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SOCIAL ORGANIZATION AND PARASITE RISK IN MAMMALS: Integrating Theory and Empirical Studies

Sonia Altizer,[1] Charles L. Nunn,[2] Peter H. Thrall,[3] John L. Gittleman,[4] Janis Antonovics,[4] Andrew A. Cunningham,[5] Andrew P. Dobson,[6] Vanessa Ezenwa,[6,7] Kate E. Jones,[4] Amy B. Pedersen,[4] Mary Poss,[8] and Juliet R.C. Pulliam[6]

[1]*Department of Environmental Studies, Emory University, Atlanta, Georgia 30322; email: saltize@emory.edu*
[2]*Section of Evolution and Ecology, University of California, Davis, California 95616; email: cnunn@ucdavis.edu*
[3]*CSIRO-Plant Industry, Center for Plant Biodiversity Research, GPO Box 1600, Canberra ACT 2601, Australia; email: Peter.Thrall@csiro.au*
[4]*Department of Biology, University of Virginia, Charlottesville, Virginia 22904; email: ja8n@virginia.edu, JLGittleman@virginia.edu, kate.jones@virginia.edu, abp3a@virginia.edu*
[5]*Institute of Zoology, Zoological Society of London, London, United Kingdom, NW1 4RY; email: Andrew.Cunningham@ioz.ac.uk*
[6]*Department of Ecology and Evolutionary Biology, Princeton University, Princeton, New Jersey 08544; email: andy@eno.princeton.edu, pulliam@princeton.edu*
[7]*Present address: U.S. Geological Survey, Reston, Virginia 20192; email: vezenwa@usgs.gov*
[8]*Division of Biological Sciences, University of Montana, Missoula, Montana 59812; email: mposs@selway.umt.edu*

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■ **Abstract** Mammals are exposed to a diverse array of parasites and infectious diseases, many of which affect host survival and reproduction. Species that live in dense populations, large social groups, or with promiscuous mating systems may be especially vulnerable to infectious diseases owing to the close proximity and higher contact rates among individuals. We review the effects of host density and social contacts on parasite spread and the importance of promiscuity and mating structure for the spread and evolution of sexually transmitted diseases. Host social organization and mating system should influence not only parasite diversity and prevalence but may also determine the fitness advantages of different transmission strategies to parasites.

Because host behavior and immune defenses may have evolved to reduce the spread and pathogenicity of infectious diseases, we also consider selective pressures that parasites may exert on host social and mating behavior and the evolutionary responses of hosts at both the immunological and behavioral levels. In examining these issues, we relate modeling results to observations from wild populations, highlighting the similarities and differences among theoretical and empirical approaches. Finally, the epidemiological consequences of host sociality are very relevant to the practical issues of conserving mammalian biodiversity and understanding the interactions between extinction risk and infectious diseases.

INTRODUCTION

Social organization, including the size and composition of social groups, and mating systems, including partner exchange rates and variance in male and female mating success, should directly influence host proximity and the number and duration of contacts in a population. These behaviors are therefore expected to have major effects on parasite spread within host species and should influence the distribution of parasites among host species. This point is illustrated vividly in the case of sexually transmitted diseases (STDs), in which the expected risk of infection increases with the number of mating partners (e.g., Anderson & May 1992, Thrall et al. 2000). Consequently, differences in promiscuity among host species should influence the prevalence and diversity of STDs. Social interactions also provide key opportunities for parasite spread, and parasites transmitted by social contacts are expected to be more common in larger groups and in denser populations (Arneberg et al. 1998, Freeland 1976, Loehle 1995). In fact, parasites may ultimately represent a major cost to both social organization and promiscuity, and changes in host behavior may arise from increased parasite prevalence or severity. A cogent example is the shift in human sexual behavior resulting from the AIDS pandemic (e.g., Anderson et al. 1989, Mills et al. 1997).

General expectations for how host behavior might affect parasite spread may seem relatively straightforward (Figure 1), but questions remain to be addressed at two levels: patterns of parasitism within populations, and comparative patterns among host and parasite communities. For example, do highly social hosts harbor greater parasite diversity, and do observed patterns depend on parasite characteristics? Do promiscuous host species experience a greater risk of STDs, and how does host mating behavior affect the evolution of parasite transmission? Have behavioral or immune defenses evolved in highly social species to minimize disease risk? Answering such questions provides an important step toward predicting patterns of disease incidence and will aid in identifying the potential for new parasites to emerge and spread.

This review discusses recent advances in this field and raises new questions for understanding the links between host sociality and the transmission of infectious disease. Two complementary approaches are needed to address these questions: theoretical studies that examine how key variables influence parasite spread, and empirical studies that assess infection risk within populations and the distribution

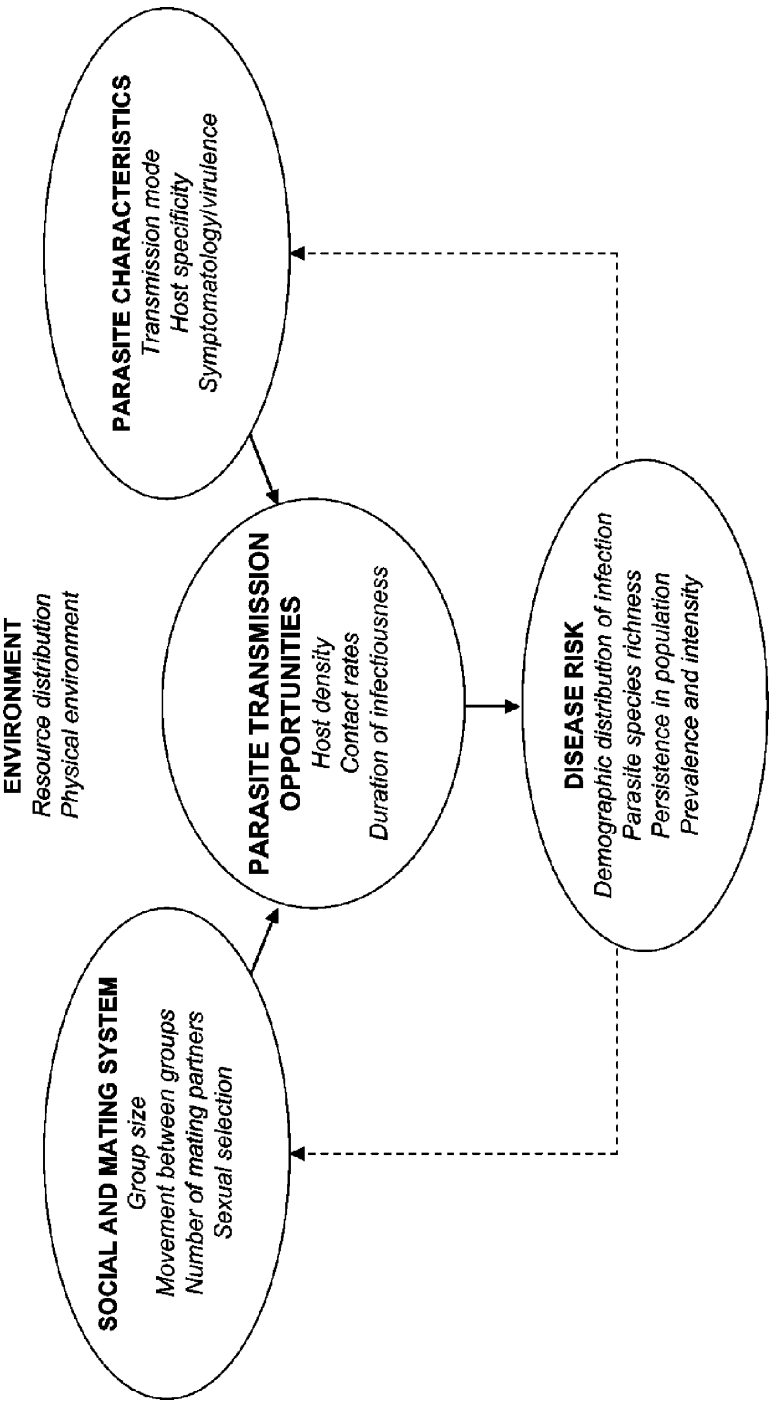


Figure 1 Key variables affecting the ecology and evolution of host-parasite interactions in wild mammal populations. Both host and parasite traits will interact with environmental characteristics to determine opportunities for parasite transmission (*solid lines*), which may in turn select for evolutionary changes in both host and parasite (indicated by *dashed lines*).

of parasites across host populations or species. Throughout this review, we apply the term parasite to any infectious organism capable of colonizing a host, utilizing host resources, and spreading to new hosts. Our conclusions apply to both microparasites (such as viruses, bacteria, and protozoa) and macroparasites (such as helminths and arthropods; Anderson & May 1992).

Integrating theoretical results with empirical approaches is best accomplished using a model system, or a group of related species in which studies have addressed basic natural history and conceptual issues (cf. Dugatkin 2001). We focus on mammals because comprehensive data are available on social, ecological, life history, and biogeographic parameters for a large proportion of species (e.g., Geffen et al. 1996, Gittleman 1996, Smuts et al. 1987). Moreover, because of their uses in farming and biomedical research, a great deal of information is available on the parasites of wild and captive mammal populations, making them particularly well suited for comparative studies (e.g., Arneberg et al. 1998, Morand & Poulin 1998). The evolutionary history of mammals is becoming increasingly well known (e.g., Bininda-Emonds et al. 1999, Jones et al. 2002, Liu et al. 2001) enabling examination of questions in a phylogenetic context. Finally, mammals are important foci for conservation efforts, and understanding the role of parasites in wild populations will become vital for future conservation and management decisions (e.g., Dobson & Lyles 2000, Funk et al. 2001).

GENERAL BACKGROUND

In pioneering research, Freeland (1976, 1979) suggested that primate social interactions and behavior have evolved to reduce the spread and pathogenicity of new and existing parasites. Assuming that larger social groups experience increased disease risk, selection for pathogen avoidance should influence social group size, composition, and intergroup movements. For example, high parasite pressure may lead to increased rates of juvenile dispersal, and activities that induce behavioral or nutritional stress should increase the susceptibility of particular classes of individuals such as newcomers trying to enter a group or individuals vying for potential mates. Despite clear intuitive links and a growing number of theoretical studies that address host social and mating systems and infectious diseases, empirical studies that examine parasite spread and host sociality face many challenges. These include the need to control for a large number of potentially correlated host and parasite traits (Figure 1) and a lack of correspondence between methods for quantifying host behavior and parameters from epidemiological models, in addition to difficulties associated with population-level experimental work with mammalian species.

Social and Mating Systems in Mammals

Social organization, defined by the size and composition of social groups and patterns of intergroup dispersal, should directly influence host density and the number and duration of contacts within a population with important consequences for parasite transmission (Figure 1). For example, monogamous species with strictly

defended territories are expected to exhibit fewer parasites because their number of intraspecific contacts is small, whereas social mammals that live in multi-male multi-female groups may afford greater opportunities for parasite spread. Mammalian social systems are distinguished primarily by the temporal and spatial interactions between adults and by the genetic relatedness among individuals (e.g., Alexander 1974). For example, solitary species often come together only to mate, whereas gregarious species may breed colonially in family groups or form herds or packs with either stable or variable composition. Social interactions can also be influenced by territoriality or exclusion of other groups and the presence of substructuring or dominance within groups (e.g., Dunbar 1988, Eisenberg 1981, Smuts et al. 1987).

Mating systems can be defined by variance in male and female mating success within a population, and vary dramatically among mammalian species. Observed patterns range from monogamy to polygynandry (where both sexes mate with multiple partners during a breeding season; e.g., Clutton-Brock 1989, Eisenberg 1981). Mating systems also exist in which males, but not females, have multiple partners (polygyny) and vice versa (polyandry), and such mating bonds can last throughout life or be limited to a few reproductive events.

Several ecological factors are thought to influence mammalian mating and social systems (Clutton-Brock 1989, Emlen & Oring 1977). Both sexes require resources and access to mates. However, sex differences in parental investment may skew the operational sex ratio toward males (Clutton-Brock & Parker 1992), and the relative importance of resources versus mating opportunities affect the two sexes differently (Trivers 1972). In mammals, females often invest more in offspring than do males, leading to the prediction that female social strategies should reflect environmental risks and resource availability, whereas male social strategies should reflect the opportunity to control groups of females (Emlen & Oring 1977). In microtine rodents, for example, female distributions are linked to environmental heterogeneities, and male distributions depend on female densities (e.g., Ostfeld 1985). In polygynous species such as red deer, clumped resources result in males defending areas that attract females, whereas dispersed resources lead to direct male defense of roving females (Carranza et al. 1995). Finally, intersexual conflict may influence social and mating systems. In primates, for example, stable male-female relationships and home range defense may protect against infanticide by extragroup males (as in gibbons; van Schaik & Dunbar 1990), and female promiscuity may confuse paternity in larger social groups (as in baboons and macaques; van Schaik et al. 1999).

A particular challenge arises from the need to quantify mating and social structure in ways that are meaningful to host-parasite dynamics. As a case in point, mating systems defined categorically (as monogamous or polygynous) only poorly capture characteristics that are important from an epidemiological perspective. To assess STD risk in different mating systems, it is necesary to quantify variance in male and female mating contacts, how mating contacts vary with age and social status, the duration and fidelity of contacts within groups, and rates of intergroup migration (Figure 1). Many of these variables are difficult

to measure directly in wild populations, and indirect measures, including sexual dimorphism in body size or other traits under sexual selection, may be used to infer the degree of reproductive skew (Andersson 1994). For example, it has been shown that relative testes mass (after controlling for body mass) can indirectly reflect variation in mating promiscuity among host species (Nunn et al. 2000). Similarly, grooming rates with different partners or social group size can be used to quantify social contacts. Finally, intergroup migration is rarely measured in wild populations of social mammals, yet movement among groups should have major consequences for parasite spread (Thrall et al. 2000).

Parasite Ecology and Epidemiological Parameters

A general understanding of parasite ecology and epidemiology provides a set of predictions regarding ecological factors that influence parasite spread and persistence. In simple host-parasite models with direct transmission, the probability that most parasites will spread in a host population is an increasing function of host density and longevity and a decreasing function of parasite-induced mortality and recovery (Anderson & May 1992). New infections usually depend on host contact rates and per contact probabilities of successful infection. This leads to the straightforward prediction that hosts living at high density or with frequent intraspecific contacts will increase the spread and prevalence of any given parasite species, and, by extension, the number of parasite species harbored by a host population (Figure 1; Anderson & May 1979, Arneberg 2002, Roberts et al. 2002).

Parasites exhibit an impressive variety of transmission modes, with a major dichotomy between direct transmission (where parasites are spread directly from host to host by sexual, social, or other close contact) and indirect transmission (where hosts encounter parasites in the environment, or through intermediate hosts or vectors). Different transmission modes should interact with host traits to influence parasite spread and persistence (Figures 1 and 2). The establishment of an STD, for example, depends on both host sexual behavior and parasite adaptations to increase infection probability. Increased sociality and greater host population density are predicted to increase the transmission of parasites spread through direct contact (Thrall & Antonovics 1997), whereas parasites spread by biting vectors or exposure to contaminated soil or water may be less sensitive to changes in host contacts or density (e.g., Anderson & May 1992). Interestingly, sexual (between mating partners) and vertical (parent to offspring) transmission have been suggested as parasite strategies for persistence in low density or solitary host species, as sexual reproduction is one of the few times that conspecifics come into contact in these species (Smith & Dobson 1992).

One useful epidemiological perspective is to characterize conditions for initial spread and persistence in a host population among parasites that vary in their transmission mode. Much recent work has considered the differences between STDs

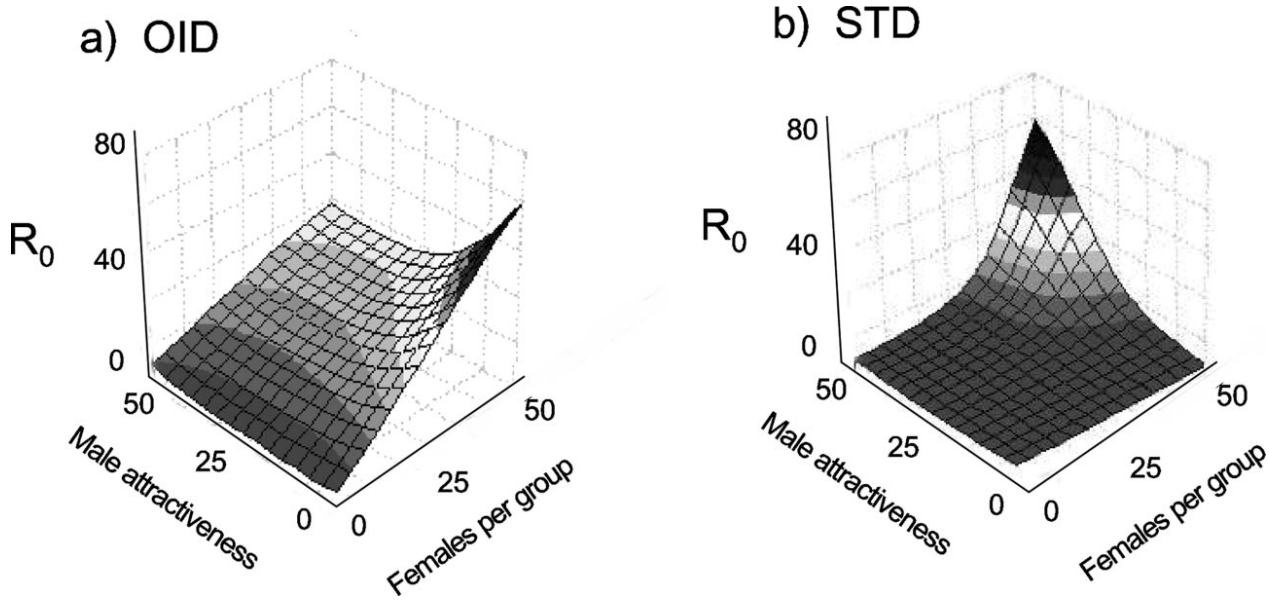


Figure 2 Relative change in R_0, the basic reproductive ratio, for OIDs (*left*) and STDs (*right*) given variation in female group size and male mating success. Here, R_0 provides a measure of the number of secondary infections produced by a single infected host introduced to an entirely susceptible host population. In each figure, the relative magnitude of R_0 is plotted as a function of the number of females in each social group and "male attractiveness," an index of the number of females each breeding male mates with in an annual mating season. For STDs, initial spread is maximized by large female group size and high male attractiveness, which leads to relatively few breeding males mating with a large number of females. For OIDs, R_0 is maximized for large female group size but low male attractiveness, so that many breeding males each mate with a small number of females (A. Dobson et al. unpublished manuscript).

and ordinary infectious diseases (OIDs) that are transmitted by nonsexual direct contact. The temporal dynamics and criteria for establishment of STDs and OIDs are expected to differ, as are their regulatory effects on host abundance (Figure 2). For example, the number of "effective contacts" leading to parasite transmission should increase directly as a function of host density for parasites spread by social contact. However, for STDs, the effective number of sexual contacts is expected to saturate quite rapidly with increasing host density. Therefore, the rate of spread of STDs should depend more strongly on the proportion of infected hosts rather than on total host density (Getz & Pickering 1983, Thrall et al. 1993). This dichotomy has been captured mathematically as the difference between density-dependent transmission (where the change in the number of infected hosts, I, depends on βSI, or the product of the transmission parameter, β, and the number of susceptible and infected hosts) versus frequency-dependent transmission (where the change in the number of infected hosts depends on $\beta SI/N$, where N is the total host population size). Recent studies suggest that parasite transmission is neither purely frequency nor density dependent but is a complex function of both (Antonovics et al. 1995, Begon et al. 1999, Knell et al. 1996).

Another parasite characteristic relevant to successful maintenance of parasites involves the degree of host specificity (Figure 1). Parasites may be classified as generalists that infect many host species, or specialists that infect only one or a few host species. Host specificity can be measured by the actual number of susceptible host taxa (Poulin 1998) but is more accurately measured relative to host taxonomy or phylogeny. The capability and opportunity to infect multiple host taxa should interact with parasite transmission mode as some transmission routes (e.g., sexual, close contact) provide almost no opportunities for cross-species transfers (Figure 1). By comparison, vector transmission or transmission through contaminated soil, water, or intermediate hosts can expose multiple host species to the same parasite (Woolhouse et al. 2001).

Finally, parasite virulence plays a key role in host-parasite dynamics and may coevolve with transmission mode and host behavior (Figure 1; Levin 1996, Messenger et al. 1999). Although precise definitions of virulence vary (e.g., Bull 1994, Ewald 1994, O'Keefe & Antonovics 2002), this term usually refers to reductions in host survival or fecundity stemming from parasite replication or damage to host tissues. Counter to the traditional wisdom that parasites should evolve to cause minimal harm to hosts, virulence may provide a selective advantage to parasites if disease symptoms increase transmission to new hosts, or when multiple strains compete within the same individuals (Bull 1994). Parasites transmitted by sexual or vertical routes require that hosts survive long enough to mate or reproduce, so that in these cases, reductions in host longevity may have strong negative effects on parasite transmission (Lockhart et al. 1996, Thrall et al. 1993). Interestingly, STDs are more likely to induce host sterility than OIDs, an effect that may enhance their transmission if infected (and hence sterile) females undergo more frequent reproductive cycles and mate more often (Lockhart et al. 1996, Nunn & Altizer 2003).

SOCIALITY AND PARASITE SPREAD

If close proximity or contact among host individuals increases parasite transmission, then greater degrees of host sociality or gregariousness should translate to higher parasite prevalence, intensity, and diversity (Møller et al. 1993). Here, prevalence refers to the proportion of infected or diseased hosts, intensity refers to the average number of parasites within infected hosts, and diversity includes the total number of parasite species documented in host populations. Thus, social hosts are predicted to suffer greater exposure to parasites (Brown & Brown 1986, Møller et al. 2001), experience increased selection for innate or acquired immune defenses, and evolve behavioral defenses against parasites (Freeland 1976, Loehle 1995).

A large number of epidemiological models, supported by data from several empirical and comparative studies, point to strong links between host density or local group size and the spread and diversity of directly transmitted parasites (Figures 2 and 3; Anderson & May 1979, Arneberg 2002). For example, Dobson & Meagher (1996) summarized evidence that brucellosis in North American bison has a host density threshold for establishment, and Packer et al. (1999) showed that the incidence of infection with four different viruses in African lions increased with the estimated number of previously unexposed individuals. A comparative study of parasites in wild primates showed that host density was the most consistent factor predicting increases in the diversity of both macro- and microparasite communities (Nunn et al. 2003a; Figure 3).

Among mammals and other vertebrates, social group size appears to be an important predictor of parasite risk (Côté & Poulin 1995, Davies et al. 1991). At the level of single host populations, parasite prevalence, intensity, and occasionally diversity have been shown to increase with group size in a wide range of host taxa including prairie dogs (Hoogland 1979), mangabeys (Freeland 1979), cliff swallows (Brown & Brown 1986), bobwhites (Moore et al. 1988), and feral horses (Rubenstein & Hohmann 1989). A few studies have found similar patterns in cross-species comparative analyses including studies of birds (Poulin 1991) and fishes (Ranta 1992). However, the association between group size and infection risk is often confounded with other host traits (e.g., Rózsa 1997, Clayton & Walther 2001). In particular, because both host density and social group size have been shown to correlate positively with parasite prevalence and diversity, it is difficult to determine the relative importance of social contacts versus host density for parasite transmission (Arneberg 2002, Morand & Poulin 1998).

It is important to note that associations between parasite transmission and group size will depend on other host and parasite traits (Figure 1). For example, Moore et al. (1988) found consistent relationships between bobwhite covey size and helminth intensity only for directly transmitted parasites with relatively short life cycles. Furthermore, using a meta-analytical approach, Côté & Poulin (1995) found strong positive correlations between vertebrate group size and both the prevalence and intensity of directly transmitted helminths, whereas the intensity of

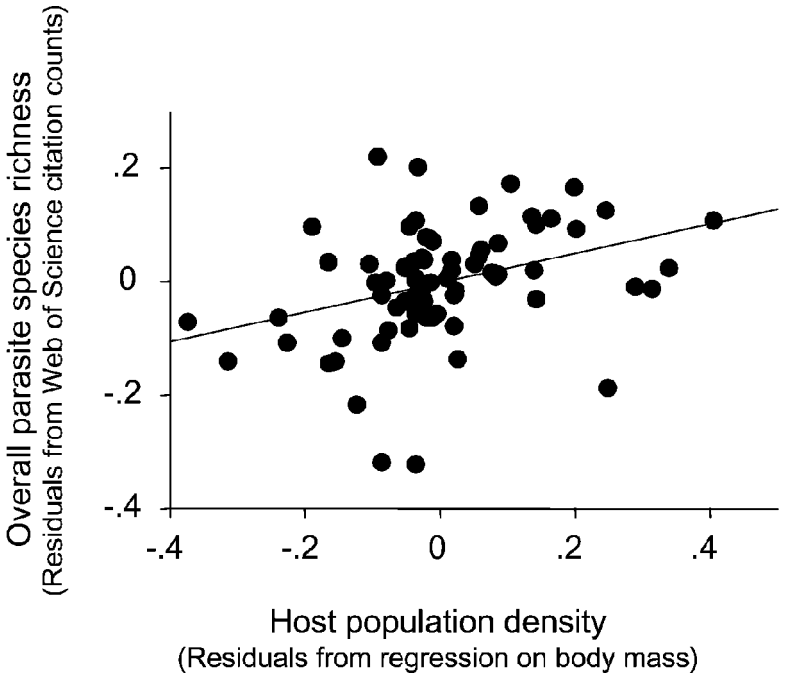


Figure 3 Effects of host density on overall parasite species richness in primates. Plot shows independent contrasts calculated using CAIC (Purvis & Rambaut 1995) and Purvis' (1995) composite estimate of primate phylogeny. Parasite species richness includes parasites in six functional classes (helminths, protozoa, viruses, bacteria, fungi, and arthropods), from free-ranging hosts only, collated from the published literature (Nunn et al. 2003a). Sampling effort was included as a covariate in statistical analyses using information on the number of citations for the different species from the Web of Science citation index, years 1975 to 2001. Results remain significant when using species values and controlling for body mass and geographic range size ($b_{popln\text{-}density} = 0.17$, $F_{1, 75} = 5.49$, $P = 0.02$, two-tailed; Nunn et al. 2003a).

infection by indirectly transmitted parasites decreased with social group size. Host traits such as movement and territoriality may further confound the relationship between parasitism and host group size. For example, a field study comparing intestinal parasite loads among 11 species of African bovids (Ezenwa 2002) showed that the prevalence of coccidian parasites increased with group size only among host species with closed group structures (and not for species with high rates of intergroup exchange). The same study also found that nematode infections were more prevalent among territorial antelopes compared with nonterritorial species, possibly as a result of increased exposure to parasites resulting from infective stages accumulating in the environment (Ezenwa 2002). These results highlight

the fact that many other features of host and parasite ecology are important to identifying the effects of group size.

A common modeling approach to investigating heterogeneity in patterns of social contacts in human populations is to group individuals into classes (e.g., social status, degree of sexual activity) and describe contacts among classes in terms of a "mixing matrix," where the entries in each of the cells describe the frequency distribution of contacts per unit time (Blower & McLean 1991). The most important prediction gained from these models is that the pattern of contacts between different activity classes has a major impact on parasite spread (Jacquez et al. 1988). Specifically, a high degree of mixing within an activity class results in a more rapid initial spread but a lower population-wide prevalence than a high degree of mixing among different activity classes. Despite their importance in human epidemiology, mixing matrices have not been applied to animal social and mating systems because detailed information for their construction (contact rates within and among social classes or mating groups) has generally not been available.

An important question related to parasite spread in socially structured populations involves identifying individuals that are at greatest infection risk. Parasitism is likely to correlate with dominance rank, age, sex, and mating status (Hausfater & Watson 1976, Muller-Graf et al. 1996) because these factors influence habitat use, the frequency of intraspecific contacts, and the effectiveness of immune defenses. For example, Halvorsen (1986) showed that dominant reindeer were more frequently exposed to nematode infections because they consumed more vegetation. Among African antelopes, territorial males were exposed to more parasites than bachelor males or females (Ezenwa 2002), possibly owing to the immunosuppresive effects of testosterone or the accumulation of parasites on resident male territories. Courchamp et al. (1998) showed that among feral cat populations, older males (with greater dispersal and a higher number of aggressive encounters) were more likely to be infected with feline immunodeficiency virus (FIV). Although processes that underlie individual differences in infection risk have been identified for some species, understanding their relative importance in a cross-species context is necessary for a broader understanding of factors that determine patterns of parasite occurrence.

Nearly all empirical research on infectious disease and sociality in wild populations has focused on opportunities for transmission in different mating and social systems (solid line in Figure 1). An important area for future research involves the effect of infectious diseases on mammalian sociality (dashed line in Figure 1). There is growing evidence that parasites represent a strong selective force for the evolution of mating systems and social interactions (e.g., Møller et al. 2001). Perhaps the best example from mammals is the observation that increased presence of ectoparasites (flies, ticks, and other arthropods) increases host tendency to form large groups, possibly as a way of avoiding high parasite loads through the dilution effect (Mooring & Hart 1992, Rubenstein & Hohmann 1989). These results suggest that parasitism can shape host sociality, but no studies have shown that species evolve behaviors that decrease group size following high parasite pressure.

SEXUALLY TRANSMITTED DISEASES

STDs are increasingly recognized as an important parasite group with potentially large impacts on host reproduction and evolution, in many cases increasing the chances of sterility (Lockhart et al. 1996, Smith & Dobson 1992). Despite apparent overlap between social and mating systems in mammals, and potentially similar mechanics of sexual and social contact, the characteristics and dynamics of STDs differ from many other infectious diseases. The pathogens causing such diseases have smaller host ranges, longer infectious periods, and are less likely to cause host mortality or induce protective host immunity (Lockhart et al. 1996, Oriel & Hayward 1974, Smith & Dobson 1992). Animals with promiscuous mating systems (or species in which females engage in frequent extrapair copulations) are predicted to experience a greater risk of acquiring parasites through sexual contact. However, empirical patterns illustrating potential links between host mating behavior and infectious disease risk have not been well documented among mammals or other vertebrates. The dynamics of most STDs cannot be understood without considering heterogeneity in sexual activity (Anderson & May 1992). For this reason, population models developed to predict HIV dynamics and control have focused on human sexual contact patterns (e.g., Anderson et al. 1988, 1989; Boily & Masse 1997), and this focus has extended to other human STDs such as gonorrhoea and syphilis (Garnett et al. 1997, Hethcote & Yorke 1984).

Characteristics of many STDs cause their dynamics to differ from other directly transmitted parasites. In particular, STDs tend to persist as endemic (rather than epidemic) infections with transmission relatively unaffected by increased host density or crowding. They have also been described as a unique class of pathogens well adapted to persisting in small, low density host populations (Smith & Dobson 1992), although their presence in large populations is certainly not theoretically precluded.

Mathematical models that incorporate heterogeneity in mating behavior show that STD transmission increases with increasing variance in partner exchange rates and that highly promiscuous individuals (“superspreaders”) can facilitate STD persistence even when the mean number of sexual partners is low (Anderson & May 1992). Consistent with models that predict a higher risk of infection among more promiscuous subgroups, surveys of HIV and other STDs in human populations show that prevalence increases with increasing numbers of sexual partners per year (reviewed in Anderson & May 1992). One might expect this generalization to apply to wild mammals with polygynous mating systems with variance in male mating success at the population level being proportional to increased transmission of STDs. Using an individual-based simulation model of polygynous mating systems, Thrall et al. (2000) showed that variance in male mating success affects the spread of STDs only when the migration of females among mating groups is limited. Their model assumed that males varied in their attractiveness to females, that females had only one mate per breeding season, and that females could change groups between breeding seasons. Two mating system parameters were examined: variation in male mating success and variation in

female fidelity to males. When females moved frequently among groups, their model demonstrated that variance in male mating success (meaning increasing skew in the number of females associated with any given male) had almost no effect on parasite spread. When intergroup movement was limited, parasites spread rapidly in groups where males monopolized a large number of females, but transmission was highly limited in smaller groups.

A second notable outcome of the model by Thrall et al. (2000) was that equilibrium STD prevalence was significantly greater in females than in males, with parasite prevalence in females increasing to an asymptote with increasing skew in male mating success (Figure 4*a*). Thus, when variance in male mating success was high, many males remained unmated, lowering the equilibrium prevalence among males relative to females. Using published data on two sexually transmitted retroviruses in wild primate populations, Nunn & Altizer (2003) tested the prediction that STD prevalence should be higher in females than in males among nonmonogamous species. Data from sexually mature adult primates showed that in a majority of the sample populations, seroprevalence was higher among females (Figure 4*b*), and prevalence differences for males and females were statistically significant and in the predicted direction when tested using a matched pairs t-test (Nunn & Altizer 2003). Although these analyses were consistent with the model predictions, alternative explanations (for example, sex-based differences in per contact transmission probabilities or disease susceptibility) are possible. Higher STD prevalence among females has also been reported among captive breeding primate colonies including sooty mangabeys and baboons (Fultz et al. 1990, Levin et al. 1988).

These differences in STD prevalence between males and females are more striking because theory predicts the opposite pattern for OIDs owing to a presumed sex-based difference in disease susceptibility. For example, prevalence is expected to be higher in males (Alexander & Stimson 1988, Bundy 1988) as a consequence of the energetic costs associated with competition for mates or the deleterious effects of testosterone on immunocompetence (Zuk 1990). Moore & Wilson (2002) showed that among mammal species where male-male competition is most extreme, male-biased mortality coincided with greater male susceptibility to parasitic diseases. This pattern may result from effects of testosterone on immunocompetence, but effects of body size on parasite infection can also explain male-biased parasitism. In this context, Moore & Wilson (2002) found that in general the larger sex suffered greater parasitism, regardless of gender. Further studies are needed to determine the consequences of sex-biased susceptibility for the evolution of mate choice, particularly with respect to traits that signal parasite infection.

STDs and the Evolution of Host Mating Strategies

An important epidemiological consequence of host sexual behavior is that "attractive" males are predicted to suffer a greater risk of STD infection (Graves & Duvall 1995, Thrall et al. 2000). Thus, parasites transmitted during mating may

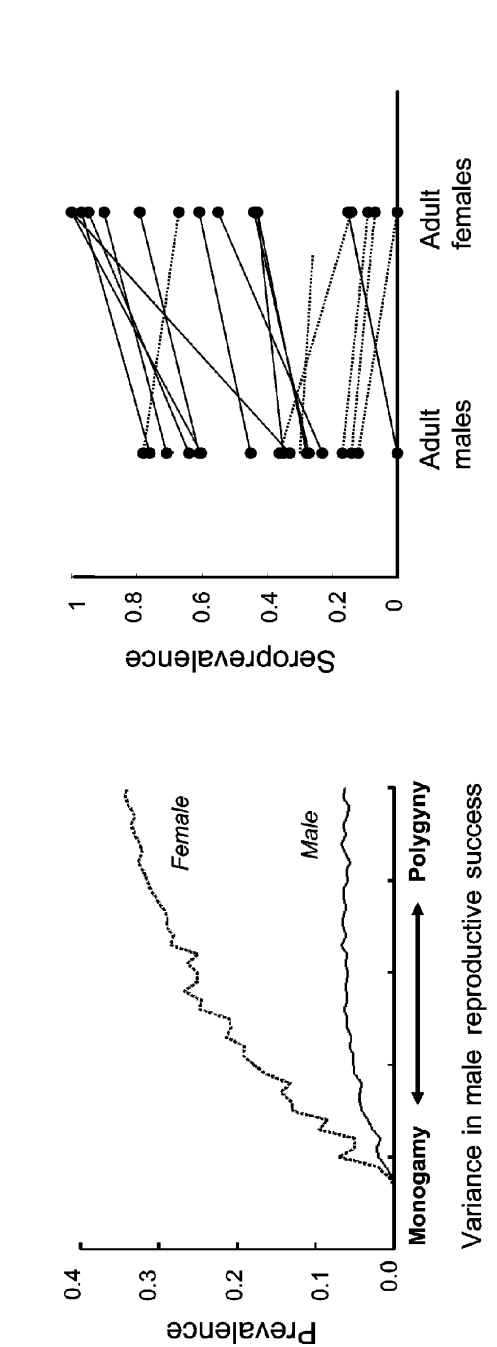


Figure 4 (*a*) Differences in prevalence of STDs in males and females as predicted from individual-based mathematical models developed by Thrall et al. (2000) under the assumption of low female movement among mating groups. Increasing the variance in male mating success led to a greater proportion of unmated males in the population; (*b*) Empirical data for viral STDs in 17 populations of wild primates, showing prevalence in males (*left*) and females (*right*), with lines connecting points from the same population. The number of animals sampled ranged from 6 to 314 for males (median = 14 individuals) and 5 to 719 females (median = 21 individuals). Data are available on two viral STDs, simian immunodeficiency virus (SIV, 7 of 17 data points), and simian T-lymphotropic virus (STLV, 10 data points). Of the 17 populations, 6 showed higher prevalence among males than females (*dashed lines*), and 11 showed higher prevalence among females than males (*solid lines*). For more information, see Nunn & Altizer (2003).

have debilitating effects on dominant males, hastening their replacement by subordinates. Studies of baboons, for example, revealed a strong positive correlation between social and reproductive status and parasite loads (Hausfater & Watson 1976). Interestingly, the theoretical studies of Thrall et al. (2000) suggested that STDs would not limit the evolution of male traits that increased polygyny unless females suffered reproductively by joining multi-female groups. Even in the presence of a sterilizing STD, more attractive males still had higher reproductive success than less attractive males (although the selective advantage for polygyny was much lower in the presence of an STD).

Should monogamy be the optimal mating strategy in the presence of a potentially sterilizing STD? Thrall et al. (1997) addressed this question directly by modeling mating events that were associated with both a per-contact transmission probability and a fertilization probability. They showed that optimal strategies for males and females could differ substantially in the presence of an STD, indicating that parasites alone have the potential to influence the evolution of sex-based differences in mating behavior. For example, when both transmission rates and STD prevalence were high, monogamy was always the optimal strategy for females, but the best strategy for males was to mate with as many females as possible. Overall these results confirmed that STDs spread more rapidly in promiscuous mating systems. However, even though monogamy always resulted in the lowest parasite levels, it was not always the favored strategy owing to reproductive benefits that arise from promiscuous mating.

Evolution of Sexual Transmission and Virulence

In a full coevolutionary model, host social and sexual behavior should interact with pathogen transmission and virulence. With regard to pathogen virulence, STDs range from those that are relatively benign to those that are highly virulent, either causing high mortality or extreme sterility (Lockhart et al. 1996). In general, STD virulence is expected to be higher when extrapair copulations are common than when monogamy predominates. This idea has been discussed with respect to human sexual behavior and the evolution of HIV (Ewald 1994).

Transmission modes themselves may evolve depending on host social and mating behavior. Using differential equation models, Thrall & Antonovics (1997) derived conditions under which an STD could invade a host population and displace a pathogen transmitted by nonsexual means (OID). Invasion by the STD was easier when the equilibrium host population size with an OID was relatively small. Conversely, an OID could invade more easily if the equilibrium population size with an STD was larger. Overall, these results reflect the general expectation that sexual transmission should be favored in low density populations, whereas nonsexual transmission should be favored at high densities (Anderson & May 1992, Smith & Dobson 1992). Based on more realistic assumptions related to the importance of host population density, Thrall et al. (1998) proposed the concept of a social-sexual crossover point (SSCP) associated with parasite transmission

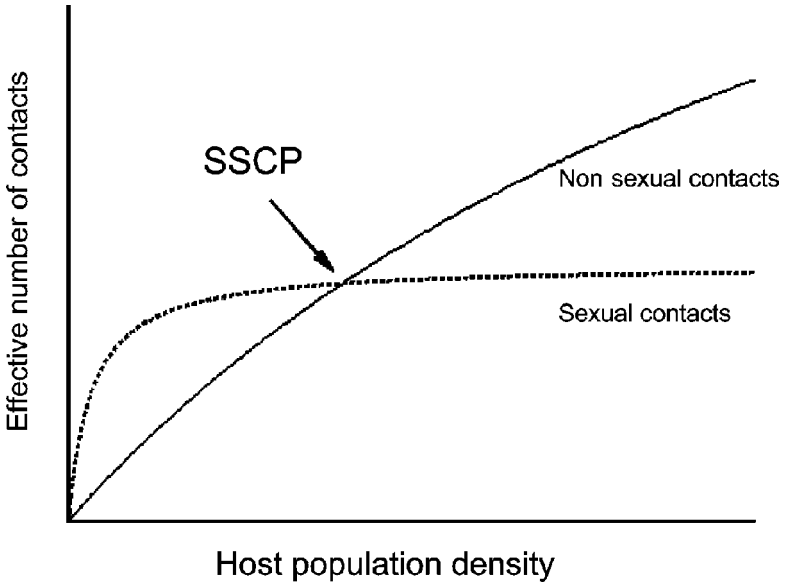


Figure 5 Relationship between effective contact number (the number of contacts per unit time that actually result in disease transmission) and host population density (see Thrall et al. 1998). The arrow indicates the social-sexual crossover point (SSCP) where the number of nonsexual contacts exceeds the number of sexual contacts. Because even at low population densities, individuals will still actively seek out sexual contacts for reproductive purposes, the number of sexual contacts is generally assumed to initially increase more rapidly with host density, but to reach an asymptote at lower numbers (owing to the greater handling time associated with sexual versus nonsexual contacts).

(Figure 5). These formulations assumed that (*a*) as population density increases, social and sexual contacts also increase; (*b*) the number of sexual contacts will initially increase more rapidly with density than the number of social contacts (at low population densities, individuals still seek mates); and (*c*) at higher densities, the number of sexual contacts will rapidly saturate (owing to longer durations associated with sexual contacts) but the number of social contacts will continue to increase. Thus, the SSCP represents a critical host population density at which the numbers of social and sexual contacts are equal (Figure 5). Clearly, the host density at which the SSCP occurs could vary considerably depending on the details of host social and mating structure, but empirical studies of this relationship remain a challenge for future research. As predicted, Thrall et al. (1998) found that increased sexual transmission was always favored if the equilibrium population size was less than the SSCP; otherwise, nonsexual transmission was favored.

HOST DEFENSES AND BEHAVIORAL AVOIDANCE

If the probability of infection increases with group size or promiscuity, then highly social or promiscuous hosts should experience more intense selection in favor of barriers (behavioral or immunological) to parasite transmission (Freeland 1976, Loehle 1995). For example, grooming, preening, and selective foraging have been suggested as parasite avoidance mechanisms in social vertebrates (Hart 1990, Loehle 1995, Moore 2002), although it is important to note that behaviors such as grooming may actually facilitate the transmission of some parasites while reducing the transmission of others. Ironically, if highly social hosts evolve more elaborate defenses as obstacles to parasite infection and impacts (Møller et al. 2001), this may eliminate expected relationships between parasitism and host sociality (the "ghost of parasitism past").

In vertebrates, the innate component of the immune system, including phagocytic cells such as monocytes and neutrophils, controls the immediate host response to general classes of pathogens. As such, white blood cell (WBC) counts may reflect a baseline defense against parasite invasion, particularly for those WBCs that target specific pathogen groups. The strength of innate immunity should therefore increase among taxa that experience high parasite pressure (Møller et al. 1998), especially if defenses are costly to maintain (Nordling et al. 1998, Sheldon & Verhulst 1996). However, it is important to note that other factors (including age and stress) will influence WBC counts, and other components of host immunity play a role in antiparasite defense.

Using data acquired from healthy zoo primates, Nunn et al. (2000) showed that mating promiscuity explained significant variation in leukocyte counts (including lymphocytes, neutrophils, monocytes, and eosinphils), whereas the effects of sociality, life history, and habitat use were nonsignificant. In a separate comparative study of carnivores, host promiscuity, sociality, and longevity explained significant variation in leukocyte counts across species (Nunn et al. 2003b). Moreover, a strong allometric relationship involving neutrophils was found in both primates and carnivores so that larger-bodied hosts harbored a greater neutrophil abundance, possibly indicating greater parasite exposure. Collectively, these results are consistent with experimental immunological research that demonstrates that innate immune defenses offer protection against pathogen invasions, and they draw attention to correlates between host life history, behavior, and immunity by showing that hosts more likely to encounter sexually transmitted pathogens had higher WBC counts.

A variety of behavioral traits may operate in conjunction with the immune system to limit exposure to parasites (Hart 1990, Loehle 1995). Strategies to avoid nonsexual parasites have been discussed extensively in primates, including alteration of ranging patterns (Di Bitetti et al. 2000, Hausfater & Meade 1982), ingestion of medicinal plants (Huffman 1997), and avoidance of recent immigrants that may harbor novel parasites (Freeland 1976). Behavioral avoidance of fecal-contaminated areas by selective foraging has been reported for domestic

grazing ungulates, where the risk of ingestion of fecal-borne parasites is high (e.g., Hutchings et al. 1998, Moe et al. 1999). Interestingly, some wild territorial bovids also avoid dung while foraging (Ezenwa 2002), possibly owing to increased risk of exposure to nematode infections among resident animals. Grooming rates have been shown to correlate positively with the risk of parasitism in both ungulates and primates. In primates, allogrooming is concentrated in regions of the body inaccessible to self-grooming, further suggesting that this behavior plays an important antiparasite function (e.g., Barton 1985).

Parasites with transmission modes for which behavioral counterstrategies may be relatively ineffective are predicted to select more strongly for increased immune defenses. In the case of STDs, for example, behaviors that reduce the risk of transmission may result in lower reproductive success (Thrall et al. 1997, 2000). Several possible behavioral mechanisms of resistance to STDs have been proposed. Before copulation, individuals may inspect potential partners and avoid those with signs of infection, but many STDs involve carrier states with no visible signs (Holmes et al. 1994) consistent with theoretical models showing that both parasite and host have congruent interests in obscuring infection status (Knell 1999, Thrall et al. 1997). After copulation, behavioral mechanisms may be effective and less costly. Thus, postcopulatory oral-genital grooming has been shown to reduce STD transmission in male rats (Hart et al. 1987), and postcopulatory urination has been suggested as a mechanism to reduce STD transmission in humans (Donovan 2000, Hooper et al. 1978). In a comparative study of primates, however, postcopulatory genital grooming and urination showed no correlation with mating promiscuity (Nunn 2003, Nunn & Altizer 2003). Interestingly, the two primate radiations where genital grooming is more common are the small-bodied species (lemurs and callitrichids), suggesting that physical constraints may limit the evolution of this behavior in large-bodied species.

CONFOUNDING FACTORS

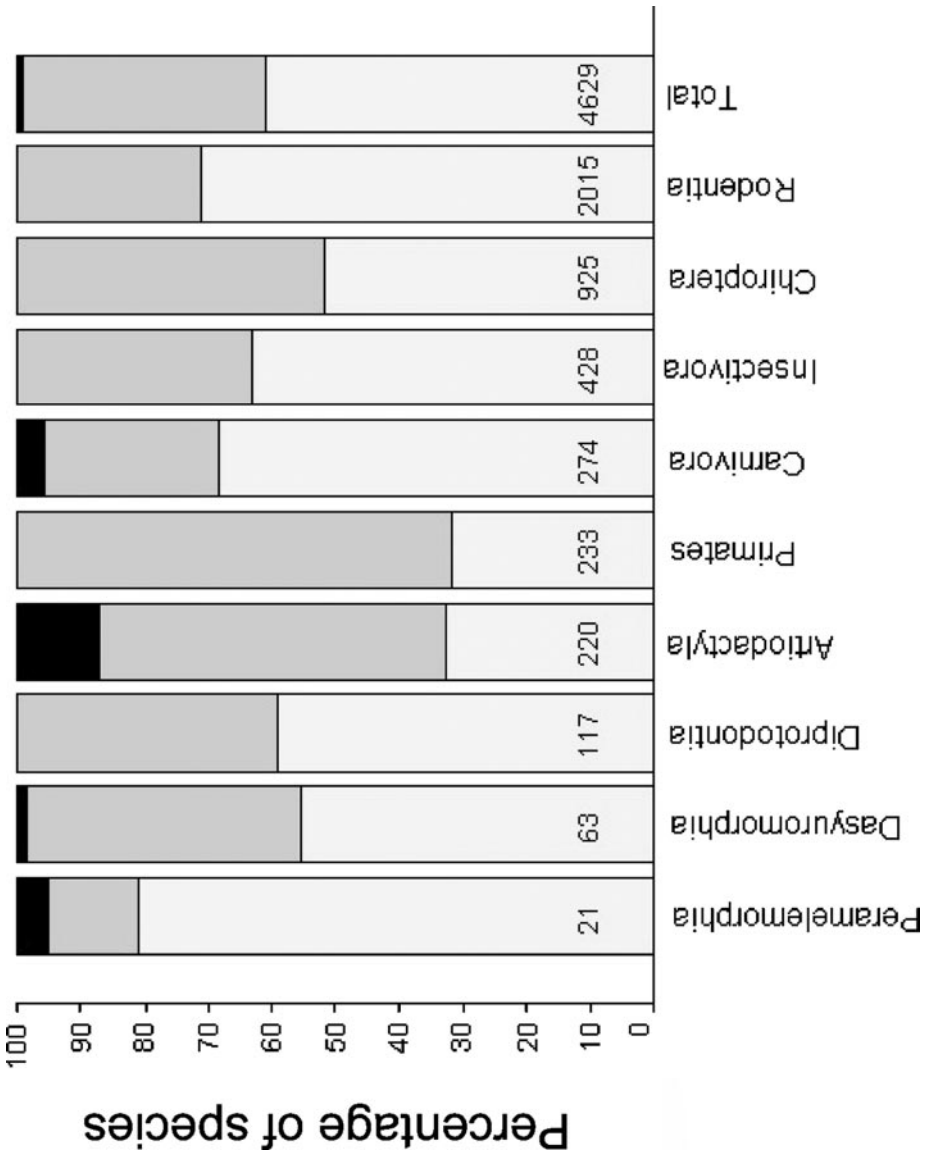
As noted throughout this review, a large number of ecological, life history, and behavioral traits of mammals should interact to influence parasite dynamics and diversity. For example, body mass is thought to result in increased parasite diversity because larger-bodied hosts represent larger "habitats" and provide more niches for colonization (e.g., Kuris et al. 1980, Poulin 1995). Many host characteristics predicted to influence parasite risk are themselves correlated across taxa, posing complications for comparative studies of multiple factors. As a case in point, large-bodied hosts of some mammalian orders, such as primates, tend to be terrestrial (Clutton-Brock & Harvey 1977, Nunn & Barton 2001) and most have "slow" life histories (e.g., increased longevity, delayed age at first reproduction). Disentangling the effects of body mass, life history, substrate use, and social organization therefore requires multivariate statistical models tested across multiple host and parasite groups.

Confounding variables are problematic in both field and comparative studies, but in comparative studies, two additional issues arise. First, closely related hosts may harbor similar numbers of parasites because of common ancestry rather than similar behavioral or ecological traits. This effect may result from specialist parasites that cospeciate with their hosts, and from geographical proximity among hosts that share generalist parasites. Methods for incorporating phylogenetic history are now well developed (Harvey & Pagel 1991, Martins & Hansen 1996), although debate on when correction for phylogenetic relatedness is overly conservative, or perhaps even misleading, continues (Harvey & Rambaut 2000, Westoby et al. 1995). Second, host species may differ in the size and diversity of their parasite communities because of uneven sampling effort, and many studies have shown that parasite species richness (the number of parasite species per host) is correlated positively with the degree to which host species have been examined for parasites. The most common approaches to control for uneven sampling effort are to use residuals from a linear regression of parasite species richness against host sample size or other measures of sampling effort (by a log-log transformation; Gregory 1990, Poulin 1998) or to include sampling effort directly as a predictor in multivariate models.

IMPLICATIONS FOR MAMMALIAN CONSERVATION

Management of parasites and infectious disease has increasingly become a focus in conservation biology (Cleaveland et al. 2002) because parasites can threaten already-reduced populations and because infectious diseases can trigger catastrophic declines in otherwise robust host populations. Severe negative impacts from introduced pathogens such as rabies (African wild dogs), canine distemper (African lions, black-footed ferrets), and phocine distemper (harbor seals) have occurred in recent decades, and reports of parasite outbreaks in wild populations are on the rise (Funk et al. 2001). Environmental factors such as habitat fragmentation, increased contact between wildlife and domesticated species, and climate change may further increase parasite prevalence and impacts (Daszak et al. 2000, Harvell et al. 2002). Nevertheless, global assessments of the causes and patterns of extinction risk often relegate the impact of parasites to "other causes" (MacPhee & Fleming 1999). Surprisingly, the *2002 IUCN Red List* (Hilton-Taylor 2002) does not include comprehensive records of parasites that threaten wild host species even though many listed species are known to have experienced recent declines or challenges from infectious diseases (Figure 6).

Increasing human population size and encroachment on native habitats will influence the impacts and emergence of infectious diseases in wildlife in several ways (Daszak et al. 2000, Dobson & Foufopoulos 2001). First, encroachment by humans alters animal foraging and social behavior, leading to increased stress and greater risk of acquiring infectious disease. Second, crowding animals onto wildlife reserves may further increase rates of parasite transfer among species. Finally,


Percentage of species
100
90
80
70
60
50
40
30
20
10
0
Peramelemorphia
Dasyuromorphia
Diprotodontia
Artiodactyla
Primates
Carnivora
Insectivora
Chiroptera
Rodentia
Total
21
63
117
220
233
274
428
925
2015
4629

infectious disease may spread from domesticated species to their close relatives in the wild. In fact, many species listed as at risk from infectious disease in the *IUCN Red List* (Figure 6) have largely acquired their parasites from domesticated species.

Which Parasites Pose the Greatest Threats?

The potentially large number of parasites shared by humans, domesticated animals, and wildlife creates severe problems for conservation management. As populations decline or become fragmented, specialist parasites may be lost, and generalist parasites present in overlapping reservoir populations may pose a more significant threat to endangered species (Cleaveland et al. 2002). In carnivores, for example, most extinctions or near-extinctions are caused by generalist pathogens rather than specialists (Woodroffe 1999). During the past decade, rabies and canine distemper virus, both of which infect domestic dogs as reservoir hosts, have emerged as significant pathogens of wild carnivores in the Serengeti ecosystem. The persistence and impact of both of these viruses is expected to increase because domestic dog populations are growing rapidly in many African countries (Cleaveland 1998). Thus, an understanding of parasite specificity, with detailed understanding of host geographical and niche overlap, should play an important role in conservation efforts.

Transmission mode is also important for determining which parasites pose the greatest threats to declining host populations. For many directly transmitted parasites, persistence is unlikely at low densities without an alternative host or reservoir, and parasite-driven host extinction is questionable. However, the expectation is quite different for STDs, as these are characterized by frequency-dependent transmission and high levels of host sterility (Thrall et al. 1993, Lockhart et al. 1996). Parasites transmitted via mobile vectors or contaminated water may also pose unusually high risks to small or threatened populations, as these may spread rapidly to multiple host species given favorable environmental conditions (Woolhouse et al. 2001).

The Importance of Host Characteristics

For identifying host traits associated with both the risk of parasite infection and extinction risk, carnivores represent a well-studied group (Funk et al. 2001). Small

Figure 6 Relative risk of infectious disease as a conservation threat in selected mammalian orders. White represents the proportion of nonthreatened species in each clade, gray and black combined represent proportion of threatened (including species classified as extinct in the wild, critically endangered, endangered, vulnerable, lower risk–conservation dependent, and lower risk–near threatened), and black only represents the proportion of species where infectious disease is identified as a threatening process. Numbers at the bottom of each bar refer to the number of species in each clade. Clade definitions follow Wilson & Reeder (1993) and threat data are from Hilton-Taylor (2000).

and declining carnivore populations are especially prone to extinction risk by infectious diseases (Woodroffe 1999), and the interactive effects of small population size and parasite infections on extinction risk are observed in many carnivore species including African wild dogs and black-footed ferrets (Williams et al. 1992). One hypothesis is that mammalian orders with the largest numbers of domesticated species (or species that associate with humans; e.g., ungulates, carnivores, and rodents) may experience disproportionate risks of pathogen introduction (Figure 6). For example, even though canine distemper virus did not originally extend to felids other than in captive populations, in 1994, an outbreak in African lions that originated in domestic dogs reduced the lion population by about 30%. This virus also migrated from the Serengeti to another population in the Masai Mara, where eventually 55% of lions became seropositive (Roelke-Parker et al. 1996). Thus, certain mammal groups may be especially hard hit because domesticated animals and humans act as reservoirs of virulent parasites (Wallis & Lee 1999, Woodford et al. 2002).

Parasites and Biodiversity

Relative to other processes (such as habitat destruction, invasive species, overharvesting, and pollution), we know far too little about the effects of parasites on the processes of mammalian declines and extinction. Wilcove et al. (1997) assessed the influences of each of these sources of threat on approximately 2500 imperiled and federally listed species in the United States. Of information available on 1880 species, infectious disease represented the least important threat. It is doubtful that parasites play such a minor role, and it remains to be determined if these numbers reflect poor knowledge of infectious disease dynamics in natural populations rather than the actual impact of parasites in these groups.

The concern over parasite specificity and transmission highlights the importance of understanding broad patterns of parasite community diversity in wildlife. At present, only a small proportion of parasites infecting mammals have been identified and quantified in wild populations. For example, the African wild dog has received tremendous scientific study and popular attention (see Woodroffe et al. 1997) owing to observed catastrophic effects of parasites (such as rabies wiping out a pack of 21 individuals in less than two months; Kat et al. 1995), yet only a small number of parasites are known to potentially influence rates of population decline. Furthermore, the epidemic nature of certain pathogens makes recording parasite communities challenging. Some populations may not be affected for long intervals but then experience high fatality rates over a period of days, as was recently evidenced by outbreaks of phocine distemper in northern European seals (Jensen et al. 2002).

In considering the totality of biodiversity, it is also important to include the impacts of mammalian extinctions on the biodiversity represented by their parasites. Many parasites are specific to endangered or threatened mammals, and may themselves become extinct with their specific hosts (Gompper & Williams 1998). Moreover, hosts that lose their parasites during population bottlenecks or in captive

breeding programs may also lose their ability to respond to future parasite threats following relaxed selection for immune defenses (Cunningham 1996). Lobbying efforts dedicated to preventing losses of mammalian diversity perhaps should take up the cause in defending pathogen biodiversity as an integral component of free-living host communities, particularly given the role parasites may play in shaping host phenotypic and genetic composition or their importance as potential sources of pharmaceuticals (Durden & Keirans 1996).

FUTURE DIRECTIONS

Interdisciplinary collaboration is crucial to advance our understanding of infectious disease ecology and evolution in mammalian populations. When dealing with complicated and poorly understood systems, it is always tempting to attribute causation to the most easily measured factor. In spite of recent advances in serology, PCR, and other detection methods, parasite infection—and its ecological and genetic correlates—are factors that are among the hardest to measure in natural populations. We are therefore left with a high probability of either over- or understating the importance of parasite infections based on superficial impressions. Rigorous studies are needed to understand the ecological and evolutionary roles that parasites play in natural populations. With this in mind, how can theory and data be more explicitly linked to study host and parasite features in mammalian systems? We outline several priorities for future research in this area, focusing particularly on increased correspondence between variables in theoretical models and empirical data from wild hosts and the development of new methods to assess the joint coevolution of host and parasite traits.

(**1**) **Expand empirical records of parasites from wild mammal populations.** Baseline data on the prevalence of parasites in wild mammals are critical to examining links between host behavior and parasite transmission. For most mammals, extensive data are rarely collected for nondomesticated animals outside of captive settings. For example, a recent survey of published records of nonexperimental parasite infections across 200 primate species (C. Nunn & S. Altizer, unpublished data) showed that the vast majority of records were obtained from captive animals (sampled in zoos, research centers, or semifree ranging populations). There is also a need for a centralized data repository for records of micro- and macroparasites in wild animals and monitoring programs that assess the prevalence, transmission, and population-level impacts of parasites infecting a wide range of mammalian species.

(**2**) **Improve the correspondence between behavioral data and model parameters.** At the present time, it is difficult to relate categorically defined mating systems (e.g., polygyny, serial monogamy) and social structures (e.g., solitary, fission-fusion communities) to the spread of parasites in wild populations. More precise measures of parameters suggested by theoretical

models are needed from wild mammal populations, including inter- and intragroup contact rates, dispersal rates, contact durations, and better measures of variance in mating success. Moreover, model parameters that define contacts leading to parasite transmission must reflect biologically realistic and estimable processes, a goal that can only be achieved by increasing interactions between behavioral ecologists and epidemiologists.

(3) **Include multiple factors in comparative analyses.** Increasing numbers of comparative studies are including measures of sampling effort and controlling for host phylogeny. However, to date most comparative studies of factors affecting parasite occurrence have examined only a small number of explanatory variables. Important variables to consider in the context of parasite diversity studies include not only host traits, such as life history and sociality, but also parasite traits involving transmission mode and host range. For example, the diversity of STDs should be influenced by factors that are different from those important to parasites transmitted by fecal-oral routes. Ignoring the confounding effect of transmission mode may therefore result in failure to detect traits important to patterns of parasite diversity.

(4) **Develop trait-based coevolutionary models for host-parasite interactions.** A major challenge involves developing modeling approaches to explore the joint coevolution of transmission mode, mating and social systems, and pathogen virulence. One approach is to acquire phylogenetic information on coevolving host and parasite lineages to examine the correlated evolution of host and parasite traits (Harvey & Keymer 1991, Morand et al. 2000). A complementary modeling approach would use individual-based rules for association and dissociation of males and females to generate a wide range of social and mating structures (e.g., Cohen 1969). A key issue is how to represent genetic variation for host behavior in a biologically realistic way that results in observed social and mating systems. Behavioral ecologists have extensively discussed verbal and optimality-based models, but overlook the fact that it is unrealistic to describe selection on genes that directly translate to specific mating systems. To our knowledge, no empirical studies have examined whether genetic variation in either host social/mating behavior or parasite effects on such behavior exists in wild mammal species.

(5) **Evaluate the role of spatial and metapopulation processes.** Spatial considerations are important in defining the geographic scales at which social and sexual interactions take place. There are numerous situations in wild animal populations where substructuring occurs at multiple scales. For example, hamadryas baboons (*Papio hamadryas*) have a polygynous mating system, where related males and their harems are themselves organized into larger clans; these clans are organized into yet larger groups, which may interact socially in various ways. A number of critical questions relating to the ways in which the relative transmission of STDs versus OIDs might

scale with the level of social hierarchy arise from considering these social structures. How do the overall population dynamics and persistence of parasites depend on interactions within and among these levels of organization? How would this affect group dynamics and the types of social/sexual systems that evolve? It has been suggested that interactions between primate social systems and the distribution of resources and predators will determine social organization (Dobson & Lyles 1989). Clearly, these ideas apply with equal force to pathogens and parasites.

(**6**) **Examine the links between host sociality, parasite infection, and extinction risk.** Infectious diseases have become an important threat to wildlife populations, and links between sociality and parasite transmission suggest that host behavior might play an indirect role in species extinctions. Despite the possible interaction among these factors, to our knowledge no studies have examined characteristics of parasites associated with extinction risk or whether host social organization influences the spread of novel pathogens. For example, are social species more likely to be threatened by novel parasites than nonsocial species? These questions can be answered by integrating information already available from veterinary field surveys with the results of empirical studies on host behavior and population dynamics. An understanding of the interactions between sociality, disease transmission, and extinction risk could provide important insights for wildlife conservation.

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