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RESEARCH ARTICLE

Spatial configuration becomes more important with increasing habitat loss: a simulation study of environmentally-transmitted parasites

Tyler R. Bonnell · Ria R. Ghai · Tony L. Goldberg · Raja Sengupta · Colin A. Chapman

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Abstract

Context Landscape changes can be an important modifier of disease. Habitat fragmentation commonly results in reduced connectivity in host populations and increased use of the remaining habitat. For environmentally transmitted parasites, this presents a possible trade-off between transmission potential at the local and global level.

Objectives We quantify the effects of fragmentation on the transmission of an environmentally transmitted parasite, teasing apart the relative effects of habitat composition and configuration on both host movement behaviour and subsequent infection patterns.

Methods We use a spatially-explicit epidemiological model to simulate the effects of habitat fragmentation, using, as an example, whipworm (*Trichuris* sp.) within a red colobus monkey population (*Procolobus rufomitratus*).

Results We found that habitat fragmentation did not always lead to a trade-off between population connectivity and concentration of habitat use in host movement behaviour or in final population infection patterns. However, our simulation results suggest the spatial configuration of the remaining habitat became

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T. R. Bonnell (✉)
Department of Psychology, University of Lethbridge,
4401 University Drive, Lethbridge T1K 3M4, Canada
e-mail: tyler.bonnell@uleth.ca;
tyler.bonnell@mail.mcgill.ca

R. R. Ghai
Department of Environmental Sciences, Emory
University, 400 Dowman Drive, Atlanta, GA 30322, USA

T. L. Goldberg
Department of Pathobiological Sciences and School of
Veterinary Medicine, University of Wisconsin-Madison,
Madison, USA

R. Sengupta
Department of Geography, McGill University, Montreal,
Canada

C. A. Chapman
Department of Anthropology and McGill School of
Environment, McGill University, Montreal, Canada

C. A. Chapman
Wildlife Conservation Society, Bronx, NY, USA

C. A. Chapman
Section of Social Systems Evolution, Primate Research
Institute, Kyoto University, Kyoto, Japan

increasingly influential on behavioural and infection outcomes as habitat was removed. Additionally, we found common fragmentation metrics provided little ability to explain variation in propagation of infections.

Conclusions Our results suggest an interaction between habitat configuration and composition should be considered when assessing disease related impacts of habitat fragmentation on environmentally transmitted parasites, especially in cases where habitat loss is high ($\geq$ 30%). We also propose that spatially-explicit simulations that capture a host's response to fragmentation could aid in the development of novel landscape metrics targeted towards specific host-parasite-landscape systems.

Keywords Habitat fragmentation · Infectious disease · Spatially-explicit epidemiological model · Red colobus · Kibale National Park · Landscape connectivity · Host movement

Introduction

Habitat fragmentation is a globally pervasive anthropogenic landscape alteration for which ecological implications have been the subject of extensive research (Debinski and Holt 2000; Ewers and Didham 2006; Smith et al. 2009; Wilson et al. 2016). Habitat fragmentation results in a reduction in the extent of suitable habitat (i.e., changes in landscape composition) and an increase in isolation of the remaining habitat patches (i.e., changes in landscape configuration). Changes to the composition and configuration of landscapes can alter host-parasite dynamics in substantial ways, and have been shown to play an important role in disease systems, including Lyme disease (Allan et al. 2003; Brownstein et al. 2005), chronic wasting disease (Farnsworth et al. 2005), hantavirus pulmonary syndrome (Langlois et al. 2001), and Ebola (Rulli et al. 2017) to name just a few. The exact outcomes of specific landscape changes are thought to vary by the specifics of the host-parasite interaction in question, as well as the landscape in which they are found (Brearley et al. 2013).

In the case of environmentally transmitted parasites (those with a life stage outside the host), movement patterns of the host are an important factor influencing transmission. This is especially the case for relatively immobile parasites which are reliant on potential hosts coming to them (Morgan et al. 2004). The degree to which habitat fragmentation alters animal movement patterns is thought to both facilitate and constrain transmission. At the level of a group, habitat fragmentation can alter the degree to which animals use and re-use areas on the landscape (i.e., concentration of habitat use) (Nunn et al. 2011, 2014; Bonnell et al. 2013b). At the level of the population, habitat fragmentation can reduce connectivity among groups and individuals (Coulon et al. 2004), reducing population mixing. This poses a potential trade-off in terms of transmission rates in fragmented landscapes, between local (i.e., concentration of habitat use) and global (i.e., population mixing) transmission rates. To explore this potential trade-off under alternative fragmentation scenarios, we focus on a specific study system, involving red colobus monkey (*Procolobus rufomitratus*) hosts and a whipworm parasite (*Trichuris sp.*), located in Kibale National Park, Uganda.

Previous studies within this host-parasite system have made comparisons between populations in fragmented and continuous habitat, and have identified differences in health between populations (Chapman et al. 2006, 2013; Goldberg et al. 2012). In terms of parasitism, these studies have focused on environmentally transmitted parasites, identifying characteristics of habitat fragments which predict differences in infection measures. For example, evidence of logging within a fragment was associated with increased strongyle and rhabditoid nematode prevalence in red colobus monkeys (Gillespie and Chapman 2006). Comparisons between species have also highlighted species-specific responses to fragmentation, e.g., prevalence and richness of environmentally-transmitted parasites was found to be elevated for red colobus in fragments, but not for black and white colobus (Gillespie and Chapman 2008). Longitudinal studies, i.e., those monitoring a population over time, have also identified effects of fragmentation under changing host densities, showing an initial pulse in prevalence and intensity of infections due to the immigration of red colobus occupying a nearby fragment (Chapman et al. 2005).

We add to these studies by providing a landscape-level analysis, examining population-level infections as a result of landscape-level characteristics (Wiens

1995). As infection data at this scale are not available, and experimental fragmentation is not feasible, we use a simulation approach that takes advantage of the behavioural data associated with the host species and the mechanistic understanding of the parasite's life-cycle. The ability to combine landscape, host behaviour, and parasitological data are enabled through the combination of geographical information systems and agent-based modeling, which has resulted in the development of spatially-explicit epidemiological models that can target specific study systems (Keeling et al. 2003; Linard et al. 2009; Dion et al. 2011; Lane-deGraaf et al. 2013).

In this paper we simulate the relationship between habitat fragmentation and the spread of the whipworm parasite within a population of red colobus monkeys. Simulation and mathematical models have been common tools for the study of host-parasite interaction under habitat fragmentation (McCallum and Dobson 2002; Morgan et al. 2004; Keeling and Rohani 2008). We use an approach that allows for the explicit description of the movement behaviour of a host and how landscape characteristics influence these movement patterns (Nathan et al. 2008). We monitor the spread of the parasite through the simulated population under nine fragmentation scenarios that vary in habitat composition and configuration. In simulating habitat fragmentation, we are able to create a gradient of fragmentation capturing a wide range of landscape conditions, which is difficult to achieve in natural or designed experiments considering the spatial scale we are concerned with. We then quantify the variation in infection patterns of the whipworm parasite between and within these fragmentation scenarios to gain insight into 1) the effects of landscape composition and configuration and 2) the relative effects of changes in local and global transmission on infection outcomes in the host population.

Methods

We develop a simulation model, incorporating spatial data (remotely sensed satellite imagery), behavioural data (observations of animal movement), and parasitological data (fecal samples screened for the presence of parasites) to make quantitative predictions about the relative effects of changes to the extent of habitat and its spatial configuration on an environmentally transmitted parasite. The model was extended from Bonnell et al. (2016a), which defined movement behaviour of red colobus hosts, a resource landscape, and an environmentally transmitted parasite. We extend this model by simulating habitat fragmentation, and quantify the resulting changes to host behaviour and propagation of the environmentally transmitted parasite (See supplementary material a detailed model description following the ODD protocol, and full model code is available from: https://github.com/tbonne/Landscape_Host_Parasite).

Model details

The resource surface used in the model was estimated using satellite remote sensing and ground transects of red colobus food trees, resulting in 30 × 30 m cells covering a surface of approximately 2 × 2 km, where each cell's value is an estimate of the total diameter of breast height of potential food trees (Bonnell et al. 2016a). We use this spatial estimate of food trees as a representation of the heterogeneity in food availability. When foraged on by red colobus agents, resource cells were depleted and grew back at a set rate. Regrowth, as well as red colobus energetic parameters, were estimated by comparing simulated movement patterns to observed movement patterns of red colobus groups (Bonnell et al. 2013a).

Red colobus were added to the simulated landscape, based on the estimated density (176–219 individuals/km^2; Struhsaker 2010), and group size variation (mean size = 47, sd = 4; Gogarten et al. 2014) observed at Kibale National Park, Uganda. Movement of red colobus was defined by a trade-off between feeding and safety. Individual red colobus adjusted their desired safety, measured as the number of nearby group members, based on food intake, e.g., when food intake was high the number of desired neighbours increased, while when food intake was low the number of desired neighbours decreased. When an individual was not safe it moved towards a pre-specified group leader. The movement behaviour of hosts can be seen as adaptive, as individuals vary the extent to which their movement is socially or ecologically driven. Given the match with observed data in Bonnell et al. (2013a, b), a leader-led social structure with spatial memory was imposed on red colobus groups. To this movement model, group leaders were given additional safety requirements, making safety dependent on being within a sleeping site during the last 4 h of the day. This rule allowed for the inclusion of sleeping site behaviour in the model, i.e., re-using of common sleeping sites, a potentially important behaviour determining host habitat use. Similarly, to incorporate dispersal between groups, individuals, other than the group leader, where given a probability of dispersal. If an individual chose to disperse it simply changed its number of desired neighbours to zero until it encountered a new group, at which point it resumed balancing safety and foraging competition within its new group.

A parasite was then introduced to the model, which followed a fecal–oral transmission route, approximating the life-cycle of a whipworm. Starting as an egg in the environment, the simulated parasite passed through a latency period (1), before becoming infective (2). If ingested by a red colobus it then became a developing larvae (3), and finally an adult parasite in the gut of the red colobus host (4), passing eggs back into the environment until a life-expectancy was reached and the adult parasite was removed from the simulation. Life-expectancy for eggs deposited in the environment was set for each deposit based on a negative binomial distribution. The negative binomial distribution allows for over-dispersion in the life-expectancy of eggs in the environment, allowing most eggs to perish relatively quickly and a few to remain viable for extended periods of time (e.g., few days to many months). Hosts foraging in contaminated cells had a set probability of ingesting an infectious egg for each infectious deposit in the cell. If the host was already infected and a transmission event occurred, the host's intensity of infection was increased by one. This intensity measure was used as a surrogate measure of infection intensity, and had no influence on infection life cycle (i.e., life-expectancy of the infection was based solely on the initial infection time). Host, resource, and parasite life cycle parameters were set by the best fit model parameters that produced comparable host prevalence patterns to those observed at our study site (Bonnell et al. 2016a). At the start of a simulation, ten randomly chosen individuals were infected, and after 5 years the average prevalence (#infected/total population size) and intensity (mean infection count from infected individuals) within the population were recorded.

Our focus in these simulations is on the spread of a non-virulent parasite, i.e., infection does not result in increased mortality. Given the empirical evidence suggesting that in well-fed individuals *Trichuris* infections can be considered as non-lethal, though infected individuals can show sickness behaviour (Ghai et al. 2015), we restrict our analysis to conditions in which the simulated agents were able to meet their energetic demands (see "Habitat fragmentation" section below). Our simulations also do not include a birth or death process, and as such our results only apply under conditions of a stable population size with low turnover. Extensions to these model assumptions could investigate how fluctuations in population size or demographics in response to fragmentation could influence spread of environmentally transmitted parasites. These assumptions should be kept in mind when interpreting the results.

Habitat fragmentation

To simulate habitat fragmentation, we used a simple fragmentation algorithm with two input parameters: total habitat to remove (H_{loss}) and the size of patches to remove (P_{size}). The algorithm proceeded as follows: (1) select a random cell on the landscape, (2) remove forest from that cell and neighbouring cells until a patch size of P_{size} is reached, and (3) return to step one. This algorithm was stopped once H_{loss} was reached. We simulate low (10%), medium (20%), and high (30%) habitat loss scenarios (H_{loss}; Fig. 1). We chose a maximum habitat reduction of 30%, as simulated hosts were not able to adjust their foraging behaviour (e.g., larger home ranges, travel more per day, larger group spread) to meet energetic demands above this value. As we did not include nutritional effects or flexibility in other potential response variables to cope with this situation, such as increased susceptibility (Gulland 1992; Chapman et al. 2015), we limited the maximum habitat removal to constrain our experiments to parameter ranges within which energetic demands could be met by the simulated red colobus agents. We simulated changes in habitat configuration by altering patch sizes from small (60 m), intermediate (180 m), and large (300 m). Using this approach we generated landscapes varying in habitat extent and spatial configuration along a gradient from many small patches removed, to few large ones (Fig. 1). The above fragmentation algorithm is able to generate landscapes of various configurations through random removal of patches. In the future, more detailed

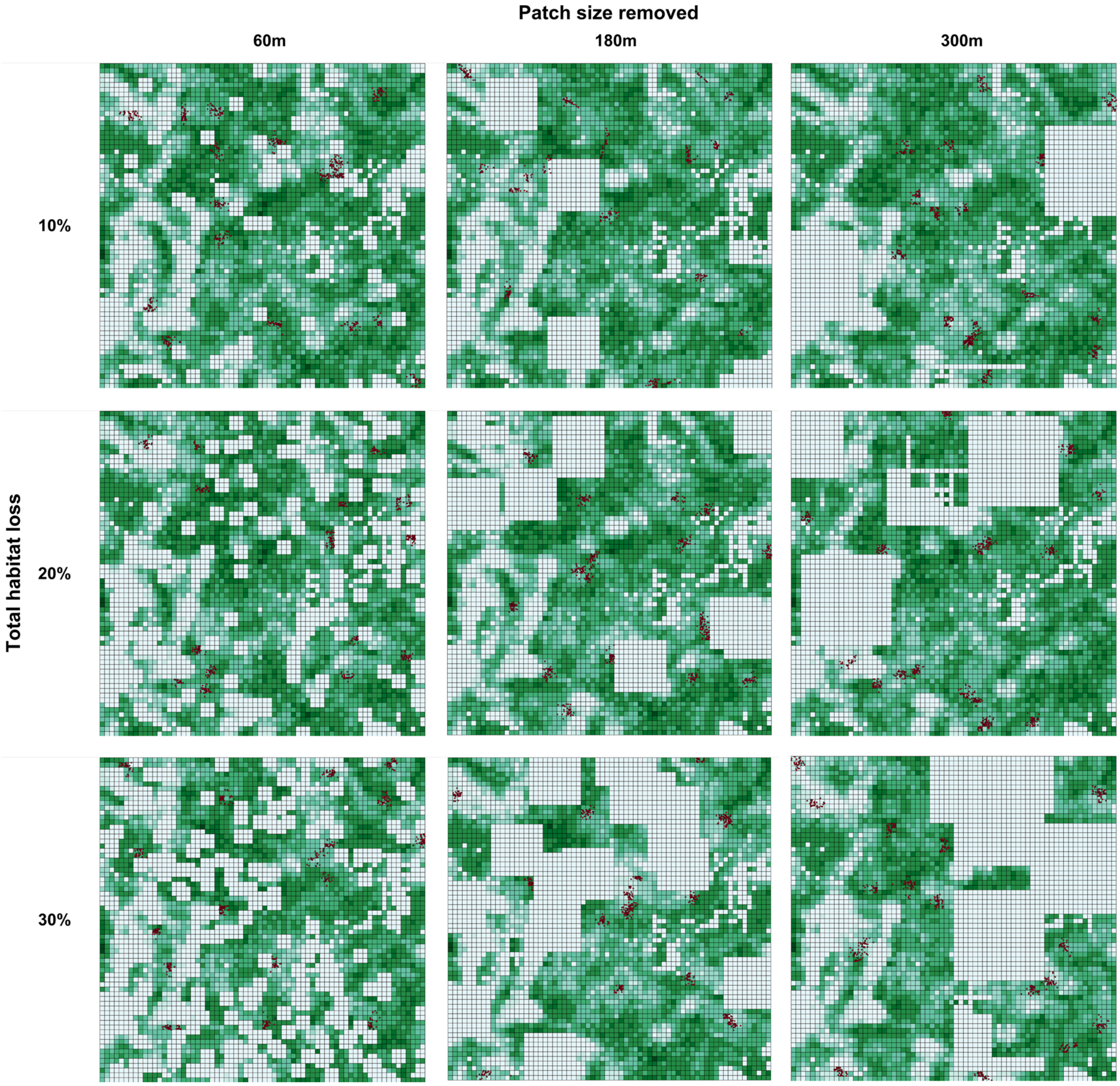

Fig. 1 Graphical display of landscape fragmentation scenarios. Grid cells are coloured based on resource availability (light green to dark green). Red colobus are represented as points. Cleared patches are visible as squares of light grey, indicating no resources present

approaches which tailor forest removal to known forest harvesting patterns could be of use for investigating the implications of harvesting practices on transmission dynamics.

We ran the model 150 times for each combination of landscape alterations (n = 1350), and measured infection outcomes (i.e., prevalence and intensity of infections) as well as host behaviour for each run. For the 1350 landscape alterations, each defined by the extent of habitat loss and patch size, a novel landscape was produced. In recording host behaviour, we focus on behaviours related to population connectivity and concentration of habitat use. Host population connectivity was measured as the average home range size of groups per month (B_{hr}), where home range size was calculated as the number of cells visited multiplied by the areas of cells (30 × 30 m). The final home range size measure used for any given simulation was then an average of these home ranges across months. The concentration of habitat use by hosts was measured as the average of visit counts to all cells visited at least once (B_{visits}).

To measure landscape connectivity under fragmentation, we make use of the connectance index (McGarigal et al. 2002), as well as cell betweenness (Estrada and Bodin 2008; Kupfer 2012). The connectance index compares the total sum of neighbouring forest cells at each cell to the total possible neighbours; cell betweenness considers each cell containing forest as a node within a network and calculates the frequency of shortest paths passing through each node, based on the shortest paths from every pair of nodes in the network. The connectance index and average cell betweenness provides a landscape level measure of connectivity, while cell betweenness provides a local measure of connectivity. We assess the use of these metrics to infer infection prevalence and intensity outcomes.

Analysis

The simulations had four outputs: average home range of groups (B_{hr}), average grid cell visit count (B_{visits}), final average intensity of infections (I_{int}) and final prevalence of infection (I_{prev}) in the population. To gain an overall view of the variability in outcomes across fragmentation scenarios, we first compared host behaviour and infection outcomes across simulations. We ran a one-way Anova using fragmentation scenario as a factor, including a scenario with no fragmentation. We compared differences in means between all scenarios, controlling for multiple comparisons using the multcomp package (Hothorn et al. 2008) in R (R Core Team 2016). To explain differences in host behaviour and infection outcomes across scenarios we made use of linear models: (1) observed host behaviours (B_{hr}, and B_{visits}) across scenarios were predicted using fragmentation parameters (H_{loss}, P_{size}, and $H_{loss} \times P_{size}$), and (2) Infection outcomes (I_{prev} and I_{int}) were predicted using fragmentation parameters (H_{loss}, P_{size}, and $H_{loss} \times P_{size}$) and host behaviour (B_{hr}, and B_{visits}). These models predicted changes across fragmentation scenarios, and therefore assessed differences in the general characteristics of a fragmentation scenario. To assess the effects within scenarios, we used linear mixed-models grouping simulation runs by scenario. We ran these models with random intercepts and slopes to test for differences in response to host behaviour (B_{hr} and B_{visits}) on infection outcomes within particular fragmentation scenarios. This within-scenario approach allowed us to quantify the effect of landscape configuration, as the only factor that changes within a scenario is the random removal of patches and hence the configuration of remaining habitat. Finally, to directly quantify the trade-off between local and global host habitat use, we used mixed models to estimate the relationship between home range size and average visit counts, as well as the relationship between population prevalence and intensity within each scenario.

To quantify the ability of landscape metrics to predict infection outcomes within fragmentation scenarios we ran a mixed model for each metric predicting infection prevalence and intensity, using fragmentation scenario as a random effect. To assess the use of the local metric we quantified the correlation between a cell's betweenness value and its contamination (i.e., the number of *Trichuris* eggs within a cell) at the end of the simulation. We then predicted the strength of this correlation across simulations using fragmentation values (H_{loss}, P_{size}, and $H_{loss} \times P_{size}$) and host behaviour (B_{hr}, and B_{visits}).

Results

Variation in outcomes across scenarios

Overall, mean home range size of groups was not sensitive to the specific fragmentation scenario, with the mean home range only differing form the non-fragmented landscape in three scenarios (Fig. 2i). However, average visit count was found to be more sensitive to fragmentation scenario (Fig. 2iii), showing an increase in visit counts within increased patch sizes and extent of habitat removed. In the case of infection outcomes, we find that both the intensity and prevalence of infections increased from the non-fragmentation scenario under scenarios with higher habitat losses (Fig. 2 ii,iv).

Landscape: host behaviour

Both the extent of habitat loss and the sizes of patches removed were influential in explaining differences in average visit counts of hosts. Average visit count was best predicted by the extent of habitat removed ($\beta = 0.71$, $se = 0.01$), the size of patches removed

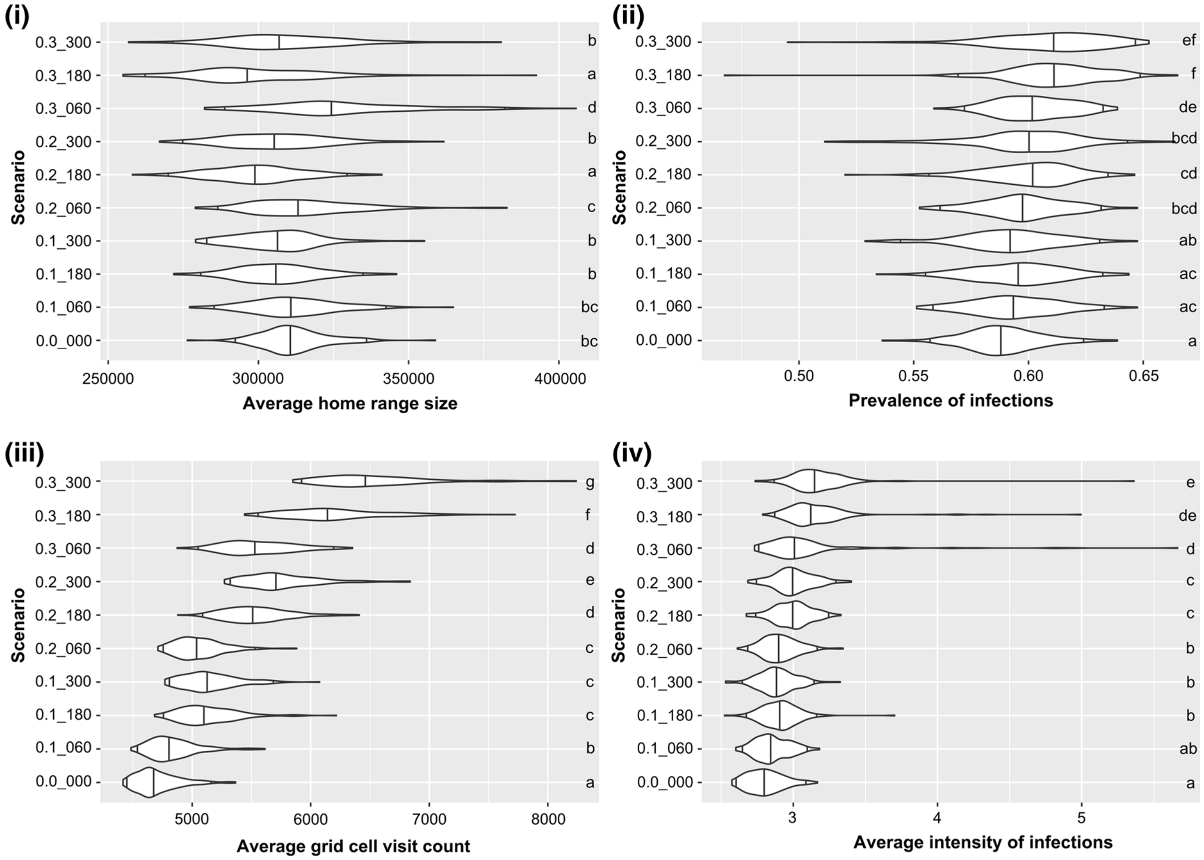

Fig. 2 Violin plots of the simulation outcomes for host behaviour and final infection for each fragmentation scenario. Scenarios, on the y-axis, are presented as the percentage of habitat loss followed by the size of the patches removed (e.g., habitat loss = 10%, patch size removed = 60 m, results in scenario = 0.1_060). Compact letter displays (a,b,c… etc.) are used to separate scenarios based on all pairwise comparisons. Lines within the violin plots represent the 0.025, 0.5, and 0.975 quantiles respectively

(β = 0.44, se = 0.01), and an interaction between total habitat removed and the size of the patches (β = 0.18, se = 0.01) (Table 1). Indeed, as habitat was lost, the effect of the size of patches removed increased, with visit counts increasing as patch sizes increased (Fig. 3i). Population wide mixing, measured as average home range size of groups, was poorly predicted by habitat fragmentation parameters ($r^2 = 0.08$), showing a negative effect for patch size (β = − 0.24, se = 0.03), a small positive effect of the extent of habitat removed (β = 0.08, se = 0.03), and an interaction between patch size and habitat loss (β = 0.11, se = 0.03) (Table 1). This interaction suggested that increased habitat loss and small patch sizes

Table 1 Parameter estimates of linear models explaining host behaviour using characteristics of habitat fragmentation

Landscape fragmentation	Visit count β (se)	Home range size β (se)
Patch size	0.44 (0.01)	− 0.24 (0.03)
Habitat loss	0.71 (0.01)	0.08 (0.03)
Patch size × habitat loss	− 0 .18 (0.01)	− 0.11 (0.03)
	$R^2 = 0.73$	$R^2 = 0.08$

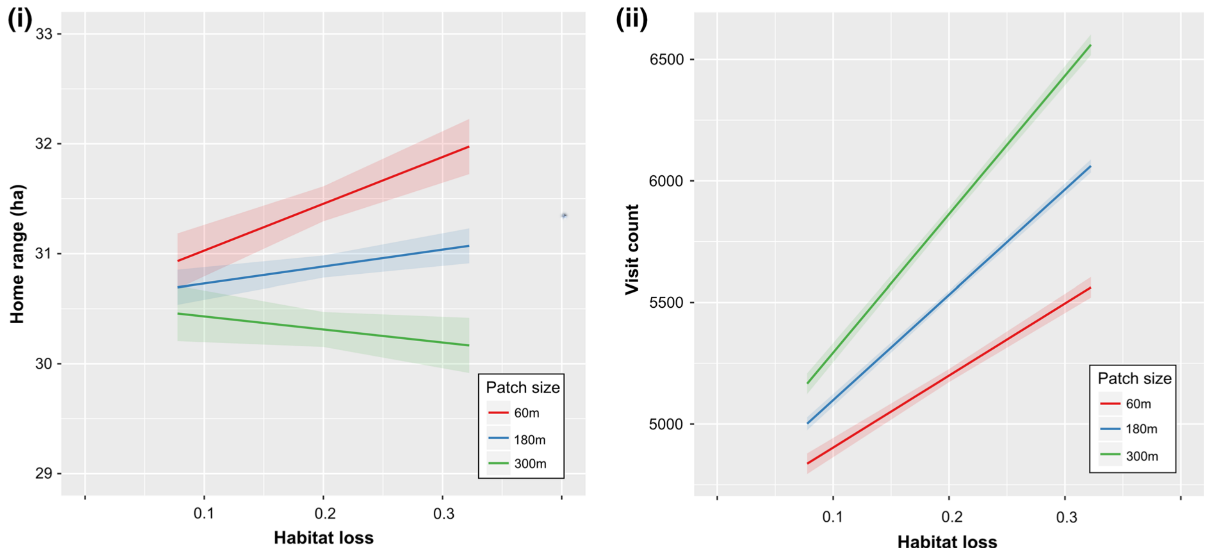

Fig. 3 Interaction between patch size removed and habitat loss for host home range (**i**) and concentration of habitat use (**ii**). Shaded areas are the 95% confidence interval

resulted in an increase in home range size, whereas an increase in habitat loss and large patch sizes resulted a decrease in home range size (Fig. 3ii).

Landscape—host behaviour: parasite transmission

Final infection prevalence across fragmentation scenarios was largely determined by a positive effect for the intensity of host habitat use (i.e., visit count), with smaller negative effects for patch size ($F_{5,1344} = 27.85$, p value < 0.001, adj $r^2 = 0.09$) (Fig. 4i, Table 2). In the case of intensity of infections, variation in average visit count had the highest influence, followed by the size of patches removed and home range size ($F_{5,1344} = 135.9$, p-value $<$ 0.001, adj $r^2 = 0.33$) (Fig. 4ii, Table 2). Of the two infection outcomes, intensity showed a more predictable response to changes in the habitat compared to prevalence (i.e., adj $r^2 = 0.09$ for prevalence, and adj $r^2 = 0.33$ for intensity).

Effects of landscape configuration within fragmentation scenarios

The model explaining differences in prevalence within scenarios identified changes in visit count as an important predictor (Table 3). In this model there was little variance attributed to random intercepts or slopes for home range size or visit counts (4%) (Fig. S1). This result suggests that, on average, a similar relationships between host behaviour and infection outcomes exists across all scenarios, although this effect may be reduced in the high habitat loss scenario with large patches removed (Fig. S1). The model explaining variation in intensity of infections within scenarios suggests that average visit counts led to higher intensity of infections, with a small positive effect of home range size (Table 3). Investigating the random effect structure identifies only small changes in relationships across scenarios (2%) (Fig. S1). There is some evidence that under high habitat loss (i.e., $H_{loss} = 0.3$) configurations which increase home range size have an increasingly positive effect on intensity of infections when small patches were removed (Fig. S1), whereas the effect of visit count on infection intensity shows almost no variation across scenarios.

Trade-off between global and local habitat use

When comparing the relationship between home range size and average visit count within scenarios, we found a positive relationship between average visit count and average home range size (Fig. 5i, Table S1), This was greatest in scenarios with high and medium habitat losses with small patches removed (i.e., 60 m patches). When comparing the relationship between infection intensity and prevalence, we found an overall

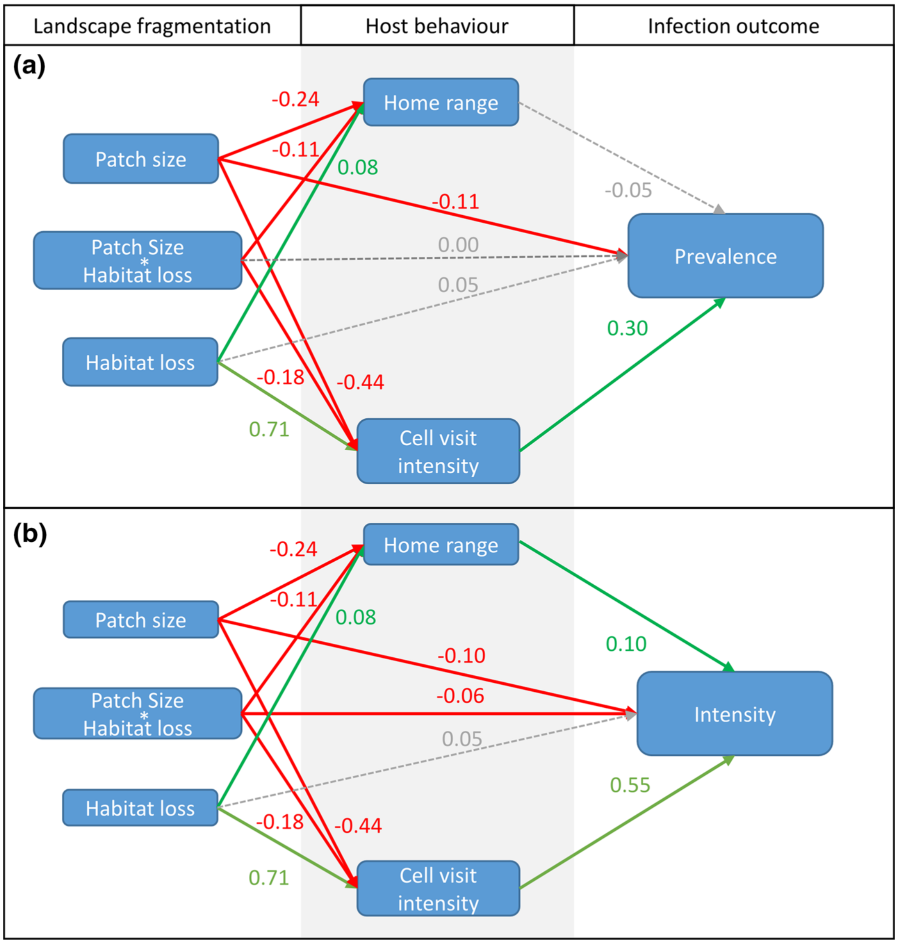

Fig. 4 Visual diagram displaying the influence of landscape fragmentation parameters and subsequent host behaviour on infection outcomes. Standardized coefficients are presented to aid in interpreting relative effects of each parameter on infection outcomes. Estimated coefficients that were found to contain 0 within their 95% CI were assigned dashed lines

Table 2 Model results predicting infection prevalence and intensity based on characteristics of habitat fragmentation and host behaviours

Predictor	Infection prevalence β (se)	Infection intensity β (se)
Habitat loss	− 0.05 (0.05)	− 0.05 (0.04)
Patch size	− 0.11 (0.04)	− 0.10 (0.03)
Habitat loss × patch size	− 0.00 (0.03)	− 0.06 (0.03)
Home range	− 0.05 (0.03)	0.10 (0.03)
Visit count	0.30 (0.07)	0.55 (0.05)
	$F_{5,1344} = 27.85$, p-value < 0.01, adj $R^2 = 0.09$	$F_{5,1344} = 135.9$, p-value < 0.01, adj $R^2 = 0.33$

positive relationship (Table S1), with habitat configurations leading to increases in intensity being associated with increases in prevalence (Fig. 5ii). However, at higher habitat losses this positive relationship between intensity and prevalence was reduced (Fig. 5ii). Interestingly, the observed changes in relationship between host behaviour does not seem to have an impact on the relationship between infection measures. This is especially visible at high habitat losses (Fig. 5).

Table 3 Model results investigating the fixed effects of host behavioural changes on infection outcomes within fragmentation scenarios

Host behaviour	Infection prevalence β (se)	Infection intensity β (se)
Home range	− 0.01 (0.05)	0.12 (0.04)
Visit count	0.31 (0.09)	0.52 (0.04)
	Marginal $R^2 = 0.08$	Marginal $R^2 = 0.31$
	Conditional $R^2 = 0.11$	Conditional $R^2 = 0.34$

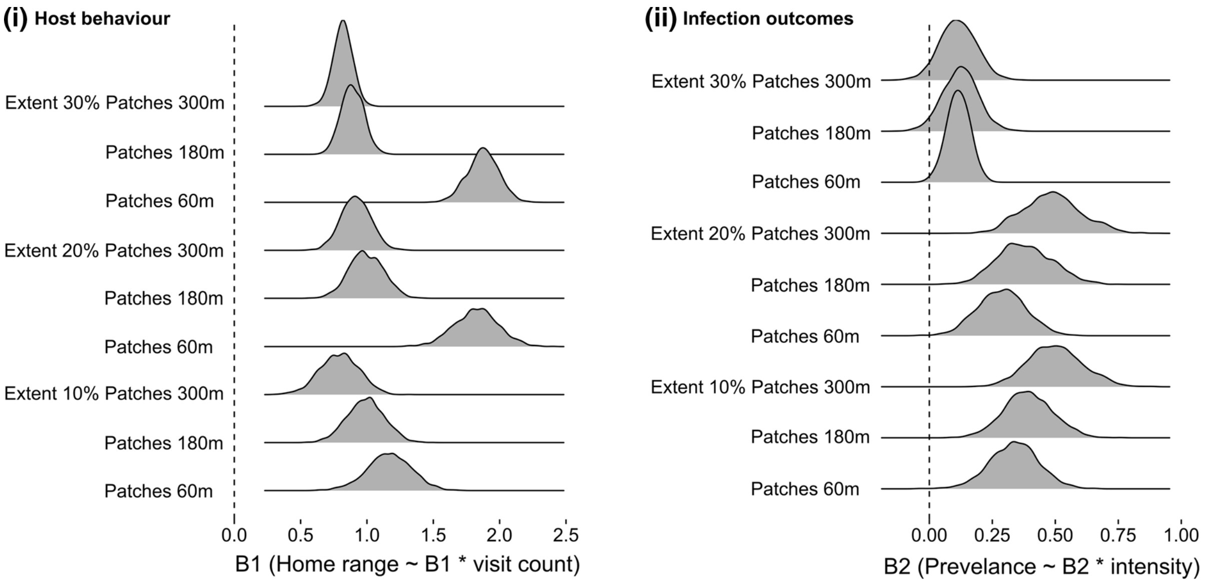

Fig. 5 The estimated relationship between **i** host behaviours (B1), and **ii** infection outcomes (B2) within each simulation scenario. The y-axis depicts the fragmentation scenario, the x-axis depicts the magnitude and sign of the relationship, and the density plots highlight the probability density of each estimated relationship, i.e., B1 and B2

Landscape measures as predictors of disease outcomes

Simulated fragmentation resulted in a range of landscape connectance measures (mean = 58.06, min = 44.36, max = 69.15). The ability of the landscape connectance measure to predict resulting infection outcomes within scenarios was slightly more successful with infection intensity, compared to prevalence (Table 4). The measure of landscape connectance was very well explained by fragmentation parameters ($\beta_{habitatLoss} = -0.96$, $\beta_{patchSize} = 0.24$, $\beta_{habitatLoss \times patchSize} = 0.08$, $F_{3,1346} = 2.16e^4$, p-value < 0.001, adj $r^2 = 0.98$) and was largely a function of habitat loss. This suggests that the measure of connectance was not influenced by habitat configuration in the range of habitats used. Using average betweenness of the landscape explained slightly less of the variation in infection outcomes compared to that of landscape connectance (Table 4). Fragmentation parameters explained a large portion of variation in average betweenness across landscapes ($\beta_{habitatLoss} = -0.91$, $\beta_{patchSize} = 0.03$, $\beta_{habitatLoss \times patchSize} = -0.01$, $F_{3,1346} = 2271$, p-value < 0.001, adj $r^2 = 0.83$), again suggesting that the measure largely captured habitat loss and was not very sensitive to habitat configuration.

Average betweenness is an aggregate measure of individual grid cells; thus it is possible to relate an individual grid cells' betweenness value to its contamination with deposited parasite at the end of the simulation. This local landscape metric produced low

Table 4 Performance of landscape metrics: connectance and average betweenness, on predicting infection outcomes of simulations

Landscape metric	Infection prevalence β (se)	Infection intensity β (se)
Connectance	− 0.26 (0.08) Marginal $R^2 = 0.06$, Conditional $R^2 = 0.10$	− 0.43 (0.14) Marginal $R^2 = 0.17$, Conditional $R^2 = 0.29$
Average betweenness	− 0.22 (0.06) Marginal $R^2 = 0.05$, Conditional $R^2 = 0.07$	− 0.16 (0.06) Marginal $R^2 = 0.02$, Conditional $R^2 = 0.20$

to moderate correlations (mean = 0.25, min = 0.03, max = 0.47), in that cells with higher betweenness values (i.e., acted as links between distant cells) had higher parasite contamination. When the variation in these correlations was explained using fragmentation parameters and host behaviour, we found that habitat loss and average home range size of groups was positively related to correlation strength ($\beta_{habitatLoss} = 0.37$, $\beta_{homeRange} = 0.22$) and that average visit count was negatively related to correlation strength ($\beta_{visitCount} = -0.28$) (Table S2).

Discussion

Habitat fragmentation typically reduces movement at the global level and increases concentration in local movements, which creates a potentially important trade-off for environmentally transmitted parasites. We used a simulation approach to explore the implications of habitat fragmentation on host response to habitat alterations and parasite transmission characteristics. Specifically, we generated fragmentation scenarios that systematically vary both alterations to habitat composition and configuration, and we recorded both host behaviour and infection as outcomes.

We found that both infection prevalence and intensity were positively affected by changes in host's concentration of habitat use (Fig. 4a, b). Although the intensity of infections showed some sensitivity to visit count (B_{visit}) and home range size (B_{hr}), both were positive effects (Fig. 4b). When we quantified the relationship between visit count and home range size, we found a positive relationship, where habitat configurations that produced higher concentrations of habitat use also showed increased population mixing (Fig. 5i). This result suggests that simulated hosts reacted to fragmentation by visiting more sites, and by revisiting these sites more often. This result, in turn, was sensitive to the specific patterns of removal, e.g., when many small patches were removed the magnitude of this positive effect greatly increased (Fig. 5i). Similarly, our results suggest that, within scenarios, configurations that increased intensity of infections also increased prevalence of infections (Fig. 5ii). As prevalence did not decrease in landscape resulting in higher visit counts, and similarly, landscapes resulting in higher home ranges did not decrease infection intensity, these results suggest no net trade-off between local and global transmission. Rather, our simulated hosts responded by increasing both local and global transmission possibilities resulting in increased population level intensity and prevalence of infections.

Interestingly, our simulation showed that host behaviour became more dependent on habitat configuration with greater habitat loss (Fig. 3). Given that both infection intensity and prevalence were largely driven by visit count, and to a lesser extent home range behaviour, our results suggest that infection outcomes are similarly more sensitive to configuration in higher habitat loss scenarios (Fig. 4). We did not initially predict that habitat configuration would covary with habitat removal in this manner, yet this is a logical outcome of the assumptions regarding host behaviour and parasite characteristics: the less a landscape consists of habitat types that provide resources or facilitates movement, the more configuration of remaining habitat patches will affect movement and grouping patterns of hosts, and hence parasite transmission patterns. Such an effect of configuration due to increased habitat loss has been suggested in population survival analysis (Fahrig 1998; Flather and Bevers 2002). Interestingly, the effect of habitat configuration on population dynamics is thought to be highly sensitive to the specific biology and habitat use of the species in question, as well as the details of the landscape in question (Wiegand et al. 2005). This conclusion limits the ability to assign a simple positive/negative outcome for habitat configuration, and suggests caution when assessing the effects of habitat composition and configuration when both population dynamics and parasite transmission are included. Future work investigating the effects of habitat fragmentation on parasite transmission should examine how transmission varies with increasing habitat loss beyond the parameter spaces we have tested ($H_{loss} > 30\%$). Because our model of host behaviour failed to reproduce host patterns above 30% habitat loss, a more nuanced description of host behaviour in response to habitat loss might be required. For example, a more detailed behavioural model of the host would likely require incorporating nutrition, immunology, and mortality related to infection status (Chapman et al. 2007, 2015).

Our model of host movement did not include any strong effects on movement through non-forested habitat, i.e., matrix habitat. Alternative parameterization of a host movement model, e.g., restricting movement through non-forested habitat, would likely result in stronger effects of habitat fragmentation on host population mixing and concentration of habitat use (Fig. 2). In a more generalized simulation of environmentally-transmitted parasites, Nunn et al. (2011) found that increasing habitat use constraints within groups and reducing range overlap between groups did not result in a reduced population level prevalence, but rather led to an increase in overall prevalence. The authors suggest that the increased reuse of smaller core areas resulted in higher within-group transmission, and that between-group infections required only small chance events to colonize non-infected groups. It is thus likely that more restrictive assumptions about the barrier posed by matrix habitat, the more configuration will matter, as it would increasingly constrain movement to habitat corridors, increasing overall concentration of habitat use (Stoner 1996). Overall, our model likely presents a conservative estimate of the importance of habitat configuration on transmission of environmentally-transmitted parasites.

When landscape fragmentation measures were used to predict infection outcomes in the host population, we found only limited predictive capacity. The measure of landscape connectance explained only 6% of the variance in prevalence and 17% in the case of intensity. Average betweenness of the landscape was found to explain even less (5 and 2% respectively). Using a local level measure, the betweenness of cells was found to be low-to-moderately correlated to the contamination of parasites within that cell. This correlation of cell betweenness and contamination increased in scenarios which had higher overall loss and configurations which lead to larger home range sizes of groups, and decreased with higher concentrations of habitat use. This finding suggests that this local measure might become more important as habitat is further removed and populations continue mixing through connected patches. In our analyses we tested only two landscape measures (connectance and average betweeness) and one local measure (cell betweenness). Further work could identify spatial metrics which optimally predict disease outcomes. Indeed, simulation models incorporating details about landscape structure, host behaviour, and disease characteristics could be used to tailor metrics for specific host-parasite-landscape systems, accounting for the mechanism by which landscape alteration influence specific host movement and grouping behavior (Bélisle 2005). Similarly, given that most hosts are infected by multiple parasites, and given that many parasites can infect more than one host species, the inclusion of multiple hosts and parasites could be highly informative (Morgan et al. 2004; Telfer et al. 2010), especially, where hosts show differing behavioural responses to habitat fragmentation and where parasites survival outside the hosts vary in the degree to which they are affected by micro-climate (Morgan et al. 2004;Bonnell et al. 2016a; White et al. 2017).

The strength of our inferences depend strongly on how generalizable our simulated behaviours are to hosts reacting to landscape changes. Our simulations used a movement model based on scramble competition for food within a group, calibrated by group level movement patterns of observed groups. However, it is becoming increasingly possible to map movement at the individual level, and thus to test behavioural models of social and ecological influences on grouping patterns (Sarkar et al. 2014; Kays et al. 2015; Strandburg-Peshkin et al. 2015; Bonnell et al. 2016b; Rivest et al. 2016). Refinement of behaviour models using such methods could lead to improved estimates of how landscape changes will influence movement and grouping patterns of hosts. Similarly, the ability to map landscape change has increased dramatically with new sensor technologies that capture diverse spectral and spatial data (e.g., Landsat, MODIS, Aster, Spot, Sentinel). The incorporation of landscape and behavioural data into simulations has great potential, both in developing theory related to the disease related impacts of landscape changes and in providing site specific predictions (Evans et al. 2013).

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