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Environmentally transmitted parasites: Host-jumping in a heterogeneous environment

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HIGHLIGHTS

- We model the expected time elapsing for an environmentally transmitted parasite to jump from a reservoir host to a novel host.
- Mean parasite density in the environment does not depend on reservoir host group size, but the variance increases with group size.
- Increasing the novel host's group size increases the expected waiting time for the first infection.

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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 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 the 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 (Breban et al., 2009; Cizauskas et al., 2014; Miller et al., 2006). Models of environmentally transmitted parasites emphasize how the dynamics differs from infections spread through between-host contact (Bani-Yaghoub et al., 2008; Garira et al., 2014); these distinctions have significant implications for epidemiological invasion (Breban et al., 2010; Lahodney et al., 2014; Rohani et al., 2009) and for the likelihood a parasite can "jump" to a novel host species (Caraco et al., 2014; Reluga et al., 2007; Woolhouse et al., 2005).

Our study of environmental transmission focuses on host-jumping, the initial infection of a novel-host population or species. 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 explore the potential importance of social structure in environmentally transmitted infections, and ask how each species' group size affects the random waiting time until the parasite first jumps to the novel host. Then, under more restrictive assumptions, we find the expected time elapsing until all groups have at least one infected member.

2. Background

This section introduces the ecological context for our analysis of host-jumping. Then we motivate our specific assumption of chronic infection in the reservoir host.

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2.1. Host-jumping

Every phylogenetic group of parasites, from viruses to phytophagous arthropods and parasitic plants, includes some species that exploit a wide range of hosts, and other species with a very narrow host range (Ives and Godfray, 2006). Generalist parasites presumably have crossed multiple species-barriers, without subsequent speciation, during niche expansion. Even presumed specialists sometimes jump to a new host, often in conjunction with a mutational event; some recent epidemics in human populations have emerged after a virus expanded its host range (Woolhouse et al., 2005). Contemporarily, climate change and increased species-introductions are driving biogeographical range expansion of many parasites (Crowl et al., 2008; Jolles et al., 2008). Ecological disturbance, especially human landscape alteration, is placing new host–parasite combinations in common environments (Patz et al., 2000). These processes clearly increase opportunities for parasites to jump to novel hosts (Cooper et al., 2012).

The prevalence of parasitic infection can increase with host group size (Brown et al., 2001; Freeland, 1979). Given an established host–parasite association, the frequency of transmission opportunities (*via* direct contact or through the environment) should increase with group size (Hoogland, 1979). In a limited review, Côté and Poulin (1995) concluded that both the prevalence and intensity (parasites/host individual) of macroparasistes increased with social group size. A more extensive meta-analysis (Rifkin et al., 2012) cautioned that infection frequency sometimes declines in larger groups. In any case, a role for group size in host-jumping apparently has not been addressed.

To motivate our analysis, consider the Hausfater and Meade (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 h, 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.

2.2. Chronic infection

Our model assumes chronic infection of a set of reservoir hosts. That is, infected individuals do not recover. Chronic infections are not uncommon among mammalian hosts; both microparasites (Wright, 2006) and macroparasitres (Else et al., 2006) frequently infect individuals chronically. Cizauskas et al. (2014) offer a good example. Gastrointestinal helminths infect wild plains zebra (*Equus quagga*) chronically. Parasite prevalence remains at 100 per cent year-round, although the intensity of infections shows strong seasonality; in Section 4 we return to consequences of seasonality for our model.

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 (Woolhouse et al., 2005), we treat deposition of the parasite by the reservoir species, and acquisition of the parasite by the novel host, as stochastic processes. Combining these processes identifies the distribution of waiting time associated with initial infection of the novel host.

We dichotomize the 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 fix N_1 as a constant. The N_1 reservoir hosts are distributed uniformly across groups of size G_1, where $G_1 \in \{1, 2, \ldots, N_1\}$. 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 any 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 (Caraco et al., 1995).

Table 1
Definitions of model symbols.

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	Random 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
θ_r	Probability of host-jump in a single visit to patch r
$\zeta_r(\tau)$	Probability novel host has not acquired parasite in patch r by time τ
T	Random waiting time until parasite first jumps to novel host

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 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 the 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 (Cizauskas et al., 2014).

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 $\sigma^2>\mu$ 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 documented for some macroparasites (Hausfater and Meade, 1982) and assumed for viral (Rohani et al., 2009) and bacterial (Turner et al., 2006) microparasites. 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 (Laio et al., 2001; Lemoine and Wenocur, 1986). 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 (Lowen and Teich, 1990; Ross, 1983). Expected values are:

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

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

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

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 $t\to\infty$:

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

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 $G_2\in\{1,2,\ldots,N_2\}$. 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 $\mathrm{E}[X_{2r}(t)]=p_{2r}\lambda_2 N_2 t/G_2$. 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 $\mathrm{E}[M_r]$ and variance $\mathrm{V}[M_r]$, 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 that 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 (Strachan et al., 2005; Tenuis et al., 1996), 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; $g = 1, 2, \ldots, G_2$. The conditional probability of infection is $\Pr[g \text{ infected} \mid M_r] = 1 - e^{-\gamma M_r}; \gamma > 0$. 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:

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

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 visit to patch r is:

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

by similarity to the normal distribution's moment generating function. As a convenience, we let θ_r represent this probability in some expressions below.

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:

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

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

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

for both $r=1$ and $r=2$ independently. Recall that $\Pr[X_{2r}(\tau)]$ follows a Poisson probability function with mean $\mathrm{E}[X_{2r}(\tau)] = p_{2r}\lambda_2 N_2 \tau / G_2$. Then Eq. (9) has the form of a Poisson probability generating function. 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:

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

$\mathrm{E}[T]$ 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. Appendix A gives details about the probability distribution of the number of patch visits prior to novel-host infection. Appendix A.1 also shows that the time of initial infection, T, has an exponential density with rate $(\lambda_2 N_2 / G_2)\sum_r p_{2r}\theta_r$.

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 ζ_r, after Eq. (10), and the second depends on that expression's derivative. So, $\partial \mathrm{E}[T]/\partial G_2 > 0$ requires:

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

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 equation (7) a proper probability.

The increase in the waiting time 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,

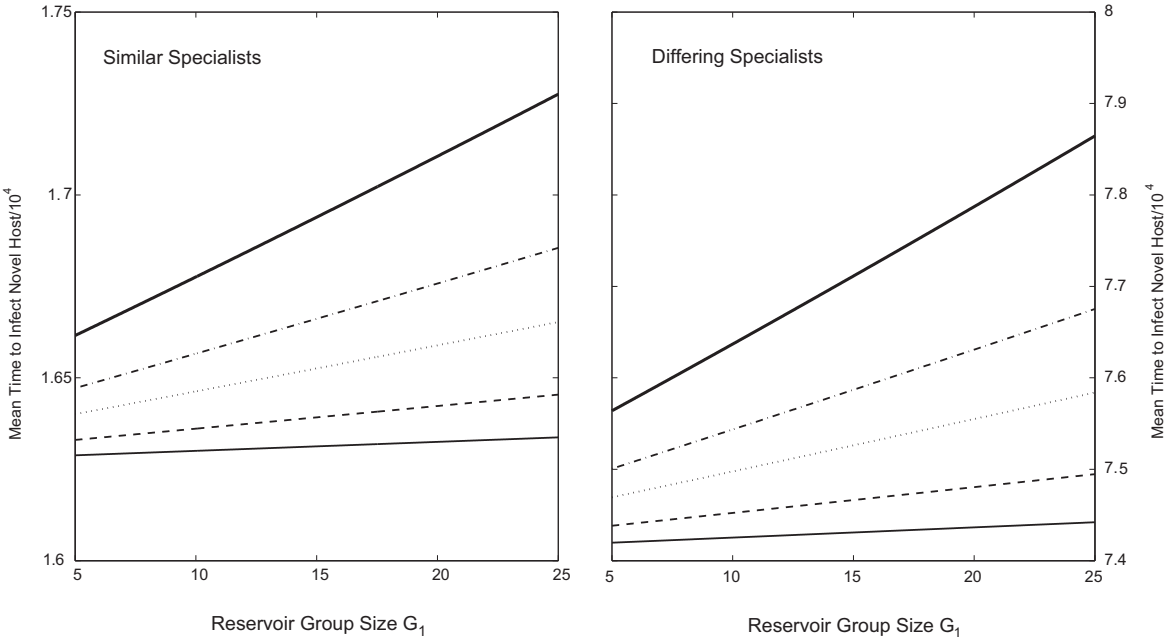

Fig. 1. Mean time to jump to novel host: reservoir specializes. Abscissa: reservoir group size G_1. Ordinate: waiting time E[T]/10^4. Reservoir host prefers patch 1; $p_{11}=0.9$ for all G_1. Left panel: Novel host patch preference identical to reservoir; $p_{21}=0.9$. Right panel: Novel host specializes on patch 2; $p_{21}=0.1$. Each panel: G_2 increases as lines ascend; thin solid line: $G_2=2$, dashed solid line: $G_2=5$, dotted line: $G_2=10$, dot-dash: $G_2=15$, thick solid line: $G_2=25$. Note different scaling of ordinates. For any (G_1, G_2)-combination, waiting time for host-jumping is 4–5 times greater for differing specialists. Parameters: $\lambda_1 N_1=\lambda_2 N_2=0.5$, $\mu=3$, $\sigma^2=50$, $\xi=1$, $\gamma=10^{-4}$.

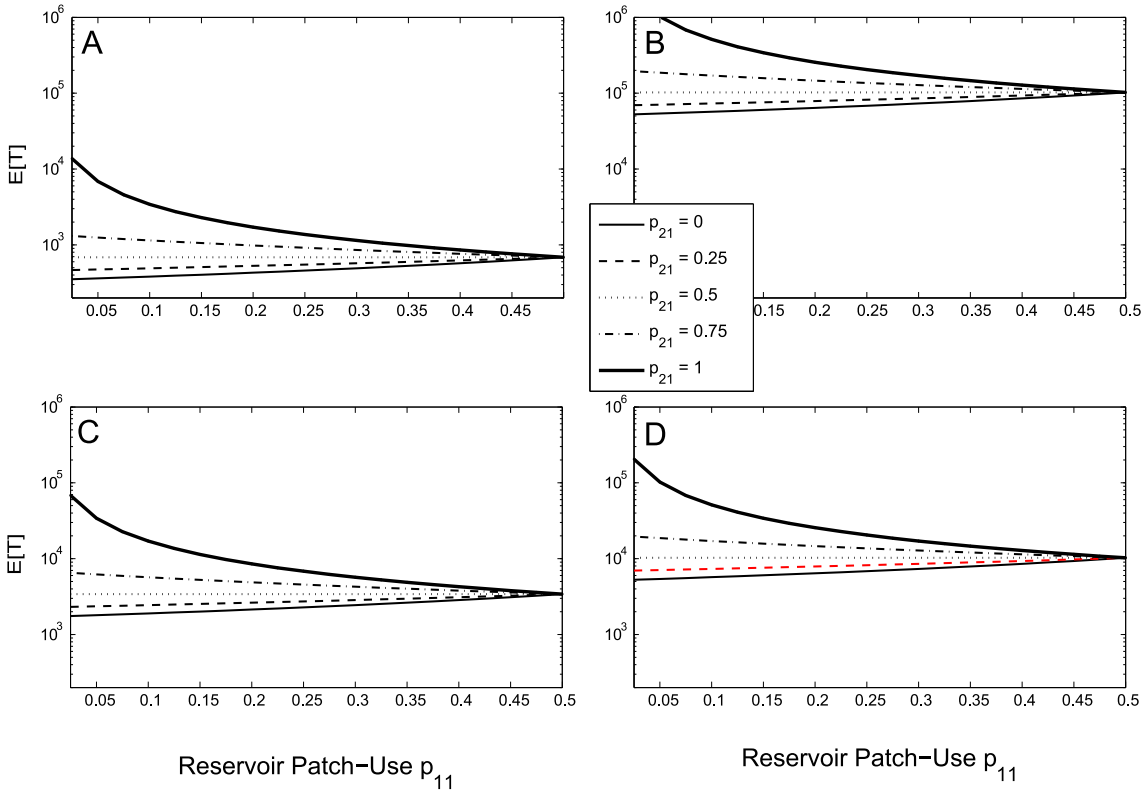

Fig. 2. Mean time to jump to novel host as patch-use varies. Abscissa: reservoir preference for patch 1, p_{11}. Ordinate: waiting time E[T], logarithmic scale. A. $G_2=1$, $\lambda_2 N_2=0.5$, $\sigma^2=60$. B. $G_2=15$, $\lambda_2 N_2=0.5$, $\sigma^2=60$. C. $G_2=15$, $\lambda_2 N_2=15$, $\sigma^2=60$. 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: $G_1=10$, $\lambda_1 N_1=0.5$, $\mu=10$, $\xi=2$, $\gamma=2.5\times10^{-3}$.

by the non-linearity in Eq. (6). Hence larger novel-host groups expect more time to elapse before the first infection.

Fig. 1 shows how the mean waiting time E[T] depends on the respective host species' group sizes. For any novel-host group size G_2, waiting time for host-jumping increases with G_1. Increasing novel-host group size G_2 accelerates the response of E[T] to reservoir-host group size. Plots in Fig. 1 assume that the reservoir host strongly prefers patch 1; $p_{11}=0.9$ in both left and right panels. For plots in the left panel, the reservoir-host has the same patch preference; $p_{21}=0.9$. For the right panel, the reservoir-host prefers patch 2; $p_{21}=0.1$. Given specialization of the reservoir host's patch use, E[T] must increase, intuitively, when the novel host becomes a specialist on the other patch. The change in p_{21}, left *versus* right panel of Fig. 1, produces an approximately 5-fold increase in E[T].

Fig. 2 displays the waiting time E[T] as a function of the reservoir host's proportional use of patch 1, p_{11}. G_1 remains fixed across panels; $G_1=10$. Each panel shows how E[T] can vary as the novel host's patch use, specifically p_{21}, changes. Note that E[T], as a function of p_{21}, converges to the same value when the reservoir host uses each patch equally ($p_{11}=p_{12}=0.5$).

In panel A of Fig. 2, the novel host visits patches solitarily; $G_2=1$. In panel B, G_2 has been increased to 15 with other parameters held constant. The larger novel host group size increases the expected time until the first infection by approximately 2 orders of magnitude.

Panels B and C of Fig. 2 have respective $\lambda_2 N_2$ values equal to 0.5 and 15; no other parameter value differs between panels. Increasing the expected number of novel-host patch visits, for any (p_{11}, p_{21})-combination, decreases E[T]. Parameter values for panels B and D differ only in the value of σ^2, the variance in the number of parasites shed per reservoir host. Reducing σ^2 strongly decreases the expected time until the parasite jumps to the novel host. Reducing σ^2 decreases the stationary variance of the parasite densities, from Eq. (4). Any decrease in the parasite-density variances V[M_r] decreases E[T], from Eq. (11).

3.4.1. Waiting time density

Appendix A.1 gives the probability density of the time elapsing until the novel host is first infected. Here we plot the density $f(T)$ for different group sizes, to show how $E[T]$, and its variance, increases with group size. Fig. 3 indicates how an increase in either the reservoir or novel-host group size diminishes the likelihood of short waiting times, and increases the chance of longer times elapsing before the host-jump. Hence larger groups increase the mean and variance of the exponential waiting time.

3.4.2. Number of infected novel-host groups

Assuming that the parasite's reproduction is better adapted to the reservoir host, we let infected novel hosts have a negligible effect on the parasite densities M_r. If all infections of novel-host individuals occur within the set of R patches (perhaps a tenuous assumption) we can track the number of novel-host groups with at least one member infected.

Let $I(\tau)$ count the number of novel-host groups where one or more members have acquired the parasite; $I(\tau)=0,1,\ldots,(N_2/G_2)$. Then $[(N_2/G_2)-I(\tau)]$ novel-host groups have no infected individuals at time τ. From our model above, each novel-host group independently awaits *its own* first infection at stochastic rate $\lambda_2\sum_r p_{2r}\theta_r$, where θ_r is given by Eq. (7). Therefore, the collective rate at which novel-host groups acquire a first infection is:

$$\rho(\tau)=\frac{N_2-I(\tau)G_2}{G_2}\lambda_2\sum_r p_{2r}\theta_r \tag{13}$$

where $I(\tau=0)=0$. The rate of infecting a member of *any* of the all-susceptible groups declines linearly as the number of groups with an infection increases. Consequently, the expected time elapsing between consecutive increases in $I(\tau)$ grows larger as the number of groups with an infection increases.

Given the assumed independence of infections, $I(\tau)$ increases as a binomial process:

$$Pr[I(\tau)=I]=\binom{N_2/G_2}{I}\left[1-e^{-\lambda_2\tau\sum_r p_{2r}\theta_r}\right]^{I}\left[e^{-\lambda_2\tau\sum_r p_{2r}\theta_r}\right]^{[(N_2/G_2)-I]} \tag{14}$$

Our analysis has focused on the waiting time for the ecologically most important of these events, the first, since that is the "founding event" (Reluga et al., 2007), when host-jumping occurs. Appendix A.1. gives the probability density for the total waiting

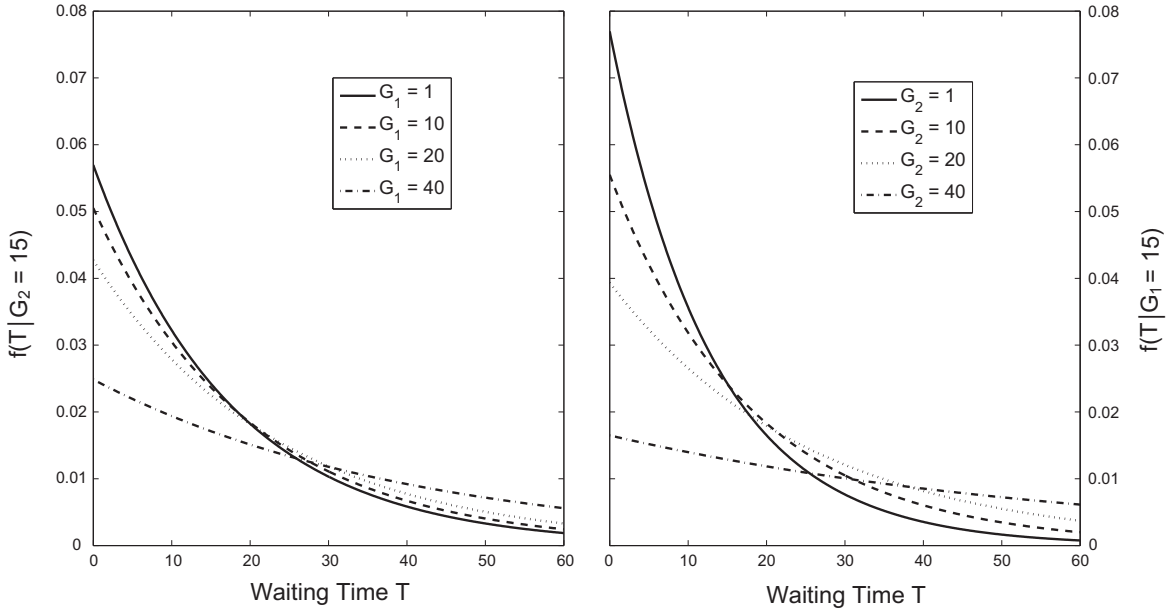

Fig. 3. Probability density of waiting time for initial infection of novel host. See Eq. (A.13). Left panel: $G_2=15$; G_1 increases a indicated in the legend. Given G_2, increasing reservoir-host group size increases the mean and variance of waiting time T. Right panel: $G_1=15$; G_2 increases as indicated in the legend. Given G_1, increasing novel-host group size increases mean and variance of T. In each panel $\lambda_1 N_1=\lambda_2 N_2=2$, $\mu=4$, $\sigma^2=60$, $\xi=0.1$, $\gamma=10^{-3}$, and $p_{11}=p_{21}=0.5$.

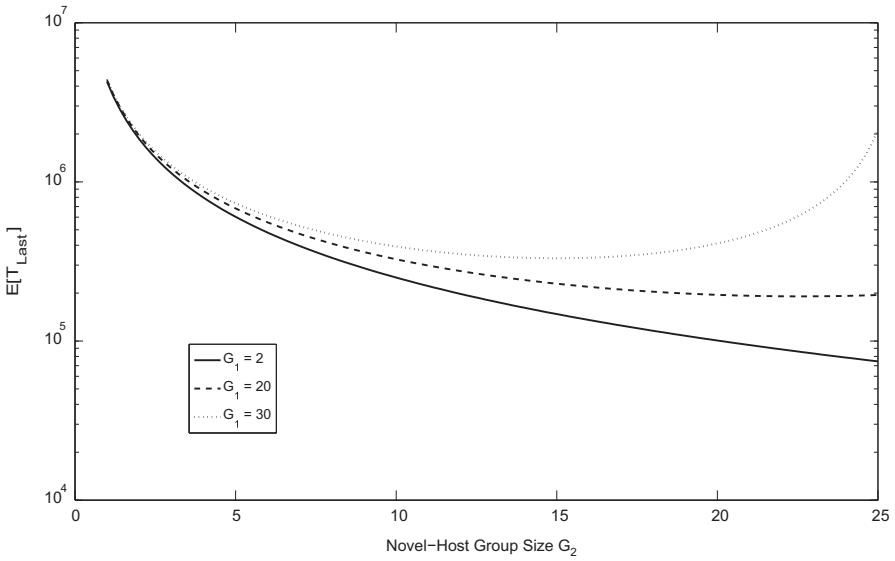

Fig. 4. Expected time until each group has one or more infected members. Ordinate: $\mathrm{E}[T_L]$ plotted on logarithmic scale. Given G_2, increasing reservoir group size G_1 increases $\mathrm{E}[T_L]$. Note that $\mathrm{E}[T_L]$ declines strictly monotonically with G_2 for smaller values of G_1. Equivalently, $\mathrm{E}[T_L]$ declines as the number of novel-host groups declines. $\lambda_1 N_1 = 0.5$, $\lambda_2 = 10^{-3}$, $N_2 = 200$, $\mu = 5$, $\sigma^2 = 25$, $\xi = 1$, $\gamma = 10^{-3}$, and $p_{11} = p_{21} = 0.5$.

time for infection of k of the (N_2/G_2) novel-host groups (Boswell et al., 1979).

3.4.3. All groups infected independently

Finally we consider the total time elapsing until each of the (N_2/G_2) novel-host groups has one or more infected member. This event occurs at time $T_{N_2/G_2} \equiv T_L$ (L for last) which has probability density:

$$f(T_L) = \frac{\lambda_2 N_2}{G_2}\left(\sum_r p_{2r}\theta_r\right) e^{-\lambda_2 T_L \sum_r p_{2r}\theta_r}\left[1 - e^{-\lambda_2 T_L \sum_r p_{2r}\theta_r}\right]^{(N_2/G_2)-1} \tag{15}$$

The expected waiting time for an infection in all novel-host group is:

$$\mathrm{E}[T_L] = \left(\lambda_2 \sum_r p_{2r}\theta_r\right)^{-1} \sum_{j=1}^{N_2/G_2}\left(\frac{N_2}{G_2} - j + 1\right)^{-1} \approx \frac{\ln(N_2/G_2)}{\lambda_2 \sum_r p_{2r}\theta_r} \tag{16}$$

$\mathrm{E}[T_L]$ always exceeds $(N_2/G_2)\mathrm{E}[T]$, from Eq. (11) since the time between infection of consecutive groups increases as $I(\tau)$ increases.

Group size can modulate density-dependent effects on individual survival and reproduction; consequently, the relationship between group size and population size can be complex (Coolen et al., 2007; Trainor and Caraco, 2006). We have assumed fixed population size, and shown how the time of host-jumping can grow large as group size in either the reservoir or novel host increases. Larger novel-host groups, with N_2 fixed, reduces the total number of groups; hence the total number of infections leaving no group without an infected member is reduced. Essentially, larger (hence, fewer) groups increase the time elapsing to the first infection ($\mathrm{E}[T]$), and can decrease the time elapsing until each group has one or more infected members ($\mathrm{E}[T_L]$). With N_2 fixed, larger group size G_2 delays host-jumping, but (ironically) can reduce the waiting time for the parasite to be introduced into every group. Fig. 4 shows an example; larger, hence fewer, groups all have at least one infection faster until both G_1 and G_2 are relatively large. In the latter case, the variance in each parasite density is large enough that sufficiently large G_2 will increase $\mathrm{E}[T_L]$.

3.4.4. 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 governs exposure to infection, and so affects the waiting time until the parasite can jump to the new host. To clarify the role of patch preferences, we show how the hazard of infection varies with the p_{jr}.

From Eq. (6), the conditional probability of a host-jump on a single visit to patch r, given parasite density M_r, is $1 - e^{-\gamma M_r G_2}$. We associate a hazard function with this infection probability: $h_r(G_2) = \gamma M_r G_2$. If we average the single-visit hazard between patches, we have $\langle h_r(G_2)\rangle = \mathrm{E}\left[\gamma G_2 (p_{21}M_1 + [1-p_{21}]M_2)\right]$.

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

$$\langle h_r(G_2)\rangle = \frac{\gamma G_2 \lambda_1 N_1 \mu}{\xi}(p_{21}[2p_{11} - 1] + 1 - p_{11}) \tag{17}$$

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 bias in the reservoir host's patch (i.e., $p_{11} \neq 1/2$) implies that strong specialization by the novel host will increase $\mathrm{E}[T]$. These considerations might apply if hosts avoid locations where a parasite's infectious stage is concentrated (Freeland, 1976; 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 assumptions best match intestinal parasites of humans, non-human primates and gregarious herbivores, ordinarily transmitted via the fecal-environment-oral route (Cizauskas et al., 2014; Hutchings et al., 2001). But many macroparasites, as well as certain bacteria and viruses, also use this mode of transmission. Jumps between species may be rare events, but their ecological

and epidemiological significance cannot be dismissed. Historically, host-jumping has preceded the emergence of a series of major infections of humans (Wolfe et al., 2007).

Our results offer two important insights regarding a parasite's jump between hosts. First, the time expected to elapse before an environmentally transmitted parasite first infects a novel host increases both with group size in the chronically 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.

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 (panels A and C in Fig. 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 (panels B and D in Fig. 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 (Joh et al., 2009; 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 $E[M_r]$, could alter predictions. To focus tractably on effects of group size, we kept each host-population size constant. We consider this an acceptable assumption for chronic infection. But for infections sufficiently virulent (Alizon et al., 2009; Cressler et al., 2015) that host mortality increases significantly, host-jumping would be addressed within a two-species population dynamics.

Reservoir patch use and the distribution of parasite densities can, of course, exhibit complex relationships. Some microparasite populations grow not only within hosts, but also in the environment (Bani-Yaghoub et al., 2008). Abiotic variation and biotic processes can affect within-patch parasite densities, or modulate the infectiousness of a given parasite density, as well as the susceptibility of a given host (Cizauskas et al., 2014). A reviewer notes that seasonality can impact parasite densities at the among-patch scale. Cizauskas et al. (2014) found that the prevalence of helminth infection in plains zebra remained very high throughout the year, but infection intensities peaked just after the rainy season. In our model's reservoir host, this sort of seasonality would imply fluctuations in the average impact of an individual on parasite densities and, consequently, on the likelihood a novel-host is infected during any patch visit.

Our model's analysis suggests some ideas linking social organization and a population's acquisition of a new parasite. However, we restrict transmission to exposure events distributed across a set of discrete locations. Most models of environmental transmission assume homogeneous mixing of susceptible hosts and a parasite's free-living stage (Kysela and Turner, 2007); somewhat similar constructions can capture the dynamics of vector-borne transmission (Feng and Velasco-Hernández, 1997). Some more recent work combines direct and indirect infection transmission, to point out the role of environmental reservoirs in pathogen persistence (Rohani et al., 2009). Our study simplifies certain plausible dynamics to emphasize effects social group size might exert on environmental transmission of a parasite across species boundaries.

As climate change and habitat destruction increasingly alter a host species' use of resources, its social behavior, and spatio-temporal overlap with other species, we can anticipate that parasite host-jumping will occur more frequently. Our focus suggests how social organization and 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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Appendix A. Some model details

Expected parasite densities are:

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

For completeness, we develop the probabilistic independence of the parasite densities $M_r(t)$; the approach is standard (Ross, 1983). First, we assure that the numbers of patch visits are independent. A host species enters the set of patches 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 $W(t)$:

$$Pr[X_1(t) = y, X_2(t) = z] = \sum_{w=0}^{\infty} Pr[X_1(t) = y, X_2(t) = z \,|\, W(t) = w] Pr[W(t) = w] \tag{A.2}$$

Given the definition of $W(t)$, the right-hand side of Eq. (A.2) 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:

$$\Pr[X_1(t) = y, X_2(t) = z \,|\, W(t) = y+z] = \binom{y+z}{y} p^y (1-p)^z \tag{A.3}$$

Then, unconditionally:

$$\begin{aligned}\Pr[X_1(t) = y,\ X_2(t) = z] &= \frac{(y+z)!}{y!z!} p^y (1-p)^z e^{-\lambda(N/G)} \frac{\left(\lambda(N/G)\right)^{y+z}}{(y+z)!} \\ &= e^{-p\lambda(N/G)} \frac{\left(p\lambda(N/G)\right)^y}{y!} e^{-(1-p)\lambda(N/G)} \frac{\left((1-p)\lambda(N/G)\right)^z}{z!}\end{aligned} \tag{A.4}$$

Eq. (A.4) shows 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 a characteristic scale of parasite mortality. 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, we have:

$$t_c = \left(\int_0^\infty e^{-\xi t}\,dt\right)^2 \Big/ \int_0^\infty e^{-2\xi t}\,dt \tag{A.5}$$

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

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 at a probabilistic rate $\lambda_2 N_2/G_2$; 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 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 does not depend on w. From Eq. (7), we have for $r = 1, 2$:

$$\theta_r = 1 - \exp\left(-\gamma G_2 \mathrm{E}[M_r] + \frac{(\gamma G_2)^2}{2}\mathrm{V}[M_r]\right) \tag{A.6}$$

If the host-jump occurs at visit w, the distribution between patches of the previous $(w-1)$ visits, none of which led to infection, must be binomial. Then the probability that the first infection occurs at visit w is:

$$\Omega(w) = [p_{21}\theta_1 + (1-p_{21})\theta_2] \sum_{x=1}^{w-1} \binom{w-1}{x} p_{21}^x (1-p_{21})^{w-1-x} (1-\theta_1)^x \times (1-\theta_2)^{w-1-x} \tag{A.7}$$

Then:

$$\Omega(w) = [p_{21}\theta_1 + (1-p_{21})\theta_2] \sum_{x=1}^{w-1} \frac{(w-1)!}{x!(w-1-x)!} [p_{21}(1-\theta_1)]^x [(1-p_{21})(1-\theta_2)]^{w-1-x} = [p_{21}\theta_1 + (1-p_{21})\theta_2][p_{21}(1-\theta_1) + (1-p_{21})(1-\theta_2)]^{w-1} \tag{A.8}$$

$\Omega(w)$, for $w = 1, 2, \ldots$ 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 expression for E[T] in Section 3.3.

A.1. Waiting time distribution

At time $\tau = 0$, N_2/G_2 novel-host groups enter the system and encounter the set of patches at a combined probabilistic rate $\lambda_2 N_2/G_2$. The waiting time between consecutive visits to the set of patches has an exponential density. Let τ_v be the random waiting time between novel-host visits; the probability density is:

$$f(\tau_v) = (\lambda_2 N_2/G_2) e^{(\lambda_2 N_2/G_2)\tau_v};\ \tau_v > 0 \tag{A.9}$$

Each τ_v is independent and identically distributed.

As above, suppose that the first infection of a novel host individual occurs at the w-th visit to the set of patches. Then the waiting time for the host-jump is $T = \sum_1^w \tau_v$, and the conditional distribution of T is a gamma density:

$$f(T|w) = \left(\frac{\lambda_2 N_2}{G_2}\right)^w T^{w-1} e^{(\lambda_2 N_2/G_2)T}/(w-1)! \tag{A.10}$$

Of course, w varies randomly according to $\Omega(w)$, a geometric probability function; see Eq. (A.8). Then, unconditionally:

$$f(T) = \sum_{w=1}^{\infty} \left(\frac{\lambda_2 N_2}{G_2}\right)^w T^{w-1} \frac{e^{(\lambda_2 N_2/G_2)T}}{(w-1)!} [p_{21}\theta_1 + (1-p_{21})\theta_2][p_{21}(1-\theta_1) + (1-p_{21})(1-\theta_2)]^{w-1} \tag{A.11}$$

where θ_r, $r = 1, 2$, the probability a novel host is infected during a single patch visit, is given by Eq. (A.6). After rearrangement, we have:

$$f(T) = [p_{21}\theta_1 + (1-p_{21})\theta_2] \frac{\lambda_2 N_2}{G_2} e^{-(\lambda_2 N_2/G_2)T} \times \sum_{z=0}^{\infty} \left(\frac{\lambda_2 N_2}{G_2} T[p_{21}\theta_1 + (1-p_{21})\theta_2]\right)^z / z! \tag{A.12}$$

Then:

$$f(T) = \frac{\lambda_2 N_2}{G_2}[p_{21}\theta_1 + (1-p_{21})\theta_2] \exp\left(-\frac{\lambda_2 N_2}{G_2}[p_{21}\theta_1 + (1-p_{21})\theta_2]T\right) \tag{A.13}$$

The waiting time for host-jumping has an exponential density; the novel-host population approaches its initial infection at constant probabilistic rate $(\lambda_2 N_2/G_2)\sum_r p_{2r}\theta_r$. $f(T)$ yields the expectation given in Eq. (11). Generalizing, let T_I represent the total waiting time elapsing until I different novel-host groups have each have at least one member infected. The associated probability density is (Boswell et al., 1979):

$$f(T_I) = \binom{N_2/G_2}{I-1} [(N_2/G_2) - I + 1] \left(\lambda_2 \sum_r p_{2r}\theta_r\right) \times \left[1 - e^{-\lambda_2 T_I \sum_r p_{2r}\theta_r}\right]^{I-1} \left[e^{-\lambda_2 T_I \sum_r p_{2r}\theta_r}\right]^{[(N_2/G_2)-I+1]} \tag{A.14}$$

The waiting time for infection of every group, $T_{N_2/G_2} = T_L$, is discussed in Section 3.4.3.

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