Environmentally transmitted parasites: host-jumping in a heterogeneous environment

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Abstract. Groups of chronically infected reservoir-hosts contaminate resource patches by shedding a parasite's free-living stage. Novel-host groups visit the same patches, where they are exposed to infection. We treat arrival at patches, levels of parasite deposition, and infection of the novel host as stochastic processes, and explicitly derive the expected time elapsing until a host-jump (initial infection of a novel host) occurs. At stationarity, mean parasite densities are independent of reservoir-host group size. But within-patch parasite-density variances increase with reservoir group size. The probability of infecting a novel host declines with parasite-density variance; consequently larger reservoir groups extend the mean waiting time for host-jumping. Larger novel-host groups increase the probability of a host-jump during any single patch visit, but also reduce the total number of visits per unit time. Interaction of these effects implies that the waiting time for the first infection increases with novel-host group size. If the reservoir-host uses resource patches in any non-uniform manner, reduced spatial overlap between host species increases the waiting time for host-jumping.

1 Introduction

Many ecologically and epidemiologically important pathogens are transmitted through the abiotic environment [Miller et al. 2006, Breban et al. 2009, Cizauskas et al. 2014a]. Analyses of environmentally transmitted parasites emphasize how the dynamics differs from infections spread through direct contact between hosts [Bani-Yaghoub et al. 2012, Garira et al. 2014]; these distinctions have significant implications for epidemiological invasion [Rohani et al. 2009, Breban et al. 2010] and for the likelihood a parasite can "jump" to a novel host species [Woolhouse et al. 2005, Caraco et al. 2014].

Our study of environmental transmission focuses on host-jumping, the initial infection of a novel-host population or species. Our assumptions best match intestinal parasites of humans, non-human primates and gregarious herbivores, ordinarily transmitted via the fecal-environment-oral route [Hutchings et al. 2001, Cizauskas et al. 2014b]. But other macroparasites, as well as certain bacteria and viruses, use this mode of transmission. Therefore, our model's predictions should apply broadly to group-living hosts. We envision a reservoir population and a novel-host population inhabiting the same environment. Chronically infected reservoir individuals shed the parasite at resource patches, exposing the novel host to infection. We ask how each species' social group size affects the random waiting time until the parasite first jumps to the novel host.

2 Background

Climate change and increased species-introductions have driven biogeographic range expansion of many parasites [Crowl et al. 2008, Jolles et al. 2008]. Ecological disturbance,

especially human landscape alteration, has placed new host-parasite combinations in common environments [Patz et al. 2000]. These processes increase opportunities for parasites to jump to novel hosts [Cooper et al. 2012].

We motivate our general analysis by citing Hausfater and Meade's [1982] study of yellow baboons (*Papio cynocephalus*) infested by intestinal nematodes. A population of approximately 200 individuals was partitioned into groups; individuals associated consistently with the same group. Each evening a group would enter a spatially distinct cluster of yellow-barked acacia (*Acacia xanthophloea*). Group members would rest in the trees, safe from predators, for about 10 hours, and then spend time socializing/foraging beneath the trees.

After one or two nights at a sleeping site, the group would move to another location, leaving a substantial fecal accumulation beneath the trees. Soil from the sleeping area yielded ova and larvae of nematodes at densities exceeding those in samples from outside sleeping sites [Hausfater and Meade 1982]. Intestinal nematodes of baboons are transmitted environmentally [Olsen 1974], and the same acacia clusters used by yellow baboons provided feeding/resting sites to other mammals, including vervet monkeys (*Cercopithecus aethiops*). Our model addresses host jumping when different species occupy locations where a parasite may be deposited by one host species and acquired by a second.

3 Model

We extend the conceptual framework for environmentally transmitted parasites in two ways. First, we consider host socio-ecology; group size in each population affects the parasite's invasion of a novel host. Second, since host-jumping occurs relatively rarely

Symbols	Definitions
N_1	Number of reservoir (infested) host individuals, Species 1
R	Number of resource patches, indexed by r
G_1	Group size, reservoir species
λ_j	Rate at which each group of Species-j ($j = 1, 2$) arrives at the set of R patches
p_{jr}	Probability Species j uses patch r; $r = 1, 2$
$X_{1r}(t)$	Total visits by Species 1 to patch r on $(0, t)$
$M_r(t)$	Parasite density in patch r at time t, continuous random variable
$f(M_r)$	Stationary probability density of M_r
m	Randomly increment to parasite density; mean = $G_1\ \mu$, variance = $G_1\ \sigma^2$
ξ	Decay rate of free-living parasite density
t_c	Characteristic time for parasite mortality dynamics
N_2	Number of novel host individuals, Species 2
G_2	Group size, novel host
γ	Susceptibility parameter, novel host
$\zeta_r(\tau)$	Probability novel host has not acquired parasite in patch r by time τ
T	Random waiting time until parasite first jumps to novel host

Table 1: Definitions of model symbols

[Woolhouse et al. 2005], we treat deposition of the parasite by the reservoir species, and acquisition of the parasite by the reservoir host, as stochastic processes. Combining these processes leads to an analytic expression for the mean waiting time until the parasite jumps to the novel host.

We dichotomize model development. To begin, we consider how the reservoir species generates densities of the parasite's infectious stage across resource patches. We then address the novel host's use of the same patches and consequent exposure to the parasite. We fix population sizes of both species, to focus links between group size and parasite densities. Table 1 collects model symbols.

3.1 Reservoir-host and parasite densities

We index the reservoir host as Species 1. The reservoir population contains a total of N_1 individuals that chronically shed the parasite's infectious stage. We treat N_1 as an endemic

equilibrium, a constant. The N_1 reservoir hosts are distributed uniformly across groups of size G_1, where $G_1 \in \{1, 2, ..., N_1\}$. Reservoir group size G_1 can range from solitary to the entire set of parasitized hosts; uniformity of G_1 implies that group size is a species characteristic [Trainor and Caraco 2006]. For convenience we treat N_1/G_1 as an integer, and ignore Species-1 individuals free of parasitism.

3.1.1 Reservoir patch use

The environment includes a set of R discrete resource patches. For clarity and simplicity, we set $R = 2$, but generalization is straightforward. A patch might offer food, drinking water, refuge from predators, or a place to rest.

Infested hosts shed the parasite during patch visits. Each reservoir-host group visits the set of R patches as an independent Poisson process, with the same constant probabilistic rate λ_1. Since there are N_1/G_1 such groups, the reservoir population as a whole visits the set of patches at combined probabilistic rate $\lambda_1 N_1/G_1$. Visits are sufficiently brief, relative to the time between visits, that we ignore the possibility of simultaneous patch occupation. λ_1 does not depend on group size. This means that each individual in the reservoir-host population enters a resource patch at the same average rate whether groups contain g_1 or $2g_1$ members. But in the latter case the population is structured into half as many groups, so that the population-scale rate $\lambda_1 N_1/G_1$ is halved. We do not anticipate that frequencies of resting, sleeping and drinking (and perhaps feeding) will depend on group size.

Each reservoir-host group has the same resource preferences, defined by the probability distribution of visits across the R patches. p_{1r} represents the probability that any given visit by the reservoir species occurs at patch r; $r = 1, 2$. $X_{1r}(t)$ counts the cumulative number of visits by reservoir-host groups to patch r by time t; $X_{1r}(t = 0) = 0$. Then each $X_{1r}(t)$ is an

independent Poisson variable [Ross 1983] with expectation $\mathrm{E}[X_{1r}(t)] = p_{1r}\lambda_1 N_1 t/G_1$.

3.1.2 Parasite densities

The continuous random variable $M_r(t)$ represents parasite density for patch $r = 1, 2$; $M_r(t = 0) = 0$. $M_r(t)$ increases when a reservoir-host group enters patch r and sheds the parasite. $M_r(t)$ decreases between visits by the reservoir species due to parasite mortality. $M_r(t)$ depends only on the dynamics of shedding and decay in patch r; parasite density does not depend on patch area or explicit location. We treat the parasite as infectious when shed by a reservoir host. This assumption holds for most microparasites, but eggs/propagules of macroparasites may require 5-15 days before producing infectious larvae.

Suppose that the $i-th$ visit by reservoir-host groups occurs at time t_i; $i \geq 1$. Then at time $t = t_i$, parasite density $M_r(t)$ increases by an amount m_i. Each m_i is a positive, continuous random variable, with both mean and variance increasing with reservoir group size. The mean is $\mathrm{E}[m] = G_1\mu$, and the variance is $\mathrm{V}[m] = G_1\sigma^2$. That is, each reservoir-host group member independently adds to the local density of the parasite‘s infectious stage.

Macro-parasitic burdens *per* host commonly exhibit statistical aggregation; the variance exceeds the mean [Hudson and Dobson 1995, Poulin and Morand 2000]. Aggregation should hold even with zero-counts truncated [Shaw et al. 1998]. Although the rate at which a reservoir host sheds a parasite's infectious stage need not be proportional to that host's burden, we maintain $\sigma^2 \geq \mu$ during model evaluation.

We make biologically reasonable assumptions concerning increments to parasite densities. m_i does not depend on t_i. The m_i are mutually independent, identically distributed, and independent of $X_{1r}(t)$, $M_r(t)$ and r; shedding by the reservoir host is unaffected by the local

parasite density. Finally, we assume that between reservoir-host visits, parasite density decays in a constant proportional manner, as Hausfater and Meade [1982] observed among infectious larvae of yellow baboons' endoparasites. The parasite-mortality rate ξ is the same in each patch. Then the parasite density in patch r at time t satisfies:

$$M_r(t) = \sum_{i=1}^{X_{1r}(t)} m_i e^{-\xi(t-t_i)} \tag{1}$$

Each $M_r(t)$ is an independent Markov shot-noise process [Lemoine and Wenocur 1986, Laio et al. 2001]. In our particular model, parasite density decays continuously, but at Poisson-process intervals the density is suddenly incremented by a reservoir-host group's shedding. Standard approaches obtain the mean and variance of each parasite density from the moment generating function of the shot-noise process [Ross 1983, Lowen and Teich 1990]. Expected values are:

$$\mathrm{E}\left[M_r(t)\right] = \left(p_{1r}/\xi\right) \lambda_1 N_1 \mu (1 - e^{-\xi t}); \quad r = 1, 2 \tag{2}$$

Mean parasite densities do not depend on reservoir-host group size G_1. The parasite-density variances are:

$$V\left[M_r(t)\right] = \left(p_{1r}/2\xi\right) \lambda_1 N_1 \left(\sigma^2 + G_1 \mu^2\right) (1 - e^{-2\xi t}); \quad r = 1, 2 \tag{3}$$

Variance in the number of patch visits and variance in the level of parasite-shedding per visit contribute to the overall parasite-density variances.

As time grows large, parasite deposition and mortality will approach stochastic equilibrium. Parasite densities consequently approach their stationary probability

distributions, with respective means and variances as $t \to \infty$:

$$\mathrm{E}\left[M_r\right] = p_{1r}\lambda_1 N_1 \mu/\xi; \qquad \mathrm{V}\left[M_r\right] = p_{1r}\lambda_1 N_1(\sigma^2 + G_1\mu^2)/2\xi \tag{4}$$

Each mean parasite density, and each variance, increases as $\lambda_1 N_1$ increases, since the rate at which patches are visited will increase. As we noted, mean parasite densities are independent of reservoir-host group size, but the variance increases with G_1. If reservoir-species groups are small ($G_1 \to 1$) parasite density is renewed frequently by relatively small increments, and M_r will fluctuate less through time. If reservoir groups are large ($G_1 \to N_1$), less frequent visits, each with larger average increments, will produce greater variance in the parasite densities. Below we show how this effect of reservoir group size on parasite-density variance influences the expected waiting time until a host-jump occurs.

Appendix A shows that the characteristic time scale of parasite mortality is $t_c = 2/\xi$. If the rate at which reservoir-host groups arrive at patches is sufficiently large, we have:

$$p_{11}\lambda_1(N_1/G_1), \quad (1-p_{11})\lambda_1(N_1/G_1) \gg t_c^{-1} = \xi/2 \tag{5}$$

If these expressions hold, the stationary distribution of each parasite density will be, by the central limit theorem, approximately normal [Lowen and Teich 1990]. Since host-jumps are far rarer than patch visits, we can assume that each M_r has a normal density with mean and variance given by the stationary values in Eq. (4).

3.2 Novel-host patch use

We index the novel host (*i.e.*, initially parasite free) as Species 2. N_2 novel-host individuals are distributed uniformly among groups of size G_2, where $G_2 \in \{1, 2, ..., N_2\}$. We take the number of groups N_2/G_2 as an integer.

The rate at which novel hosts visit patches, and their patch preferences, will together govern novel-host exposure to the parasite and, therefore, affect the probability of a host-jump. In general, Species 1 and 2 may differ in population size, the overall rate at which groups visit patches, and in the distribution of those visits across patches.

Each novel-host group enters the set of patches as an independent Poisson process. The combined probabilistic rate is $\lambda_2 N_2/G_2$; λ_2 is a constant. p_{2r} is the probability that any given visit by a novel-host group occurs at patch r. $X_{2r}(t)$ counts all novel-host visits to patch r over t time units; $r = 1, 2$. The expected value of $X_{2r}(t)$ is $\mathrm{E}\left[X_{2r}(t)\right] = p_{2r}\lambda_2 N_2 t/G_2$. Larger group size G_2 implies fewer total groups, hence fewer patch-visits per unit time. The $X_{2r}(t)$ are independent Poisson random variables; see Appendix A.

3.3 Host-jumping

We take parasite densities as stationary random variables. $f(M_r)$ represents the probability density of M_r, an approximately normal variate with mean $\mathrm{E}\left[M_r\right]$ and variance $\mathrm{V}\left[M_r\right]$, each given in Eq. (4). The N_2/G_2 novel-host groups enter the system at time $\tau = 0$. Each visit to patch r exposes the G_2 group members to an independent realization of the random variable M_r.

We assume no immunological effects of past exposure [Breban et al. 2009]. Each exposure to the parasite infects/fails to infect each group member independently. The

chance any novel host is infected upon exposure must increase monotonically in M_r.

Given exposure to the parasite, the probability of infection is a matter of dose-response analysis [Tenuis et al. 1996, Strachan et al. 2005], and we use a model favored by empirical studies. Consider the $g-th$ group member of a novel host-group during a single visit to patch r; $g = 1, 2, ..., G_2$. The conditional probability of infection is $\Pr[g \text{ infected}|M_r] = 1 - e^{-\gamma M_r}$; $\gamma > 0$. γ is the susceptibility parameter, an attribute of the parasite and novel-host combination. If γ is very small, susceptibility presents a between-host barrier [Woolhouse et al. 2005]. Some generalist endoparasites, however, jump hosts more readily, implying larger values of γ; furthermore, γ may vary with phylogenetic distance between host species [Cooper et al. 2012].

On any *single visit* to patch r, the conditional probability of a host jump, given M_r, is simply 1 minus the probability that no host is infected:

$$\Pr\left[\text{Host jump}|M_r\right] = 1 - \left(e^{-\gamma M_r}\right)^{G_2} \tag{6}$$

We assume that the same value of γ applies to all novel-host individuals. If susceptibility varies among individuals as a beta variate [Tenuis and Havelaar 2000], the results do not affect the model's qualitative predictions. Proceeding, the unconditional probability of a host-jump during a single patch-visit is:

$$\begin{aligned} \Pr\left[\text{Host jump}\right] &= 1 - \int_0^\infty e^{-\gamma G_2 M_r} f(M_r) dM_r \\ &\approx 1 - exp\left(-\gamma G_2 \mathrm{E}[M_r] + \frac{(\gamma G_2)^2}{2} \mathrm{V}[M_r]\right) \end{aligned} \tag{7}$$

by similarity to the normal distribution's moment generating function. The chance that the

parasite jumps to the novel host during a group's single exposure must increase with mean parasite density. But the same probability decreases with the variance of the parasite density. Larger reservoir-host group size G_1 increases each $V[M_r]$, and so can decrease the probability of a host-jump.

Intuitively, a larger novel-host group size must increase the probability of host-jumping on a single visit, since more hosts are exposed per visit. However, increasing G_2 decreases the number of novel host groups (N_2/G_2), so that the expected number of patch-visits per unit time declines. To explore these effects in combination, consider the probability that the parasite has failed to invade the novel-host population after multiple visits.

Applying the unconditional probability of avoiding infection on a single visit, and conditioning on the number of novel-host visits to patch r by time τ, the probability the novel-host remains without infestation after χ_r visits is:

$$\Pr[\text{No jump}|X_{2r}(\tau) = \chi_r] = \left[exp\left(-\gamma G_2 \mathrm{E}[M_r] + \frac{(\gamma G_2)^2}{2}\ \mathrm{V}[M_r]\right)\right]^{\chi_r} \tag{8}$$

Unconditionally, the probability that the novel host has not acquired the endoparasite within patch r by time τ is:

$$\Pr[\text{No jump by } \tau] = \sum_{X_{2r}(\tau)=0}^{\infty} \Pr[\text{No jump}|X_{2r}(\tau)]\ \ \Pr[X_{2r}(\tau)] \tag{9}$$

for both $r = 1$ and $r = 2$ independently. Recall that $\Pr[X_{2r}(\tau)]$ follows a Poisson probability function with mean $\mathrm{E}\left[X_{2r}(\tau)\right] = p_{2r}\lambda_2 N_2 \tau / G_2$. Then Eq. (9) has the form of a Poisson probability generating function [Ross 1983]. If $G(z)$, where $0 < z < 1$, is the generating function for a Poisson random variable x, then $G(z) = \sum_{x=0}^{\infty} z^x Pr[x]$. If k is the mean of the

Poisson variable x, we have $G(z) = exp\,(k[z-1])$. We can substitute the exponential term in Eq. (8) for z, and $\mathrm{E}\,[X_{2r}(\tau)]$ for k. Assembling the pieces, the probability that the reservoir host avoids parasite infection across all visits to patch r through time τ is:

$$\zeta_r(\tau) = exp\left([p_{2r}\lambda_2 N_2 \tau / G_2]\ \left[exp\left(-\gamma G_2 \mathrm{E}[M_r] + \frac{(\gamma G_2)^2}{2}\mathrm{V}[M_r]\right) - 1\right]\right) \tag{10}$$

Of course, the chance that the novel host avoids infection at patch r declines as time τ increases. Since the random variables $X_{21}(\tau)$ and $X_{22}(\tau)$ are independent, the overall probability that the parasite has not invaded the novel host by time τ is $\zeta_1(\tau)\ \zeta_2(\tau)$.

Let the random variable T $(T > 0)$ represent the first time a novel host is infected; T is then the waiting time until the parasite jumps to the novel host. We have $\Pr\,[T > \tau] = \zeta_1(\tau)\ \zeta_2(\tau)$. Then the expected waiting time until the parasite jumps is:

$$\begin{aligned} \mathrm{E}[T] &= \int_0^\infty \Pr\,[T > \tau]\ d\tau = \int_0^\infty \zeta_1(\tau)\ \zeta_2(\tau)\ d\tau \\ &= \frac{G_2}{\lambda_2 N_2}\left\{\sum_{r=1}^{2} p_{2r}\left[1 - exp\left(-\gamma G_2 \mathrm{E}[M_r] + \frac{(\gamma G_2)^2}{2}\mathrm{V}[M_r]\right)\right]\right\}^{-1} \end{aligned} \tag{11}$$

$\mathrm{E}[T]$ the product of the average time elapsing between consecutive patch-visits by the novel host, and the expected number of visits (to both patches) for occurrence of the first infection. The latter may not be obvious; see Appendix A.

3.4 Predictions

Inspection of the solution for $\mathrm{E}[T]$ yields some intuitive predictions. The mean waiting time for the first infection must decrease as $\lambda_2 N_2$ increases. Given group size G_2, increasing λ_2 or N_2 increases exposure to the parasite since the rate at which the novel host visits patches is

increased.

Recall that increasing reservoir group size G_1 increases each variance $\mathrm{V}[M_r]$, but does not affect mean parasite densities $\mathrm{E}[M_r]$. As a consequence, larger group size G_1 increases the mean waiting time $\mathrm{E}[T]$. Increasing G_1 reduces the rate at which reservoir hosts visit patches, but increases the level of parasite shedding when a visit occurs. This combination leaves stationary parasite densities more variable which, in turn, increases the mean time elapsing before the parasite first infects a novel host.

Increasing the variance of parasite shedding by an individual reservoir-host (*i.e.*, increasing σ^2 with μ fixed) increases parasite-density variances $\mathrm{V}[M_r]$, and so increases the expected waiting time $\mathrm{E}[T]$. Increasing the mean individual-level parasite shedding μ increases both the mean and variance of the stationary parasite densities. The former effect can prove stronger, assuming $\gamma G_2 < 1$, so that increasing μ should decrease $\mathrm{E}[T]$. In some cases, σ^2 may depend on μ, and predictions would be revised accordingly.

A primary focus concerns the effect of novel-host group size G_2 on the waiting time until the parasite jumps to the novel host. Differentiating $\mathrm{E}[T]$ with respect to G_2 does not yield a simple expression, but allows us to state two necessary, though not sufficient, conditions such that the waiting time for the first infection should increase with novel-host group size. The first condition depends on the the ζ_r, after Eq. (10), and the second depends on that expression's derivative. So, $\partial \mathrm{E}[T]/\partial G_2 > 0$ requires:

$$\mathrm{E}[M_r] > (\gamma \mathrm{V}[M_r]/2)\, G_2 \qquad \text{and} \qquad \mathrm{E}[M_r] > \gamma V[M_r] G_2 - \frac{1}{\gamma G_2}; \qquad \text{for } r = 1,\ 2 \tag{12}$$

Each condition should hold when the novel host's susceptibility to the parasite is not too large. If host-jumping involves crossing a "species barrier," novel-host susceptibility will, by

definition, be quite small. These conditions assume that parasite-density means and variances make Eq. (7) a proper probability; conditions (12) predict reasonably that the waiting time for the parasite to invade the novel host population will increase with group size G_2.

The increase in the waiting time $\mathrm{E}[T]$ with G_2 has a simple, intuitive explanation. Following our example above, doubling novel-host group size halves the expected number of patch visits per unit time. But the probability that at least one member of the larger group is infected during a single visit is less than doubled, by the non-linearity in Eq. (6). Hence larger reservoir-host groups expect more time to elapse before the first infection.

Figure 1 shows how the waiting time $\mathrm{E}[T]$ depends on the respective host-species group sizes. For any novel-host group size G_2, waiting time for a host-jump increases with G_1. For any reservoir-host roup size G_1, waiting time increases with G_2. The former effect is reduced for $G_2 = 1,\ 2$, but $\mathrm{E}[T]$ increases strongly with G_1 once $G_2 > 5$. Figure 1 assumes the reservoir prefers patch 1; $p_{11} = 0.9$. Intuitively, as the novel host‘s use of patch 1 declines, $\mathrm{E}[T]$ increases.

Figure 2 displays the waiting time $\mathrm{E}[T]$ as a function of the reservoir host‘s proportional use of patch 1, p_{11}. Note that $\mathrm{E}[T]$ is independent of p_{21} when the reservoir host acts as an ideal generalist ($p_{11} = p_{12} = 0.5$). Comparing plots 2A and 2B shows, for given parameter values, that as the novel-host changes from visiting patches as solitaries to moving in groups of 15, the expected time until their first infection increases approximately 2 orders of magnitude.

Comparing plots 2B and 2C shows that increasing the absolute number of reservoir-host patch visits, for any $(p_{11},\ p_{21})$-combination, decreases $\mathrm{E}[T]$. Comparing plots 2B and 2D shows that decreasing the variance in the number of parasites shed per reservoir host, σ^2,

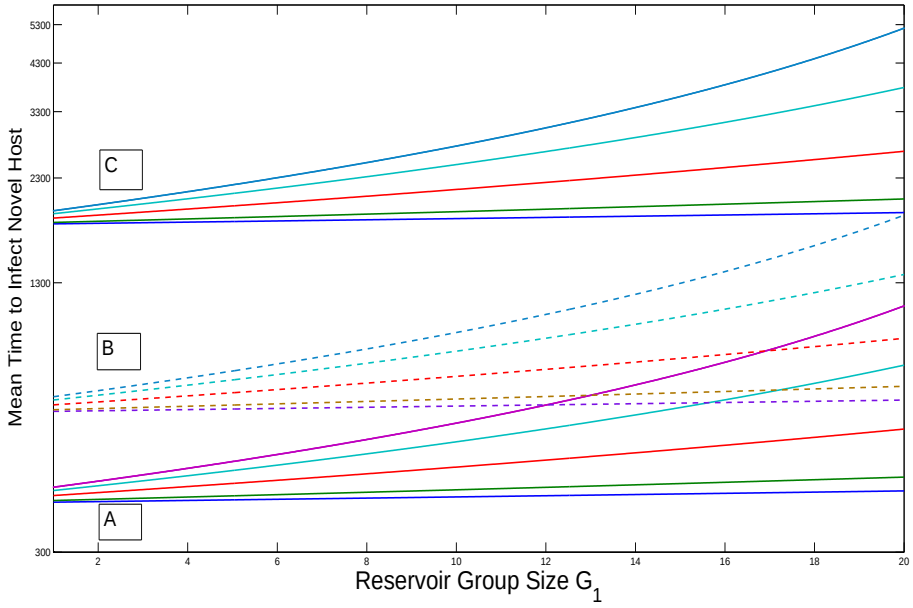

Figure 1: Mean time to jump to novel host: reservoir specializes. Abscissa: reservoir group size G_1. Ordinate: waiting time $\mathrm{E}[T]$, logarithmic scale. Reservoir prefers patch 1; $p_{11} = 0.9$. A. Novel host identical to reservoir; $p_{21} = 0.9$. B. Novel host generalizes; $p_{21} = 0.5$ (broken lines). C. Novel host specializes on patch 2; $p_{21} = 0.1$. A, B, C: As lines ascend in each set, $G_2 = 1, 2, 5, 8, 10$. Longest waiting times occur for different specialists. Parameters: $\lambda_1 N_1 = \lambda_2 N_2 = 0.5$, $\mu = 5$, $\sigma^2 = 25$, $\xi = 1$, $\gamma = 2.5 \times 10^{-3}$.

strongly decreases the expected time until the parasite jumps to the novel host. A decrease in σ^2 decreases the stationary variance of the parasite densities, from Eq. (4). Any decrease in the parasite-density variances $\mathrm{V}[M_r]$ decreases $\mathrm{E}[T]$, from Eq. (11).

3.4.1 Spatial overlap and infection hazard

The reservoir host's use of space influences parasite abundances. Given parasite densities, the novel host's use of space will govern exposure to infection, and so affect the waiting time until the parasite can jump to the new host. So, $\mathrm{E}[T]$ depends directly on overlap between novel host and parasite, and indirectly on overlap between reservoir and novel hosts. To clarify the role of patch preferences, we show how the hazard of infection varies with the p_{jr}.

Eq. (6) gives us the conditional probability of a host-jump on a single visit to patch r, given parasite density M_r, as $1 - e^{-\gamma M_r G_2}$. We associate a hazard function with this

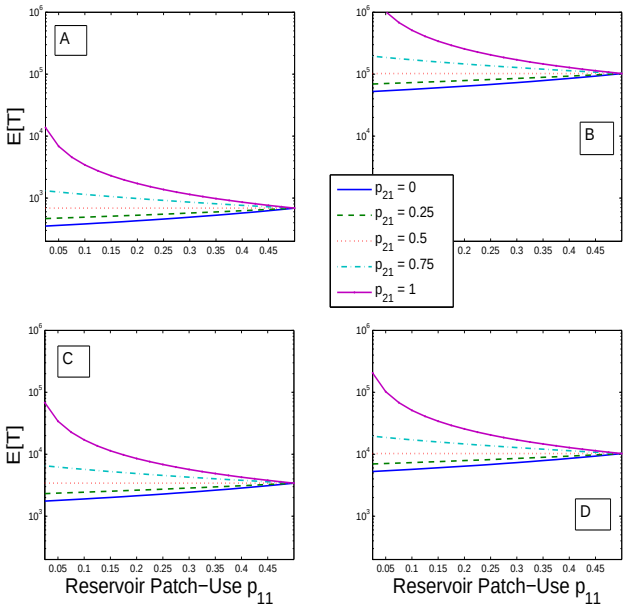

Figure 2: Mean time to jump to novel host as patch-use varies. Abscissa: reservoir preference for patch 1, p_{11}. Ordinate: waiting time E$[T]$, logarithmic scale. A. $G_2 = 1$, $\lambda_2 N_2 = 0.5$, $\sigma^2 = 60$. B. $G_2 = 15$, $\lambda_2 N_2 = 0.5$, $\sigma^2 = 60$. C. $G_2 = 15$, $\lambda_2 N_2 = 15$, $\sigma^2 = 60$. D. $G_2 = 15$, $\lambda_2 N_2 = 0.5$, $\sigma^2 = 0$. Legend shows that in each plot E$[T]$ increases as p_{21} increases, but results converge across levels of p_{21} as reservoir host changes from patch-2 specialist to generalist. Fixed parameters: $G_1 = 10$, $\lambda_1 N_1 = 0.5$, $\mu = 10$, $\xi = 2$, $\gamma = 2.5 \times 10^{-3}$.

infection probability: $h_r(G_2) = \gamma M_r G_2$; we recall that a larger novel-host group increases the hazard of parasite invasion on a single visit. If we average the single-visit hazard between patches, we have $\langle h_r(G_2) \rangle = \mathrm{E}\left[\gamma G_2 \left(p_{21} M_1 + [1 - p_{21}] M_2\right)\right]$.

Eq. (4) provides the mean parasite densities E$[M_r]$, so that the mean hazard of infection becomes:

$$\langle h_r(G_2) \rangle = \frac{\gamma G_2 \lambda_1 N_1 \mu}{\xi} \left(p_{21}[2p_{11} - 1] + 1 - p_{11}\right) \tag{13}$$

Intuitively, the infection hazard has the minimal feasible value at $p_{21} = 1$ when $p_{11} < 1/2$, and at $p_{21} = 0$ when $p_{11} > 1/2$. Any degree of bias in the reservoir host's patch (i.e., $p_{11} \neq 1/2$) implies that strong specialization by the novel host will increase E$[T]$. These considerations might prove important if hosts avoid locations where a parasite's infectious stage is concentrated

[Freeland 1976, Hausfater and Meade 1982, Hutchings et al. 2001, Turner et al. 2014]. If

$p_{11} = 1/2$, $f(M_1) = f(M_2)$, and any p_{21} generates the same mean infection hazard. The greatest hazard rates occur when both species exhibit low patch-use diversity and prefer the same patch. The lowest hazard rates occur when spatial overlap is minimal; each host prefers a different patch. Both infection-hazard extremes occur when each species has low resource-use diversity [Patil and Taillie 1979]; the difference between low and high hazards then depends on spatial niche overlap.

4 Discussion

Our results offer two important insights regarding a parasite's jump between host species. First, the time expected to elapse before an environmentally transmitted parasite first infects a novel host increases both with group size in the endemically infected reservoir population, and with group size in the novel host. Second, overlap in the two species‘ use of space will not always predict the likelihood of novel-host infection; its predictive utility varies with the degree of reservoir-host patch specialization.

The mechanisms generating the increase in the waiting time for the host-jump differ between the two host species, but both results involve interaction between host group size and the parasite-density variances. Our model's novel host uses patches independently of both reservoir behavior and parasite densities. As a consequence of the parasite-density variances interacting with a non-linear probability of infection, larger novel-host group sizes extend the expected time elapsing until the first infection occurs. Once a host-jump has occurred, however, the parasite‘s initial spread might be faster in a larger group [Cross et al. 2005].

Reservoir patch preferences govern the degree of spatial heterogeneity. Reservoir group

size and random among-individual variation in parasite contamination influence temporal heterogeneity of each within-patch parasite density. When the reservoir host specializes in its patch use (plots A and C in figure 2) parasite densities become spatially heterogeneous, and the waiting time for host-jumping increases as between-host species similarity in patch use declines. However, when the reservoir host generalizes in its patch use (plots B and D in figure 2) spatial heterogeneity of parasite densities disappears, and the novel host‘s patch preferences have no effect on the waiting time.

For simplicity we have treated space implicitly. In particular applications, the number of resource clusters will be large, and groups may visit specific locations in an approximately periodic manner [Hausfater and Meade 1982]. Some gregarious species may actively avoid food patches to reduce exposure to parasitism [Hutchings et al. 2001]. Other species may seek the most productive food patches and, in doing so, increase the chance of infection [Turner et al. 2014]. We also assumed that the probability of infecting a given host increased with parasite density in a strictly concave manner. The minimal parasite exposure required to infect a host, often termed the infective dose, varies substantially across host-parasite interactions [Leggett et al. 2012]. As long as environmental densities of the parasite (hence, exposure) characteristically exceed the infective dose, our model should apply. However, more complex dose-response relationships, and sufficiently small $\mathrm{E}[M_r]$, could alter predictions.

Relationships between reservoir patch use and the distribution of parasite densities can, of course, exhibit more complexity than we assume. Some microparasite populations grow not only within hosts, but also in the environment [Bani-Yaghoub et al. 2012]. Abiotic variation and biotic processes can affect patch-scale parasite densities, or modulate the infectiousness of a given parasite density, as well as the susceptibility of a given host

[Caraco and Wang 2008, Cizauskas et al. 2014b].

As climate change and habitat destruction increasingly alter a host species‘ use of resources, social behavior, and spatio-temporal overlap with other species, we anticipate that parasite host-jumping will occur more frequently. Our results demonstrate how host species‘ social organization and collective use of a spatially heterogeneous resource environment can influence parasites switching hosts or changing from host specialization to generalization, and suggest directions for empirical research.

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A Online appendix: Some model details

The expected parasite densities are:

$$\mathrm{E}\left[M_1(t)\right] = \frac{p_{11}}{\xi}\lambda_1 N_1 \mu(1 - e^{-\xi t}) \quad \mathrm{E}\left[M_2(t)\right] = \frac{1 - p_{11}}{\xi}\lambda_1 N_1 \mu(1 - e^{-\xi t}) \tag{A1}$$

It seems useful to establish probabilistic independence of the parasite densities $M_r(t)$. First, we assure that the number of patch visits are independent; the demonstration is standard [Ross 1983]. A host species enters the resource set (patches 1 and 2) at constant probabilistic rate $\lambda N/G$; given entry, p is the probability of visiting patch 1. At time t $(t > 0)$ patch 1 has been visited $X_1(t)$ times, and patch 2 has been visited $X_2(t)$ times; the respective means are $p\lambda(N/G)$ and $(1 - p)\lambda(N/G)$.

Let $W(t) = X_1(t) + X_2(t)$, the total number of visits by time t. We want the joint distribution of the $X_r(t)$, and begin by conditioning on the realization of $W(T)$:

$$Pr[X_1(t) = y,\ X_2(t) = z] = \sum_{w=0}^{\infty} Pr[X_1(t) = y,\ X_2(t) = z|W(t) = w]Pr[W(t) = w] \quad \text{(A2)}$$

Given the definition of $W(t)$, the right side of Eq. (A2) must be:

$$Pr[X_1(t) = y,\ X_2(t) = z|W(t) = y + z]Pr[W(t) = y + z]$$

Since p is a constant, $Pr[X_1(t) = y,\ X_2(t) = z|W(t) = y + z]$ is simply a binomial random variable:

$$Pr[X_1(t) = y,\ X_2(t) = z|W(t) = y + z] = \binom{y+z}{y} p^y(1-p)^x \quad \text{(A3)}$$

Then, unconditionally:

$$\begin{aligned} Pr[X_1(t) = y,\ X_2(t) = z] &= \frac{(y+z)!}{y!z!}p^y(1-p)^z\ e^{-\lambda(N/G)}\frac{(\lambda(N/G))^{y+z}}{(y+z)!} \\ &= e^{-p\lambda(N/G)}\frac{(p\lambda(N/G))^y}{y!}\ e^{-(1-p)\lambda(N/G)}\frac{((1-p)\lambda(N/G))^z}{z!} \end{aligned} \quad \text{(A4)}$$

Applying Eq. (A4), we infer that $X_{11}(t)$ and $X_{12}(t)$ are independent Poisson random variables. Our model assumes that each m, the increment to the local parasite density, is an independent, identically distributed realization of the same random variable. Then we conclude that the shot-noise processes, the parasite densities, $M_r(t)$ and their stationary distributions are probabilistically independent.

Here we specify the characteristic time of the parasite mortality process. The characteristic time t_c is usually taken as the square of the survival integral divided by the

integral of the square [Lowen and Teich 1990]. Since we assume simple exponential decay,

368 we have:

$$t_c = \left(\int_0^\infty \ e^{-\xi t} dt \right)^2 / \int_0^\infty \ e^{-2\xi t} dt \tag{A5}$$

Then $t_c = (\xi)^{-2}/(2\xi)^{-1} = 2/\xi$, twice the mean longevity of a parasite.

370 The expected waiting time for the first infection, Eq. (11), can be approached informally, as follows. Novel-host groups collectively enter the set of patches R at probabilistic rate

372 $\lambda N_2/G_2$; the mean time between consecutive visits is the inverse of this rate. Suppose that the host-jump were to occur at the $w-th$ visit; that is, when $W(\tau) = w$. Conditioned on

374 no infection during the first $(w-1)$ visits, the *a priori* probability of infection at $W = w$ is $p_{21}\theta_1 + (1-p_{21})\theta_2$. θ_r is the probability of a host jump during a single visit to patch$r$, and

376 does not depend on w. From Eq. (7), we have for $r = 1, 2$:

$$\theta_r = 1 - exp\left(- \gamma G_2 \mathrm{E}[M_r] + \frac{(\gamma G_2)^2}{2} \ \mathrm{V}[M_r] \right) \tag{A6}$$

If the host-jump occurs at visit w, the distribution between patches of the previous $(w-1)$

378 visits, none of which led to infection, must be binomial. Then the probability that the first infection occurs at visit w is:

$$\Omega(w) = [p_{21}\theta_1 + (1-p_{21})\theta_2] \sum_{x=1}^{w-1} \binom{w-1}{x} p_{21}^x (1-p_{21})^{w-1-x} (1-\theta_1)^x (1-\theta_2)^{w-1-x} \tag{A7}$$

380 Then:

$$\begin{aligned} \Omega(w) &= [p_{21}\theta_1 + (1-p_{21})\theta_2] \sum_{x=1}^{w-1} \frac{(w-1)!}{x!\ (w-1-x)!} \left[p_{21}(1-\theta_1)\right]^x \left[(1-p_{21})(1-\theta_2\right]^{w-1-x} \\ &= [p_{21}\theta_1 + (1-p_{21})\theta_2] \left[p_{21}(1-\theta_1) + (1-p_{21})(1-\theta_2)\right]^{w-1} \end{aligned} \tag{A8}$$

$\Omega(w)$, for $w = 1, 2, ...$ follows a geometric probability function. The mean number of visits until the host-jump occurs is then $\left[\sum_{r=1}^{2} p_{2r}\theta_r\right]^{-1}$. Multiplying the mean time between novel-host patch-visits and the mean number of visits until first infection yields the text's expression for $\mathrm{E}[T]$.

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