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Mating Competition, Promiscuity, and Life History Traits as Predictors of Sexually Transmitted Disease Risk in Primates

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Abstract Competition among males influences the distribution of copulations and should therefore influence the spread of sexually transmitted diseases (STDs). We developed a model to investigate STDs in the mating and social systems found in primates, and we tested predictions using comparative methods. In the model, groups were distributed on a square lattice in which males or females disperse and males undergo characteristic dominance trajectories at maturity (challenge vs. queuing). We investigated the impact of mating rate, mating skew, migration rate of males or females, and group size on disease spread and prevalence. The model generated several

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predictions: 1) STD prevalence is higher in females than males; 2) STD risk increases with copulation rate; 3) high skew is negatively associated with STD risk; 4) STD risk is higher for all individuals when females disperse and 5) when mortality rates are lower; and 6) reproductive skew and later age of male dominance (queuing) produce more strongly female-biased STD prevalence. In comparative tests, we quantified STD risk as prevalence and richness of sexually transmitted organisms at the host species level. We found positive associations between host longevity and higher STD richness, and only (nonsignificant) weak trends for females to have higher STD prevalence. Mating skew showed a weakly positive association with STD richness, contrary to predictions of our model but consistent with predictions from a previous model. In some tests, we also found that female dispersal resulted in greater STD infection risk. Collectively, these results demonstrate that mating competition and demography influence patterns of STD infection, with mortality rates having the strongest effects in comparative tests.

Keywords Comparative study · Infectious disease · Male mating competition · Sexual selection · Sexually transmitted disease

Sexually transmitted diseases (STDs) commonly infect wild and domesticated animal populations. We define STDs as micro- or macroparasites that are transmitted through sexual contact; some of these may produce genital lesions that increase transmission risk of other STDs. STDs, often characterized by long infectious periods and low recovery rates, can impact host fitness by inducing sterility, reducing offspring survival, and selecting for costly investment in immune defenses (Lockhart *et al.* 1996; Nunn *et al.* 2000). Many STDs have been identified in primate species, including papillomaviruses on the genitals of colobus monkeys (Kloster *et al.* 1988; O'Banion *et al.* 1987), simian immunodeficiency virus (SIV) in chimpanzees and other African primates (Hahn *et al.* 2000; Keele *et al.* 2009), and treponemal disease in baboons (Harper *et al.* 2012; Knauf *et al.* 2011). The potential selective pressure imposed by STD infection is illustrated by substantial fitness effects in a number of species, including elevated mortality and reduced fecundity in SIV-infected chimpanzees (Keele *et al.* 2009) and genital lesions associated with treponemal disease that interfere with successful mating in baboons (Knauf *et al.* 2011).

Among the factors that influence STD transmission, promiscuity plays a prominent role: one would expect to find more STDs—or increased investment in defenses against these pathogens—in species characterized by greater promiscuity (Nunn and Altizer 2004, 2006). Empirical studies have linked measures of promiscuity and baseline immune defenses (Anderson *et al.* 2004; Nunn 2002; Nunn *et al.* 2000; Wlasiuk and Nachman 2010). Though some studies have found evidence for behavioral counterstrategies to STDs in mammals (Hart *et al.* 1987), behavioral defenses against STDs are largely unrelated to the risks of acquiring STDs in primates (Nunn 2003; Nunn and Altizer 2004).

Variation in male mating success (mating skew) represents another behavioral factor that underlies STD transmission dynamics. Males that experience greater mating success face higher risk of STD infection, and this may influence disease transmission within and between groups. Thrall *et al.* (2000) used an agent-based

model to explore the links between STDs and mating skew, focusing on STD prevalence in a single-male seasonal mating system in which males vary in their ability to attract females and females may switch mates between seasons (see also Ashby and Gupta 2013; Kokko *et al*. 2002; Thrall *et al*. 1997, 1998). This model predicted that higher mating skew would result in higher prevalence for both males and females, that STD prevalence would be higher in females than in males, and that this gap would increase with increasing mating skew. In addition, Thrall *et al*. (2000) investigated life history features (mortality rate) and female partner-switching rate. The results showed clear positive effects of partner-switching rate on STD prevalence, and revealed that elevated mortality generally decreased prevalence. Thrall *et al*. (2000) did not investigate other types of mating systems, such as polygynandrous systems in which both sexes have multiple partners, or male dispersal.

To test the prediction that females have higher prevalence of infection than males, Nunn and Altizer (2004) compiled published and unpublished data on STD prevalence in wild primates. They first tested whether sexually mature individuals had higher prevalence of STDs than immature individuals, as expected if the infectious agent is sexually transmitted. A paired *t*-test across studies with data on STD prevalence supported this prediction. Next, they investigated whether STD prevalence was higher in females than in males, as predicted if mating skew among males (but not females) is high, leaving more males unmated compared to females. Again, a paired *t*-test of the individual studies supported this prediction.

Though these results were consistent with predictions of the STD-mating skew model (Thrall *et al*. 2000), a number of questions and future directions remain. First, a plausible alternative explanation for sex differences in prevalence is that females are more susceptible to STDs (Nunn and Altizer 2004), as indicated by some studies of HIV in humans (Padian *et al*. 1997). However, this explanation is tempered by the potentially immunosuppressive effects of testosterone, which may elevate male susceptibility to infection, although studies of this are mixed (Muehlenbein and Bribiescas 2005; Roberts *et al*. 2004). Evidence for biased transmission is also weakened by the findings that HIV infection fails to exhibit strong sex biases in low-income countries (Boily *et al*. 2009). Indeed, when sex biases occur, STDs may show different biases owing to variation in infection routes and pathogenesis (Madkan *et al*. 2006).

Second, the Thrall *et al*. (2000) model investigated these questions in strictly polygynous (single-male) mating systems in which a subset of females disperse between seasons. However, the data on primate STDs come mainly from species in which mating systems are represented by multiple sexually active males and females (polygynandry), in which males are organized in dominance hierarchies of varying steepness and linkage to mating success. In addition, males often disperse in the species for which STD prevalence data are available. Details of male dispersal, including the age at which males disperse and their dominance rank when entering or leaving a group, may yield important insights into STD dynamics. For example, postreproductive (secondary) male dispersal could lead to increased STD spread through a population.

Here, we aim to investigate questions about the impact of mating competition in primates on STD dynamics using an integrated theoretical and empirical approach (Nunn 2012). We first developed a general model that allowed us to characterize STD dynamics in group-living animals where male mating success depends on dominance rank and the degree to which rank influences mating access, dominance rank depends

on age, and dispersal occurs between neighboring groups. Either sex may disperse, and when males disperse, low-ranking males are more likely to leave the group. We specifically considered that males of different species show different life history trajectories: in some species, younger males have the highest mating success, whereas in other species, older males have the highest mating success (van Noordwijk and van Schaik 2004). These older males have a greater risk of infection (Nunn and Altizer 2004). Our overall goal was to incorporate demographic and life history characteristics that are of specific relevance to STD transmission in primates. We expect that this general model could be usefully extended and parameterized to investigate specific host-STD systems, but our more general model is well suited to developing comparative predictions across species.

Empirically, we reinvestigated sex differences in a wide array of primate STDs using a larger dataset and a phylogenetic paired *t*-test (Lindenfors *et al*. 2007), and in new analyses, we investigated predicted drivers of infection with STDs, including host longevity and body mass (as proxies for host mortality rates) and measures of mating skew. We analyzed both STD prevalence (proportion of individuals infected) and species richness, where richness refers to the number of STDs known to infect a given primate host. Though the model mainly focused on prevalence and rates of disease spread, we expected that a more prevalent pathogen is more readily detected and reported in the literature, resulting in higher parasite richness at the species level. More generally, a parasite that is more prevalent should also better meet the conditions for parasite establishment, resulting in higher richness.

As noted previously, a key motivation for the theoretical model was to generate predictions for comparative studies in primates. Thus, we attempted to use empirically justified ranges of parameters in the model. We also aimed to identify consistent associations in spite of variation across primate species in the traits of interest. To do this we sampled variation in seven demographic, life history, and mating variables in the simulations, and we investigated associations in simulation output using both bivariate and multivariate tests. A strong association in the bivariate tests reveals that a particular parameter is strongly predictive of STD risk given variation in the other six parameters that varied across simulations.

Methods

Theoretical Model

Simulated populations were divided into mating groups of equal size and equal sex ratio organized on a square lattice. At the start of each simulation run, we randomly selected one male and one female within a single mating group to be infected. The infection was transmitted probabilistically during mating, and infected individuals remained infectious for life, with no added mortality due to disease. The simulation proceeded in discrete time steps, where a single time step corresponded to the length of a female mating cycle. Pregnancy and lactation were ignored, such that all females were assumed to mate in all mating cycles, as might occur in the context of situation dependent mating to reduce infanticide risk (van Schaik *et al*. 1999). Model parameters are summarized in Table I. In each time step of the simulation, five processes occurred in sequential order:

Table I Parameters in the simulation model

Variable	Name in R	Description
n	NumSteps	Number of time steps, each time step corresponding to a female reproductive cycle
l	LatticeSize	Dimension of landscape lattice, containing a group in each cell
n_f	N	Number of females per group. Number of males equals number of females, and total group size is therefore $2N_f$.
λ	KokkoLambda	Value of Kokko's mating skew parameter, takes values greater than 0 and less than or equal to 1, with 1 corresponding to maximum skew
m_s	MigratingSex	Female migration = 0, male migration = 1
m_r	MigrationRate	Constant per time-step probability of migration for the migrating sex
k	CopulationRate	Average number of times a female copulates per cycle
p	PeakAge	Male ranks are formed based on closeness in age to PeakAge, represented as a proportion of age to account for variation in longevity.
d	MortalityRate	Constant per time-step probability of mortality
β	m2f and f2m	Per-mating transmission probability, sex-specific in model but set equal in simulations reported here
r	RandSeed	Number of random initial infections

1) Mating: We assumed that male rank predicts mating success according to the model of mating skew developed in Kokko and Lindstrom (1997), and described by the equation:

$$c_r = \frac{\lambda(1-\lambda)^{r-1}}{1-(1-\lambda)^n}$$

where c_r describes the expected *proportion* of copulations obtained by a male of rank r in a group with n males. This is equivalent to a geometric distribution with the shape parameter λ. In this context, λ is the "mating skew" parameter: it varies from 0 to 1, with values of 0 corresponding to no mating skew (all males have an equal probability of mating, resulting in completely promiscuous mating), and values of 1 corresponding to maximum mating skew (one male monopolizes access to females). The number of copulations that occurred within a group in a given time step depended on both c_r and the characteristic rate of copulation for females, k. The expected number of copulations with each female, C_r, for males of rank r was:

$$C_r = c_r k$$

In each time step, the number of copulations each male obtained with each female was drawn from a Poisson distribution with mean C_r.

2) Disease transmission: Mating between an infected and uninfected individual resulted in a probability of disease transmission. Here, we focused on symmetrical transmission, notated simply as β. We opted for symmetric sexual transmission because sex biases probably depend on the pathogen involved. In addition, sex biased prevalence in the absence of sex-biased transmission provides strong

evidence that mating system, life history and behavior influence STD dynamics independently of any transmission biases. Infected individuals become infectious in the time step after they become infected and never recover, reflecting that many sexually transmitted organisms show lifelong infections (Lockhart *et al*. 1996). A newly infected individual was unable to transmit infection in the same time step as infection.

3) Dispersal: Males or females dispersed between groups. For female dispersal, we assumed that all females have an equal probability of migrating (m_r) in a given time step. For male dispersal, we assumed that m_r is higher for males that have less access to females. Specifically, we assumed that m_r decreases geometrically with rank,

$$m_r = \frac{\lambda(1-\lambda)^{r-1}}{1-(1-\lambda)^n}$$

where λ is the mating skew parameter. Thus, higher levels of mating skew led to greater biases toward migration by lower-ranking males, and for top-entry systems, older males were more likely to disperse (after they already have had high mating success). To keep sex ratios and group sizes constant, migrations were modeled as swaps between pairs of neighboring groups. In each time step, the number of swaps was drawn from a Poisson distribution, with mean equal to $n_f\, m_r/2$ (we divided by two because each swap was equivalent to two migration events, and with the number of males equaling the number of females, the equation can apply to either male or female dispersal). Once the number of swaps was determined, pairs of adjacent groups were chosen randomly, and one individual from each of those groups was randomly selected to migrate according to the probabilities described above. To avoid edge effects, migration was modeled on a torus.

4) Population turnover: Across the population, births and deaths were modeled by applying mortality probability d to individuals in every time step, resulting in an average lifespan of $1/d$. On death, healthy, adult individuals of the same sex immediately replaced deceased individuals; thus, we implicitly assumed density dependent mortality.

5) Setting dominance rank based on age: Within groups, males were ranked based on closeness in age to a user-specified target age (p) of peak rank, where p was measured relative to the lifespan used in the simulation. Male rank covaried with mating success. The male closest in age to p was highest ranking, the male second closest in age to p was the second-ranking, and so on. Ties in age were resolved randomly to produce a linear hierarchy of individuals with distinct ranks. We thus simulated the alternative scenarios of young males starting out at the bottom and increasing in rank with age (queuing or "bottom-entry," as in rhesus macaques, with high p), or starting off at the top of the dominance hierarchy and declining in rank with age (challenge or "top-entry," as in savanna baboons, with low p). We assumed that these two dominance trajectories are unrelated to variation in sex-biased dispersal (in both examples just given, males disperse).

By using this particular sequence of events, we are assuming a population of organisms in which major life events occur during specific times of year, as might

occur in a seasonally breeding species. We thus make the reasonable assumption that a mating season leads to disease transmission, that dispersal occurs before the next mating season, that deaths occur predominantly outside the mating season during a seasonal shortage of resources, and that dominance rank is then set amongst the males that survive the lean season just as the next mating season starts. Though changing the order of events in the simulation could conceivably change the outcomes, the order used here is generally reasonable, and for some events would not be sensible otherwise, e.g., to have disease transmission occur before mating.

We considered two types of output from the model. First, equilibrium *population prevalence* was estimated as the proportion of individuals infected in the final time step of a simulation. We ensured that the simulation was run sufficiently long to provide a valid estimate of equilibrium prevalence (see Electronic Supplementary Materials). Second, we considered a measure of the proportion of groups in which the disease has established at a given time point, or *group prevalence*. We recorded group prevalence at time steps 20, 50, 100, and 1000. Unless stated otherwise, "prevalence" refers to population prevalence. We also recorded prevalence for males and females separately, with sex differences in prevalence calculated as female prevalence minus male prevalence (and thus reflecting female biased infection when the difference was positive).

Model Input and Analysis

To investigate the impact of dispersal and mate competition on disease dynamics, we ran 600 simulations in which we sampled variation in seven input parameters involving demography, life history, and mating behavior using random sampling (Table II). Sampling was conducted using a Latin hypercube design, which is a type of stratified Monte Carlo sampling that has been used in epidemiological modeling and is more efficient in this context than completely random or full sampling (Blower and Dowlatabadi 1994; Nunn *et al.* 2009; Rushton *et al.* 2000; Seaholm *et al.* 1988). The Latin hypercube sample (LHS) was obtained using the R package tgp (Gramacy 2007; Gramacy and Taddy 2012). Table II provides the ranges of variation that were sampled for the parameters. Parameters that required integer (n_f) or discrete values for the model

Table II Parameters used in Latin hypercube sample and focused analyses

Variable	Minimum	Maximum	Fixed in focused
n_f	2	30	18
λ	0.01	1	0.5
p	0.05	0.7	0.4
m_s	Female (0)	Male (1)	Varied
m_r	0.01	0.2	0.05
k	1	100	40
d	0.02	0.33	0.1

The following variables were fixed in the Latin hypercube sample: $\beta = 0.02$, $n = 1500$, and $L = 8$ (producing 64 social groups). For m_s, one-half of simulations had female only dispersal, and one-half had male-only dispersal

(m_s) were represented as continuously varying in the LHS and then averaged to integer units required by the simulation model.

When analyzing the output from the model, we refrained from using frequentist statistical tests of null hypotheses, such as *P*-values, because significance levels are sensitive to sample size (and more simulations can be run to increase significance). In addition, many parameter combinations resulted in extinction of the STD (256 of 600 simulations) or very low equilibrium prevalence. This produced a bimodal distribution of prevalence in which about one half of the simulations clustered around zero (Fig. 1), which proved difficult to transform to meet the assumptions of standard statistical approaches.

Thus, to gauge which effects were the strongest for comparative tests, we conducted three analyses of the simulation output. First, we simply plotted the association between pairs of variables and fit a lowess curve using functions in R (R Development Core Team 2009). We fit curves for all data, and for cases in which prevalence was nonzero (to reflect cases when the pathogen was able to persist). Second, we estimated Kendall's τ for the sample plots, and in some cases for subsets of the data in which prevalence was > 0. Finally, we calculated the partial rank correlation coefficient, r_p, as this helps to deal with nonlinear relationships and other assumption violations (Blower and Dowlatabadi 1994). These calculations were done in R using the "sensitivity" package (Pujol *et al.* 2014). The r_p coefficient is a nonparametric measure of association that examines the effect of pairs of variables while controlling for other variables. In all cases, we focused on the strongest associations in the graphical output and nonparametric tests.

In addition to the LHS analysis, we undertook additional simulations that focused on changing a pair of variables —one of which was always the migrating sex— holding other variables constant.

Phylogenetic Comparative Tests

We conducted phylogenetic comparative analyses to investigate predictions that arose from our model or earlier modeling effort (Thrall *et al.* 2000), as detailed later. More closely related species are likely to exhibit more similar trait values, creating statistical problems of non-independence (Garland *et al.* 2005; Harvey and Pagel 1991; Nunn 2011). When this non-independence is ignored, statistical tests of association may be

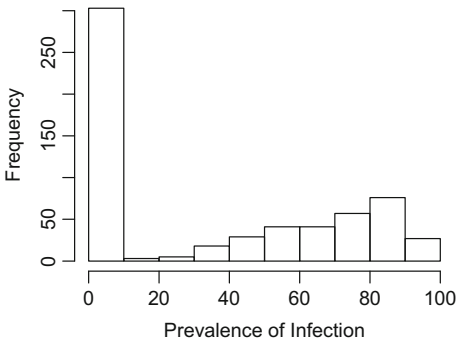

Fig. 1 Frequency distribution of prevalence (percentage of individuals infected) across 600 simulations.

invalid, resulting in higher than expected type I and II error rates (Martins and Garland 1991). We therefore used phylogeny-based comparative methods to test the comparative predictions and to quantify the degree of phylogenetic signal in residuals from our regression model (Freckleton *et al*. 2002; Revell 2010).

To conduct the comparative tests, we performed Bayesian Markov chain Monte Carlo (MCMC) analyses to generate a posterior probability distribution of statistical parameters, focusing in particular on regression coefficients. These coefficients are thus sampled in proportion to their probability. The MCMC approach controls for phylogenetic uncertainty by integrating the statistical results across a sample of trees (Pagel and Lutzoni 2002; Pagel and Meade 2006), and it gives easily interpreted support values that can be compared across different analyses. In this case, we focus on the proportion of time that a regression coefficient was in the predicted direction.

The MCMC analysis simultaneously quantifies phylogenetic signal by estimating the branch length scaling parameter λ_p (Freckleton *et al*. 2002; we use the subscript p to distinguish it from Kokko's λ used in the context of mating skew). When $\lambda_p = 0$, all internal branches are set to 0, resulting in a "star phylogeny" with all branches emanating from a common node (Felsenstein 1985). Increasing phylogenetic non-independence is modeled as λ_p increases to a value of 1. In addition to assessing phylogenetic signal of the residuals from the statistical model used to test predictions, we estimated λ_p in the estimates of prevalence used in these analyses. Most biological traits show evidence for phylogenetic signal (Freckleton *et al*. 2002; Blomberg et al. 2003). Although it is unclear if prevalence —a "trait" that varies greatly across spatial and temporal scales— also shows phylogenetic signal, we estimated λ_p to investigate phylogenetic variation and as a statistical precaution against the dangers of non-independence in comparative analyses (Felsenstein 1985).

For Bayesian estimation of parameters, we sampled regression coefficients, λ_p, and other parameters every 100 generations. We examined the distribution of likelihoods to ensure that the statistical model had converged on a stable distribution and that sampling rate was adequate. We sampled statistical parameters only from the post-burn-in sample, where burn-in was 100 generations. All hypotheses were tested using posterior probability distributions of ≥2000 estimates.

In tests of sex and age differences, we used a phylogenetic paired *t*-test, which also enables estimation of λ_p (Lindenfors *et al*. 2007). Nunn and Altizer (2004) did not incorporate phylogeny into their paired *t*-test, and a more recent study found that type I error rates in this test may exceed 70% when phylogeny is ignored (Lindenfors *et al*. 2007). Thus, it is plausible that the significant result pertaining to sex differences was a false positive in Nunn and Altizer (2004). The phylogenetic paired *t*-test was implemented in the "phytools" package (Revell 2012) of R.

To incorporate phylogenetic uncertainty, we used a sample of 100 dated trees from Version 3 of the *10kTrees* website (Arnold *et al*. 2010). This online resource provides up to 10,000 trees from a Bayesian posterior probability distribution. By using multiple inferences of phylogeny, our results are less sensitive to phylogenetic uncertainty for the primates in our sample, including uncertainty related to topology or branch lengths (Pagel and Lutzoni 2002).

Data on parasites of wild primates came from the *Global Mammal Parasite Database* (GMPD; Nunn and Altizer 2005). For each parasite or infectious disease reported, the type of parasite was recorded in the database (virus, protozoan, fungus, arthropod,

helminth, bacterium), along with parasite genus and species names, host genus and species names (later revised according to Corbet and Hill, 1991), transmission mode, prevalence of infection, and sample size. Here, we focused on sexually transmitted parasites.

For studies of prevalence, data were available on a total of 47 primate species, with data on sex differences for 54 studies of 16 primate species. These studies were not limited to studies that found an STD; in fact, 13 of the 47 species (28%) were recorded as having zero prevalence (i.e., the pathogen was looked for but not found). For analyses of richness, we included hosts if they were in GMPD and we had information on mating or reproductive skew, resulting in a dataset with 33 primate species, each having 0–6 STDs. Parasites with clear synonyms were collapsed into one species.

Estimates of mating skew were updated from Kutsukake and Nunn (2006), while data on reproductive skew were updated from Ostner *et al*. (2008). Data on group size and body mass come from Nunn and van Schaik (2002), data on longevity from Ross and Jones (1999), data on testes mass and male body mass from Harcourt *et al*. (1995) and other sources (Dixson and Anderson 2004; Nunn *et al*. 2000), and data on dispersal from an unpublished comparative dataset. Data on promiscuity were taken from van Schaik *et al*. (1999), with other species added to the data of van Schaik *et al*. from additional sources of information, including testes residuals. Host species that are studied more intensively have more parasite records in the *Global Mammal Parasite Database* (Nunn and Altizer 2005). Following previous research that used this database (Ezenwa *et al*. 2006; Lindenfors *et al*. 2007; Nunn *et al*. 2003), we obtained citation counts for each host species as a measure of sampling effort. For these analyses, we used Primate Information Network's "PrimateLit" bibliographic database (http://primatelit.library.wisc.edu/), accessed in May 2010, because it includes journals, books, and book chapters (thus matching the types of sources also included in the primate portion of the *Global Mammal Parasite Database*). The comparative data are provided as electronic supplementary files.

Results

Simulation Model: Latin Hypercube Sample

Across all 600 simulations, the mean population prevalence was 34.0% and median prevalence was 6.7% (substantially lower due to the bimodal distribution in Fig. 1). Across all simulations, females exhibited slightly higher prevalence than males (35.0% vs. 33.0%). Removing cases in which the STD failed to persist, mean prevalence was again higher in females (61%) than in males (57.5%; for median prevalence, the female-to-male comparison was 71.4% vs. 63.7%). Maximum prevalence in the 600 simulations was 97.4% of the population infected. Similar patterns were found when examining group prevalence. At time step 100, a mean of 41.6% of groups were infected based on this measure, but this actually reflects a bimodal outcome in which 188 simulations already had group prevalence of 1, and 246 had group prevalence of 0, leaving only 166 simulation runs with group prevalence between 0 and 1.

For each of the parameters varied in the LHS, we examined how variation in that parameter covaried with population prevalence, prevalence in males, prevalence in females, group prevalence,

and sex differences in prevalence. Population prevalence and group prevalence were correlated (τ = 0.79), and similar patterns were found for other measures of prevalence. For simplicity, we focus most of what follows on population prevalence. The three strongest findings involved copulation rate, mortality rate, and reproductive skew.

Copulation rate (k) had a strong positive effect on population prevalence (τ = 0.29, r_p = 0.58, Fig. 2) and group prevalence (τ = 0.31, r_p = 0.53), as expected because this increases the contact rate and potential for the STD to spread. Importantly, our plots separate the effects for all simulations, and for simulations in which the pathogen persisted, i.e., did not go extinct. Thus, the dashed lines indicated the subset of simulation runs in which only nonzero prevalence outcomes were used. Unless stated otherwise, the statistics in the text involve the full dataset.

The mortality rate (d) also had a strong negative effect on disease risk, with increasing probability of death strongly reducing population prevalence (τ = −0.44, r_p = −0.74; Fig. 3) and group prevalence (τ = −0.33, r_p = −0.60). Mortality effects were especially strong when controlling for other variables with r_p. This finding fits with theoretical expectations, as a higher rate of population turnover clears infections and reduces prevalence (Anderson and May 1991).

For mating skew (λ), we found negative associations with prevalence, especially when including cases of zero prevalence (τ = −0.24, r_p = −0.57; Fig. 4a), and slightly stronger in males (τ = −0.26) than in females (τ = −0.21). As a result, higher λ was weakly associated with more female-biased STD infection rate (τ = 0.10, r_p = 0.12), and stronger when restricted to simulations with nonzero prevalence (dashed line in Fig. 4b, τ = 0.20). Thus, when STDs exist in a population, we expect greater female-biased prevalence as mating skew increases.

For male life history (p, "challenge" vs. "queuing"), we found no indication that prevalence covaries with p (τ = −0.017); this was true even for males, which is the sex directly affected by p (τ = −0.047). A stronger result was found in terms of female sex-biased prevalence with increasing p (τ = 0.33, r_p = 0.49; Fig. 5). This makes sense, because at higher p a larger proportion of males dies before reaching the age associated with higher rank, i.e., older males have higher mating success, whereas the same effect would not be found in females, given that their mating success is independent of rank.

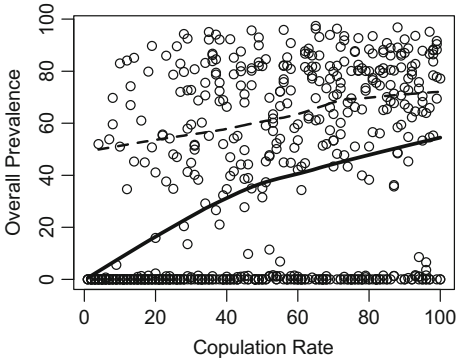

Fig. 2 Relationship between copulation rate (k) and population prevalence. Solid line indicates the lowess curve across results from all simulations. Dashed line indicates the relationship using only the output where prevalence was > 0 at the end of the simulation.

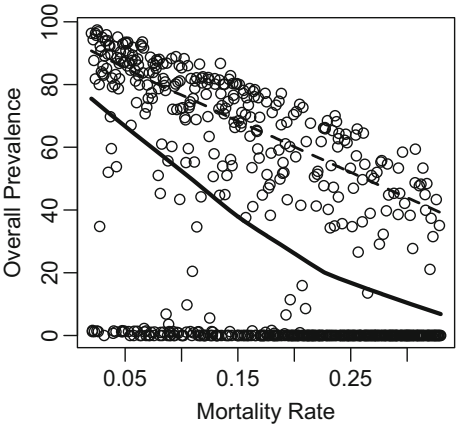

Fig. 3 Relationship between mortality rate (*d*) and population prevalence. Solid line indicates the lowess curve using results from all simulations. Dashed line indicates the relationship using only the output where prevalence was > 0 at the end of the simulation.

For group size (n_f), we found a weak negative association with population prevalence ($\tau = -0.091$, $r_p = -0.22$). Migration rate (m_r) also had very weak effects on prevalence, with Kendall's τ and r_p very close to 0 ($\tau = -0.002$ and $r_p = 0.05$).

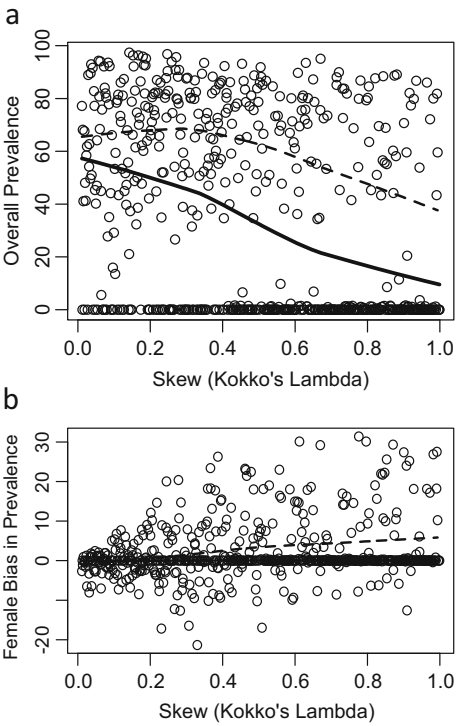

Fig. 4 Relationship between reproductive skew (Kokko's λ) and **(a)** population prevalence and **(b)** sex differences in prevalence (female prevalence minus male prevalence). Solid line indicates the lowess curve describing the relationship across all simulations. Dashed line indicates the relationship using only the output where prevalence was > 0 at the end of the simulation.

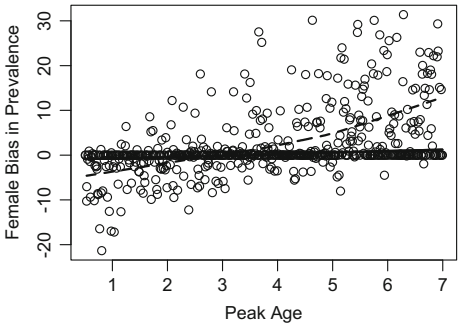

Fig. 5 Relationship between peak age of domiance (p) and sex-biased STD prevalence. Solid line indicates the lowess curve describing the relationship across all simulations. Dashed line indicates the relationship using only the output where prevalence was > 0 at the end of the simulation.

Migration rate affected the rate of spread based on group prevalence, but only weakly, and only when examining measures of group prevalence early in the spread of infection, e.g., at time step 25: $\tau = 0.054$, $r_p = 0.17$.

More interesting effects were associated with which sex migrated (m_s). When males migrated, disease prevalence was lower ($\tau = -0.15$, $r_p = -0.25$), probably because migration is more likely for lower ranked individuals with correspondingly fewer opportunities to mate. Thus, many of these migrating males would have had less exposure to the STD. This is explored further in the next section.

Simulation Model: Focused Analysis

To investigate further the drivers of infection dynamics, we ran simulations in which all but two parameters were fixed. Figure 6a shows the effects of mating skew (λ) and dispersal of males versus females (m_s) on prevalence (other parameters were "fixed"; see Table II). Prevalence clearly declines with λ, but with a striking difference depending on male vs. female dispersal. Specifically, we observed an earlier (and nonlinear) decline when males disperse (similar patterns were found for group prevalence). Given that dispersal is based on rank, and here, $p = 0.4$ (Table II), this pattern reflects that a larger proportion of dispersing individuals are low ranking males with (on average) lower mating success at higher λ, and thus less exposed to the STD. Although there will also be some males older than p that have had mating success and are now declining in rank, mortality (d) thins this class of males, resulting in lower overall prevalence of infection in dispersing males as λ increases. In other words, the results depend on both p and d, emphasizing the need to run broader random sampling, as we did with the LHS above.

We also found a strong sex difference, except under male dispersal when λ exceeded about 0.5, where the infection was unable to establish (Fig. 6b). This likely occurred because low-ranking males were more likely to disperse, and high-ranking males were more likely to mate and thus be exposed to the STD. In comparison to the LHS results shown in Fig. 4b, the pattern emerging in Fig. 6 is clearer: overall STD risk covaries negatively with skew, female-biased prevalence exists and covaries positively with skew when the STD is maintained in a population, and female-biased dispersal modifies these patterns.

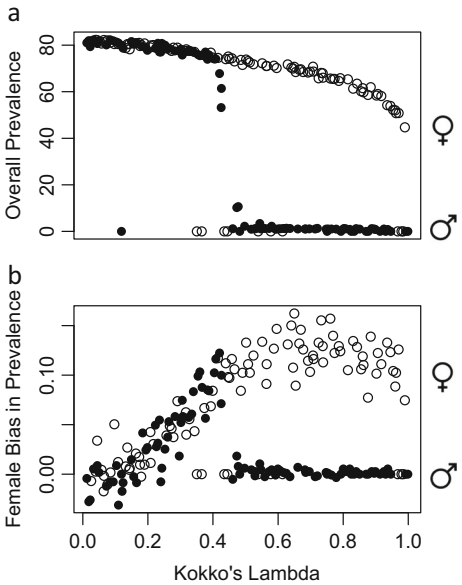

Fig. 6 Effects of sex-biased dispersal and mating skew (λ) on **(a)** population prevalence and **(b)** sex differences in prevalence (calculated as female prevalence minus male prevalence). Filled circles indicate male dispersal. Open circles indicate female dispersal.

We also ran analyses varying female group size (n_f), peak age (p), migration rate (m_r), copulation rate (k), and mortality rate (d), while including sex-biased dispersal as a random variable. Graphical results for overall prevalence are provided in online supplementary figures. These results largely corroborate previous findings from the LHS and further highlight the importance of sex biased dispersal on infection patterns. Thus, female group size has weak effects on prevalence, with a hint of a negative association (Fig. S1); peak age affects sex differences in prevalence (Fig. S2); migration rate has weak effects, except possibly at higher rates of movement between groups when males disperse (Fig. S3); finally, copulation rate and mortality rate have strong effects on prevalence, in opposite directions (Figs. S4 and S5).

Comparative Predictions

Our model makes several predictions. For reasons given earlier, our predictions involve both prevalence and richness as outcome variables, with both expected to increase based on the simulation results (which focused on prevalence).

1) Prevalence should be higher in females than in males. We found a weak sex difference in our model, whereas a stronger sex difference was predicted from the "polygyny" model of Thrall *et al.* (2000).
2) Prevalence and richness of STDs are expected to increase with copulation rate.
3) Prevalence and richness of STDs should decrease with male mating skew based on our model. However, the model of Thrall *et al.* (2000) predicts a positive association.

4) Prevalence and richness of STDs should be higher when females disperse, especially in species with exclusive female dispersal; i.e., males are philopatric.
5) Prevalence and richness should also increase with lower mortality rates, i.e., a slower life history and longer lifespans, as here we are focused on background mortality rather than disease-induced mortality.
6) Female-biased prevalence should increase with skew.

Comparative Tests

To investigate Prediction 1, we compiled data on prevalence of infection in males and females averaged to the species level. The average prevalence in females (34.8%) was slightly higher than in males (32.3%), a difference of only 2.5 percentage points. In a phylogenetic paired t-test, we found no evidence for a sex difference ($t_{15} = -0.45$, $P = 0.66$), and also no evidence for phylogenetic signal (maximum likelihood estimate of $\lambda_p = 0$). In contrast, when testing for age differences, we found a strong difference between immature animals (prevalence = 11.2%) and mature animals (prevalence = 40.0%) in a paired *t*-test ($t_{15} = 5.75$, $P < 0.0001$), again with no evidence for phylogenetic signal ($\lambda_p = 0$). Based on the low phylogenetic signal, we also investigated sex and age differences by study in a standard paired *t*-test. This analysis again revealed slight sex differences (42.1% in females vs. 37.7% in males), including when restricting the analysis to the two major STDs examined separately (SIV: 39.8% in females, 32.2% in males; STLV: 38.7% in females, 32.2% in males). As with the phylogenetic tests, these differences were not statistically significant (e.g., all STDs: $t_{53} = 1.52$, $P = 0.14$). Strong differences among age classes were again found on the sample-by-sample basis (e.g., all STDs: prevalence of 50.0% in adults vs. 17.3% in immature individuals, $t_{53} = 9.53$, $P < 0.0001$).

To test prediction 2, we used measures of testes mass (controlling for male body mass) as a proxy for copulation rate; we also used data on whether females mate with one or many males, which likely covaries with copulation rate but also reflects variation in mating skew (Prediction 3). We found no compelling associations involving STD richness, although weak effects were found in analyses of prevalence, with support levels in the range of 84–90% for a positive regression coefficient (Table III; support levels indicate the proportion of coefficients sampled in the MCMC analysis that are in the predicted direction). By comparison, the regression coefficient for citation counts was strongly supported as a positive predictor of parasite richness (support levels >95%), as expected given that more parasites and pathogens should be found when a species is studied in greater depth (Cooper and Nunn 2013; Nunn *et al*. 2003). Phylogenetic signal was intermediate. These findings were also consistent when analyzing testes residuals, and when using a larger sample of species in analyses of prevalence (Fig. 7, support level of 81.4% for a positive regression coefficient).

Prediction 3 involved two measures of skew: λ and the proportion of matings by the male with the most matings in a group ("maximum proportion matings"). Although not included in our simulations, we used maximum proportion of matings as a comparable measure of skew that covaries with λ (Kutsukake and Nunn 2006). Using estimates of λ as a predictor, we found hints of a positive relationship, with support levels at 84.9% (Table IV). Using maximum proportion of matings, we found support of 90.2% in

Table III Analyses of promiscuity (prediction 2)

Analysis	Promiscuity variable		Citation counts		Phylogenetic signal	Sample size
	Coefficient	Support (positive, %)	Coefficient	Support (positive, %)	mean λ_p	
Richness ~ testes + citations	−0.099	32	0.00072	100	0.62	22
Prevalence ~ testes	0.061	89.3	—	—	0.38	16
richness ~ promiscuity + citations	0.0084	53	0.17	97.8	0.54	33
Prevalence ~ promiscuity	0.032	84.5	—	—	0.41	23

analyses of maximum proportion matings as a positive predictor of STD richness. Thus, results involving λ were more consistent with the Thrall *et al*. (2000) model than the one developed here (although Fig. 7 suggests higher prevalence when skew is low). Again, citation counts were strongly predictive of the number of STDs recorded for a species (Table IV). However, prevalence was unrelated to λ and the maximum proportion of matings, with an approximately even number of positive and negative regression coefficients for this variable (Table IV). Phylogenetic signal was intermediate, but higher in analyses of richness than of prevalence. In analyses based on reproductive skew, results for STD richness and λ were weaker than those based on mating skew (60.3% support for positive λ), even when expanding the dataset by using reproductive skew in place of mating skew for two species without the latter measure (61.2% support for positive λ).

Prediction 4 involved sex-biased dispersal, which we examined with predictors involving female dispersal (regardless of whether males disperse) and exclusive female dispersal (when males are philopatric). We found some weak evidence for the predicted effect in two of the four tests that were run, involving both richness and prevalence (Table V). As with other tests, citation counts were strong predictors of STD richness.

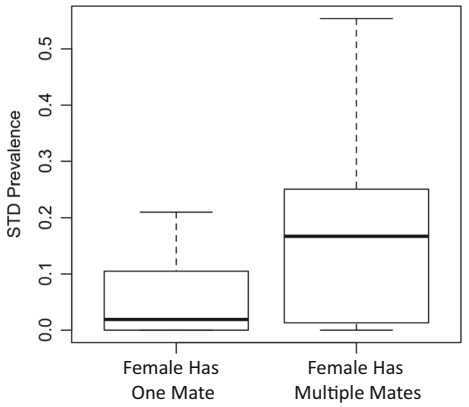

Fig. 7 STD prevalence in relation to female mating patterns. Data on mating patterns are mainly from van Schaik *et al*. (1999) (see Methods).

Table IV Analyses of mating skew (prediction 3)

Analysis	Skew variable		Citation counts		Phylogenetic signal	Sample size
	Coefficient	Support (positive, %)	Coefficient	Support (positive, %)	mean λ_p	
Richness ~ λ+ citation counts	0.69	84.9	0.19	98.9	0.59	28
Prevalence ~ λ	0.0064	51.8	—	—	0.41	19
Richness ~ maximum mating + citations counts	0.92	89.7	0.18	98.2	0.61	31
Prevalence ~ maximum mating	−0.012	47.3	—	—	0.36	21

Phylogenetic signal was again intermediate and slightly higher in studies of richness than of prevalence.

Prediction 5 focused on the effects of mortality, which were estimated in our comparative tests by using longevity and body mass, the latter standing in as a general proxy of a slow life history and a variable that was available for all species in our dataset (thus increasing sample size). As predicted, we found (Table VI) that longer-lived primate species have more STDs documented, after controlling for sampling effort (which was again supported as a predictor in the models). However, lifespan and body mass did not predict prevalence of infection across species.

Finally, for Prediction 6, we investigated sex-biased infection patterns. With the caveat that sample sizes were small, the model's prediction was unsupported: the coefficient describing the relationship between λ and sex-biased prevalence was positive, but weakly so, with only 59% of the coefficients positive in the posterior probability distribution.

We also ran multivariate models of STD richness and prevalence that included as predictors one of the variables used in each of Predictions 2–5, favoring the variable with largest sample size. These predictors thus included: body mass, promiscuity codes, mating skew, female dispersal, and citation counts (for analyses of richness). We used

Table V Analyses of female dispersal (prediction 4)

Analysis	Dispersal variable		Citation counts		Phylogenetic signal	Sample size
	Coefficient	Support (positive, %)	Coefficient	Support (positive, %)	mean λ_p	
Richness ~ female dispersal + citation counts	0.16	87.5	0.21	99.2	0.61	33
Prevalence ~ female dispersal	0.0029	52	—	—	0.39	23
Richness ~ exclusive female dispersal + citations counts	0.12	81	0.20	99.1	0.54	33
Prevalence ~ exclusive female dispersal	0.052	90	—	—	0.36	23

Table VI Analyses of mortality rates, proxied by host longevity and body mass (prediction 5)

Analysis	Mortality-related variable		Citation counts		Phylogenetic signal	Sample size
	Coefficient	Support (positive, %)	Coefficient	Support (positive, %)	mean λ_p	
Richness ~ lifespan + citation counts	0.75	97.4	0.15	90.4	0.63	28
Prevalence ~ lifespan	0.079	80.5	—	—	0.36	20
Richness ~ body mass + citations counts	0.30	95.7	0.13	95.2	0.42	33
Prevalence ~ body mass	0.0071	56.5	—	—	0.35	23

Bayesian model selection procedures in which inclusion of a particular variable was part of the MCMC analysis, resulting in models with 0–5 predictors. Support for a variable is therefore indicated by its inclusion in the model, and when included in the model, by a preponderance of coefficients in the predicted direction (for all variables, we predicted positive relationships).

In analyses of STD richness, the regression models in the posterior distribution contained, on average, 1.7 variables. Results are summarized in Table VII. Female mass and Kokko's λ were most commonly included in the model (59% and 53% of models, respectively). Of these, female mass showed strong support for a positive effect on STD richness, with 96.7% of the regression coefficients being positive. Support for Kokko's λ was weaker but indicative of a trend, with 84% of the coefficients being positive. Citation counts were included less often than female mass and λ, but when included, strong support emerged for a positive association with STD richness (95.2%). Female dispersal and level of female promiscuity were occasionally included, but showed no clear trend toward having a positive or negative effect on STD richness, with support values close to chance.

The predictor variables had less traction in predicting prevalence (Table VII). On average, less than one variable was included in the sampled statistical models (mean of 0.37), indicating weak support for any of the variables as predicting prevalence (and thus consistent with bivariate tests).

Table VII Multivariate prediction of STD richness

	Richness			Prevalence		
	Coefficient	% models included	Support (positive, %)	Coefficient	% models included	Support (positive, %)
Female mass	0.21	59	96.7	0.00054	6.4	57.0
Kokko's λ	0.34	53	84	−0.0011	21.3	49.3
Citation counts	0.062	35	95.2	n/a	n/a	n/a
Female dispersal	0.0051	14	56	−0.00071	4.0	27.5
Promiscuity codes	0.00093	11	54	0.0017	4.9	81.6

Discussion

Our approach involved building a model based on knowledge of primate behavior, using that model to generate predictions, and then testing those predictions (Nunn 2012). From this model, we predicted that STD risk covaries especially with host longevity, promiscuity, sex-biased dispersal, and mating skew, although support for these different predictions varied in strength. Empirically, we tested predictions involving STD risk using measures of both prevalence and species richness, with the latter measure based on the reasonable expectation that higher prevalence in the model indicates better chances of parasite success and thus higher STD richness. In addition, if STDs are at higher prevalence, researchers are more likely to detect them in wild populations, resulting in higher estimates of richness. Our comparative tests were mixed, but provided most compelling evidence for the effects of mortality rate, measured via data on longevity and body mass. This effect is driven by the fact that infections are often long lasting, and mating opportunities (and thus chances to obtain STDs) accumulate over the lifespan. We also often found one piece of weaker evidence in favor of each of the other variables (but inconsistent across tests of a given prediction).

It is important to understand interactions between social behavior and disease dynamics from several viewpoints. These include epidemiological, conservation, and evolutionary perspectives. Previous research suggests that host behavior influences disease risk (Nunn and Altizer 2006). However, real potential exists for pathogens to influence the evolution of host traits, including social behavior or mating behavior (Kokko *et al*. 2002). To make sense of the diversity of social structures, it is important to factor in disease as a potential causal factor (Altizer *et al*. 2003).

Results from our model demonstrate several ways in which host behavior can drive disease dynamics and persistence, often in interaction with other key demographic and developmental behaviors. For example, model output suggested that female preferences for particular males may influence patterns of STD risk (see also Ashby and Gupta 2013; Kokko *et al*. 2002; Thrall *et al*. 2000) , especially in interaction with patterns of female dispersal and male queuing, i.e., the parameter p in our model. Thus, when males disperse and hit peak mating rates at mid-life or later, high levels of male skew actually tend to reduce disease risk, with the majority of the dispersing males actually unmated and thus uninfected. We investigated these important effects of sex-biased migration on disease spread using more focused analyses.

In the immediate context of this work – and given that it is grounded in empirical studies of wild primate populations – these results help us understand different patterns of infection across host species with varying mating systems. In a practical sense, the results identify species most likely to be in high infection risk categories, and potentially in need of conservation action. Real potential exists for such threats. For example, Knauf *et al*. (2011) described high prevalence of a treponemal disease in baboons that, in severe cases, caused necrosis of the genitalia and probable reductions in reproductive success. Such a situation obviously affects mating, and even with low skew, infected males would have low mating success. The results also help to identify species that may serve as future sources of emerging diseases in humans, and to design control strategies in wild or captive populations (Knauf *et al*. 2013).

Here, we made a simplifying assumption that the number of males equals the number of females. In addition, while many males do not mate in a given time step

in our model, our approach captured lifetime mating success, in which many of the males will have mating opportunities at some point in their lives. In contrast, the Thrall *et al.* (2000) model assigns a value of mating success to a male for his whole lifetime; a male's "quality score" never changes, resulting in higher lifetime variance in mating success and producing stronger sex differences. In real animal populations, the number of males is often lower than the number of females in a group, with male–male competition resulting in higher mortality and extra-group males, including males that will eventually immigrate into a group. This variation in group composition could be incorporated in future modeling effort.

Future models could also include coevolutionary interactions involving changes in host behavior and social group structure, and variation in pathogen characteristics, e.g., transmission mode, infectivity, virulence. Some earlier studies (Thrall *et al.* 1997) indicated that optimal mating strategies for hosts or optimal virulence levels for pathogens are not straightforward to predict (but that evolutionary impacts are definitely possible). Our model had no disease-induced mortality —consistent with a study of STDs Lockhart *et al.* (1996)— and infected individuals did not suffer reductions in mating success, as might occur for STDs that cause genital lesions (such as *Treponema*). However, these impacts are likely in real populations, resulting in coevolutionary dynamics.

Building on our current effort, future studies could also help to predict patterns of parasite community diversity and structure across different hosts. This is related to the idea of assembly rules and pathogen life histories that are preadapted to invade and persist in a given host social system. Lockhart *et al.* (1996) and Thrall *et al.* (1998) discuss the kinds of traits that might favor STDs vs. infectious agents transmitted via other routes (see also Ashby and Gupta 2013). Another future direction for empirical research is to examine prevalence in more controlled settings, such as captive groups of primates.

Additional extensions for future modeling research include models that allow social groups to vary temporally in size based on local carrying capacity, and including distance-dependent dispersal scenarios, variable mortality for those in groups compared to those dispersing, vertical transmission from mother to offspring, and better incorporation of variation in group composition. We fixed the transmission probability (β) because few if any data are available on variation in transmission probability for primate STDs in the wild, and sex differences may exist (requiring two parameters). We also maintained a constant size among the groups in the population, had a constant number of groups arranged in a square matrix, and implemented dispersal as occurring only between neighboring groups. Any and all of these parameters could be varied to investigate their effects. For example, having fewer groups or enabling longer-range dispersal would potentially reduce the effects of space on disease dynamics, and would likely result in higher prevalence. Similarly, it would be possible to include population dynamics in future models.

Finally, it is worth considering the similarities and differences in predictions in our model, as compared to other models (Ashby and Gupta 2013; Thrall *et al.* 2000). In general, we see many similarities in the findings among models. For example, Ashby and Gupta (2013) also investigated links between female dispersal and STD prevalence. Similarly, Thrall *et al.* (2000) found strong effects of mortality, with higher mortality generally reducing prevalence. In their case, however, prevalence actually increased at intermediate levels of mortality, due to the dissolution of groups and

increased mixing as females moved among groups when the male of the group died (see also Nunn *et al*. 2008).

Differences also existed among the models. While the Thrall *et al*. (2000) and Ashby and Gupta (2013) studies predicted a positive association with mating skew, our model predicted a negative association. This probably reflects a point raised earlier, namely that our model considered lifetime reproductive success in the context of individual male dominance trajectories (ontogeny), whereas the Thrall *et al*. (2000) model fixed a male's mating success to be constant throughout his life. Similarly, our model produced a weaker prediction for sex-biased prevalence: Thrall *et al*. (2000) and Ashby and Gupta (2013) predicted female-biased prevalence, whereas our model produced a weaker sex-difference (but also female biased).

In conclusion, we found that several host traits predict STD risk in primate populations. We also found evidence for the predicted effects in wild primates, especially in the case of proxies for mortality rate (host longevity and body mass). In other ways, however, our model proved less able to predict effects of skew on STD prevalence, suggesting that more theoretical research is needed to understand the links between mating competition and disease risk, including research on links between dominance rank and susceptibility to infectious disease. This is particularly the case, given that —as emphasized by Ashby and Gupta (2013)— heterogeneity in partner acquisition rates and within/among group movement mixing patterns could drive the evolution of pathogen virulence.

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