheses, such as p-values. Instead, we used an information theoretic framework based on model averaging (Burnham and Anderson, 2002). Specifically, we obtained an AIC for each of the possible models, and then averaged the subset of models with Akaike weights greater than 0.001. We implemented full-model averaging, in which parameters that were not included in a model were set to 0 and included when averaging the coefficient estimates. In addition to providing the summed Akaike weights for each coefficient, we standardized coefficients and interpreted larger coefficients as representing larger effects. We provide $R^2$ as a measure of overall fit.

Analyses were conducted in R (R Development Core Team, 2009) with the packages MuMIn (Bartoň, 2011) and QuantPsyc (Fletcher, 2012). We investigated a variety of transformations, including logit and log, and interaction terms to improve the statistical performance of the models, but the bimodal nature of some output variables made it difficult to achieve perfect normality of residuals. Thus, we also provide simple graphical output for data from simulations that varied during the lean season duration while holding other variables constant.

A critical assumption is that the simulation runs reached a stable distribution, i.e., stationarity. We investigated whether simulations had reached a stationary distribution by visual inspection in which we compared prevalence across different simulated years (Fig. S1), and we investigated particular outliers. For most runs, the model had clearly reached stationarity by the last “year” of the simulation (i.e., time steps 3286–3650). As a further precaution, we re-ran the statistical analyses after removing all runs in which median prevalence differed by more than 2 percentage points across the last two “years” of the simulated output (52 cases in the 1000 total runs). These analyses produced largely congruent results and are not presented here.

Supplementary material related to this article can be found, in the online version, at http://dx.doi.org/10.1016/j.ecolmodel.2013.10.004.

3. Results

Prevalence of infection and host population size varied greatly across simulation runs, with average prevalence over the last year of the simulation ranging from 0 to 99.4% (median = 85.3%) and substantial host population decline in most simulation runs (Fig. 3). We generally found cyclical dynamics across runs, with prevalence varying according to the degree of seasonality (Fig. 4). The parasite quickly swept through the entire population, producing low levels of spatial variation relative to the source of infection (Fig. 5).

In addition to variation in the lean season duration, 12 other parameters were varied in our Latin hypercube sample. Using model averaging and standardized regression coefficients, we first investigated the predictors of average disease prevalence in the final year of each simulation. As shown in Table 2, transmission probability, the duration of parasite infectiousness in the soil, host range use intensity and group size were strong positive predictors of average prevalence, while intrinsic and disease-related mortality were strong negative predictors of prevalence. The duration of the lean season was a positive predictor of prevalence (Fig. 4) and was almost equal in magnitude to mortality rates (Table 2), with

<table>
<thead>
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<th>Parameter</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Midpoint</th>
</tr>
</thead>
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<tr>
<td>Lean season ($p$)</td>
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<td>Variable</td>
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<td>5</td>
<td>2.5</td>
</tr>
<tr>
<td>Incubation – soil ($h_f$)</td>
<td>1</td>
<td>15</td>
<td>8</td>
</tr>
<tr>
<td>Infectious – soil ($f_i$)</td>
<td>1</td>
<td>50</td>
<td>25.5</td>
</tr>
<tr>
<td>Incubation – host ($h_h$)</td>
<td>3</td>
<td>14</td>
<td>8.5</td>
</tr>
<tr>
<td>Infectious – host ($f_h$)</td>
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<td>184.5</td>
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<tr>
<td>Defecation rate ($d$)</td>
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<tr>
<td>Dispersal rate ($f$)</td>
<td>0.001</td>
<td>0.01</td>
<td>0.005</td>
</tr>
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</table>

Based on life span range of 0.5–50 years and time step of one day.

Number of movement steps per simulated time step. Range was based on values of the D-index from Mitani and Rodman (1979), i.e. 0.2–3 when converted to the D-index.

Rounded down to increments of 0, 0.1, 0.2, 0.3, and 0.4.

Integer values.

For infected hosts only, and used as a rate per day calculated as d/D.

2.3. Model parameterization and exploration

To explore how multiple parameters influence disease dynamics, we undertook multivariate analyses using random sampling via Latin hypercube sampling, which is a type of stratified Monte Carlo sampling that has been used in epidemiological modeling and is more efficient in this context than random sampling regimes or those that include all possible parameter values (Blower and Dowlatabadi, 1994; Nunn et al., 2009; Roushtan et al., 2000; Seaholm et al., 1988). Thirteen parameters were varied across flat distributions in the Latin hypercube sample: seasonality, group size, transmission probability, intrinsic and disease-related mortality, rate of dispersal, defecation rate, day range, core area, soil incubation period, soil infectious period, host incubation period, and host infectious period. Table 1 summarizes the parameters that we investigated, along with the ranges of variation that were sampled for the parameters. Parameters that required integer or discrete values for the model (e.g., host infectious period) were represented as continuously varying traits in the Latin hypercube design and then averaged to units needed for the simulation model. With this approach, we generated 1000 simulation runs from the Latin hypercube reflecting the range of variation in Table 1.

In addition to the Latin hypercube sample, we undertook an additional set of analyses to investigate how lean season length influenced disease prevalence while holding other parameters constant. We conducted these analyses using the midpoint of values from the Latin hypercube for the other parameters, as given in Table 1.

Table 1

Simulation parameters and range of values used (Latin hypercube sample).

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Integer values.

For infected hosts only, and used as a rate per day calculated as d/D.
confidence intervals that clearly excluded zero. As with these other variables, the duration of the lean season was included in all of the averaged models (i.e., importance = 1, Table 2).

We also investigated the predictors of population decline. In this case, disease-related mortality was the most influential predictor variable (Table 3), with duration of the lean season showing clear positive, but weaker, effects (i.e., larger population declines with higher mortality and longer lean season durations). The duration of the lean season was again included in 100% of the averaged models (Table 3).

Finally, in analyses of the number of groups infected, group size emerged as the most important predictor (Table 4). The duration of the lean season was again positive (with importance = 0.86), but weaker than many other variables. Thus, a longer duration of the lean season generally resulted in higher disease prevalence, greater loss of individuals from social groups, and more groups infected.

To explore the effect of the lean season further, we varied its length while holding all other parameters constant (Table 1) and found striking effects on disease dynamics. Initially, as the lean season increased, prevalence actually declined, and under the model parameters in these runs, it declined until the duration of the lean season was about 25 days (Fig. 6a). This reflects that groups are moving out of their ranges and into relatively less used parts of the range between core areas, and do not remain at shared resources for long enough for the buildup of infectious materials in those areas during short lean seasons. Thus, a short lean season may reduce disease risk by providing a means of escaping the build-up of parasites in the normal season group range and avoiding the build-up in the lean season range. However, after the initial decline, prevalence increased more or less linearly with lean season, up to about 320 days, at which point prevalence appeared to accelerate slightly. Because of this effect, it appears that up to about 75 days of lean

![Fig. 3. Distribution of simulated output: prevalence and population decline. Across 1000 simulations, average prevalence tended to be high, but showed high variance. Similarly, most simulations resulted in substantial population decline over each of the 10-year runs, where population decline is defined relative to the starting population size.](image)

![Fig. 4. Effect of lean season duration (p) on prevalence. Using three simulation runs in which lean season duration (p) and other parameters vary, average prevalence increased with increases in p. The amplitude of the variation appears to also dampen, although this may be driven by the boundary condition at 100% prevalence. Taken from the set of 1000 simulations that sampled parameters according to the Latin hypercube (ranges in Table 1).](image)
season, disease prevalence was not noticeably higher than when there were no shared resources (i.e. no lean season, see Fig. 6A). We also found a strong positive correlation between the duration of the lean season and the number of deaths due to disease (Spearman rank order correlation: $r_s = 0.77, S = 36,817, n = 99, p < 0.0001$), although some of these individuals were replenished through new births. Thus, for this set of parameters, population decline – which reflects the net effect of births and deaths at the end of the simulation - accelerated slightly as the lean season length initially
increased, but then settled into stochastic fluctuations around a mean (high) population decline of about 50% (Fig. 6b).

Looking in detail at examples from these more focused simulations, we observed that seasonal fluctuations in prevalence are basically non-existent with short lean seasons, and become much more evident for longer lean seasons (Fig. 7). In this case, however, the magnitude of prevalence fluctuations was not driven by lean season duration; the range of prevalence observed across a year is more or less constant (cf. Fig. 4). In addition, disease dynamics appeared to show three phases per year: a steady-state medium prevalence level during the normal season, elevated prevalence during the lean season, and then a drastic downward swing as the lean season ends and groups move back to their normal season ranges (by which time most of the infectious organisms will have died, especially when lean seasons are longer). For a given phase of this cycle, the prevalence of infection was relatively constant across simulations of different lean season durations. However, the phases differed in length according to lean season duration, and thus mean prevalence also varied with lean season duration (Fig. 7).

4. Discussion

Our simulations revealed strong effects of seasonal ranging on patterns of infection with a fecally transmitted infectious agent in a single-host system. Disease dynamics were driven by the intensity of range use, which increased during the lean season when ranges of more groups overlapped at shared resources, resulting in increased prevalence at the population level. At the end of the lean season, prevalence decreased rapidly in some simulations, probably due to the mortality of infected individuals (and replenishment of susceptible individuals through new births) as groups moved back to their normal season ranges. These results suggest that animals effectively escape parasites that build up in the soil around both their home ranges and shared resources, as might occur in the context of migratory behavior (Altizer et al., 2011; Berger, 2004; Loehle, 1995). How effective this is depends on the relative balance of time spent at “home” during the normal season and away during the lean season, and on specific features of parasite life history (e.g., incubation period and survival in soil). Interestingly, we found that at the start of the lean season, individuals moved into low-risk habitat for acquiring infectious organisms, especially in the interstitial space between core areas where ranging levels were low during the normal season. Thus, when lean seasons are short, animals may actually benefit from seasonal habitat use if it reduces range use intensity on the normal season range, particularly if the duration of lean season range use is too short for the accumulation of infectious organisms around shared resources. However, in terms of population dynamics, we found that host population size was negatively impacted when periodic resource sharing occurred.

Despite the importance of fecally transmitted infections in humans, remarkably little is known about their dynamics in wild animals. This gap in our knowledge is surprising because some parasitic organisms in wildlife, such as Giardia and Schistosoma, also infect humans or economically important domesticated animals, such as cattle. Our findings suggest that seasonally shared resources can amplify disease dynamics through the mechanism of multiple groups sharing a habitat. Thus, compared to a previous study, mean prevalence was substantially higher when seasonal range shifts occurred (71.8%, here) compared to when groups remained in their normal season ranges (22.4%, Nunn et al., 2011), under generally similar parameter settings and implementation of the model. In addition, population declines were lower when animals were restricted to their own ranges rather than sharing ranges during a lean season: the maximum population decline in the previous, non-seasonal simulation was 58.8% (Nunn et al., 2011), whereas in our simulations maximum population decline was 64%, with a mean loss of 41%. We also found consistent effects of the lifespan of the infectious parasite in the soil (duration of infectiousness, $f_s$) on average prevalence. Based on the magnitude of its standardized regression coefficient, $f_s$ appears to be more important in the seasonal simulation than in the model without seasonality.

Our model also predicts that when groups of animals share food or water resources differentially throughout the year, we should see strong seasonal dynamics in infection levels for fecally transmitted infectious agents, as follows: (1) an intermediate level of infection in the normal season range, especially just before transition to the lean season; (2) much higher prevalence during the lean season, driven through greater overlap at food or water sources; and (3) a marked decline during the transition back to normal season ranging, as groups escape the buildup of parasites on the lean season range and their normal season ranges have cleared many of the infectious stages. Importantly, these results suggest that the abundance of infectious stages in the soil may also undergo dramatic fluctuations, which will at least partly depend on host behavior. Without taking animal behavior into account, such fluctuations may be misinterpreted as varying relative to environmental conditions across the habitat; for example, a buildup of parasites on the normal range could appear related to greater rainfall during a normal season, when in fact it is part of the cyclical buildup in the soil as groups re-settle in their normal season ranges. Thus, our findings argue for better incorporation of host behavior into epidemiological models (see also Bonnell et al., 2010).

A recent review by Altizer et al. (2011) identified several ways that migration – here represented as seasonal shifts during a lean season – could influence patterns of infection and disease dynamics in wildlife populations. We found support for a mechanism based on migratory escape (Loehle, 1995). Indeed, previous research on primates suggested that baboons alter their use of sleeping trees to avoid the buildup of fecal contamination in the surrounding soil (Hausfater and Meade, 1982), and that mangabeys use habitat more intensively when higher rainfall has washed surfaces free of parasites (Freeland, 1980). While later findings have called these conclusions into question (Nunn and Altizer, 2006; Olupot et al., 1997), our model suggests that animals would benefit from fine-tuning their ranging behavior relative to risks from buildup of infectious organisms in the soil, possibly on a seasonal basis. It is worth noting that other types of behavior are also important. For example, ungulates are known to avoid eating near feces (Hart, 1990), and arboreal primates may defecate in ways that reduce contact with arboreal travel routes (Gilbert, 1997).

Parasites were introduced continuously into our simulated system on one edge of the reserve. This situation is similar to what would occur in the process of spillover from domesticated animals or humans and was implemented to assess spatial heterogeneity in infection patterns across the population relative to infection sources. In this study, the parasite quickly swept through the entire population, producing low levels of spatial variation relative to the source of infection (see Fig. 5). By comparison, a previous study based on the same model that lacked seasonality found that prevalence of infection was higher close to the source of infection (Nunn et al., 2011). Thus, the mixing induced by sharing of resources also amplified the spread of infections across the population. We suggest that parasites with fecal–oral transmission may be especially devastating to wildlife when sharing of resources takes place on a seasonal basis, as this seasonal sharing can lead to greater penetration of the infectious agent into the wildlife population. Disease control efforts should focus on preventing the introduction of new fecally transmitted parasites into systems from domesticated animals and humans.
In conclusion, we found that seasonal use of a shared resource greatly amplifies the spread of an infectious agent through a spatially structured population. These findings have practical importance for conservation of biodiversity, particularly in the context of parasite sharing with domesticated animals and humans. Future research could investigate more directly the behavioral mechanisms that animals use to avoid infection, and these mechanisms could be investigated within the context of a model like the one used here. Similarly, the model could be parameterized with real-world data to investigate the risk of gastrointestinal parasites at particular field locations (e.g., Bonnell et al., 2010). This may be especially important if our idealized sharing of resources evenly among groups affects disease dynamics and is violated in real-world host-parasite settings, as might be the case if groups use more than one water resource throughout the lean season. Empirically, predictions from the model could be examined experimentally, for example through use of barriers or provisioning of resources to alter ranging patterns. Finally, given the impacts of seasonal resource use on disease incidence and prevalence, there could also be implications for the evolution of resistance and virulence and parasite host-range, which could be included in further extensions of this model. This would be of particular interest in the context of shared resources such as waterholes, which often involve multi-species assemblages.

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References
Bartol, K., 2011. MuMIn: Multi-Model Inference. 1.6.5 ed.