7 • Sexual selection, behaviour and sexually transmitted diseases

CHARLES L. NUNN
Department of Biology
University of California
Davis, CA, USA

SONIA M. ALTIZER
Department of Environmental Studies
Emory University
Atlanta, GA, USA

INTRODUCTION

Factors that alter the contact structure of individuals within populations will influence the spread of parasites that are transmitted by direct contact (Anderson & May, 1991; Blower & McLean, 1991). Few cases illustrate this fundamental principle of epidemiology better than sexual selection and the spread of sexually transmitted diseases (STDs). Sexual selection involves variation in mating success mediated by male–male competition or female choice. By changing the structure of mating contacts within a population, sexual selection influences the spread of sexually transmitted infections. In particular, those individuals with the greatest mating success are at highest risk of contracting STDs, and will also contribute disproportionately to STD spread and persistence (Graves & Duvall, 1995; Thrall *et al.*, 2000). Moreover, promiscuity associated with sperm competition is predicted to increase both the spread and virulence of STDs (Thrall *et al.*, 1997). Therefore STDs may represent a substantial cost of sexual selection and non-monogamous mating behaviour (Thrall *et al.*, 2000).

Sexually transmitted diseases have been virtually ignored in studies of animal mating systems (Smith & Dobson, 1992; Lockhart *et al.*, 1996), but it is now possible to link epidemiological theory on STDs to patterns of infection in wild populations. In this chapter, we explore the consequences of sexual selection, for the spread of STDs in primates. We also examine behavioural defences to avoid infection, specifically addressing interactions between parasite fitness and host reproductive success. Primates represent an ideal system for studying STDs and sexual selection, because of the large amount of data available on their parasites and mating behaviour. It is important to note that, throughout this chapter, we apply the general term 'parasite' to any organism that lives in or on a host and utilises host resources, usually to the detriment of the host (thus including viruses, bacteria, protozoa and fungi, in addition to more traditionally defined helminths and arthropods).

First, we review evidence for STDs in non-human primates and the effects of STDs on their hosts. This is an important step, because if STDs are extremely rare or have only minor effects on host fitness, they are unlikely to represent a primary selective force influencing mating behaviour. Second, we summarise the results of a simulation study that examined the spread of STDs within populations, assuming high variance in male mating success, as expected under sexual selection (Thrall *et al.*, 2000). We test the key prediction of this model using data on prevalence of STDs in free-living primate populations. Third, we review a theoretical model that investigates the effects of mate choice on virulence evolution in STDs (Knell, 1999). This model suggests that mate choice to avoid STD infection will reduce the virulence of sexually transmitted parasites. Because the spread of STDs is sensitive to sexual selection, it might seem that male viability traits, such as those predicted under the Hamilton–Zuk (1982) model, should be particularly fine-tuned to STD infection. However, Knell's model provides the opposite conclusion and raises more general questions about behavioural counter-strategies to STDs. Thus, in the final section of this chapter we investigate the consequences of STDs for host behavioural defences by examining comparative patterns of putative behavioural counter-strategies in relation to STD risk (Nunn, 2003, in press). In so doing, we distinguish between pre-copulatory and post-copulatory defensive behaviours, and we examine the prediction that post-copulatory behaviours will be more effective than those performed prior to mating.

We use these diverse questions to illustrate the conceptual links between sexual selection, host behaviour and the spread of STDs. Moreover, we show that sufficient comparative data are emerging to test theoretical models, although our results must be considered initial explorations of these

Sexual Selection in Primates: New and Comparative Perspectives, ed. Peter M. Kappeler and Carel P. van Schaik. Published by Cambridge University Press.

questions. A major goal of our chapter is to highlight the types of data needed for future tests. We also discuss the ways in which existing theoretical models should be extended to consider the spread of parasites in primate populations, particularly with regard to polygynandrous mating systems found in many primate species.

DO STDs OCCUR IN WILD PRIMATE POPULATIONS?

STDs typically have been viewed as a curious group of parasites rather than established entities with important selective effects on their hosts (Lockhart *et al.*, 1996). In recent decades, this view has changed, primarily through our increased understanding of HIV in the context of the AIDS crisis (e.g. Garnett & Anderson, 1993; Schwartländer *et al.*, 2000). It is now well established that the simian form of HIV, the simian immunodeficiency virus (SIV), is found in many Old World monkey and ape species and appears to show a high degree of strain specificity (e.g. Phillips-Conroy *et al.*, 1994; Hahn *et al.*, 2000; Santiago *et al.*, 2002). As in humans, SIV is spread primarily through sexual contact, but the relative importance of other transmission routes (e.g. biting) may vary among species or environmental conditions (Nerrienet *et al.*, 1998). Unlike HIV in humans, SIV is not known to cause severe immunodeficiency or mortality in its native hosts (Norley *et al.*, 1999; Swanstrom & Wehbie, 1999).

Several reviews have examined the distribution of STDs across host species, but the most comprehensive review at time of going to press was conducted by Lockhart *et al.* (1996). These authors surveyed the veterinary, medical and parasitological literature and found evidence for over 200 STDs in 27 orders of hosts. Parasites that exhibit sexual transmission represented most major taxonomic groups, including viruses, bacteria, helminths, protozoa, fungi and arthropods. For primates, our own literature search on nearly 200 host species confirms the conclusion of Lockhart *et al.* (1996), that STDs are better studied in Old World monkeys. In many of the species in which STDs have been documented, individuals mate promiscuously, and none of these species is typically classified as monogamous. These phylogenetic and social correlates are not definitive, however, because surveys of STDs in wild populations may be biased toward group-living, promiscuous primates. In fact, wild populations of most primate species have not yet been examined for STD infection. As a consequence, we cannot conduct broad-scale comparative tests of STDs in primates, but analyses within subsets of taxa are possible, as illustrated below.

HOW COSTLY ARE STDs?

Parasites differ markedly in their effects on host fitness (i.e. virulence), and much of this variation is predicted to relate to transmission mode (Ewald, 1994; Herre, 1995; Sorci *et al.*, 1997). This is particularly important for parasites with limited transmission routes (e.g. STDs), where negative effects on host survival or conspicuous signs of infection may reduce new transmission events below threshold conditions for establishment or persistence (Getz & Pickering, 1983; Thrall *et al.*, 1993). As a result, some STDs, such as those caused by retroviruses, may simply hitchhike along with their hosts, causing little harm and resulting in few host counter-strategies (Norley *et al.*, 1999). We acknowledge that many STDs are relatively benign in relation to other parasites. But recent analyses by Lockhart *et al.* (1996) and others (Smith & Dobson, 1992; Holmes *et al.*, 1999) demonstrate at least three major costs of STDs: (1) A large proportion of STDs increase the risk of sterility in males or females. (2) STDs commonly exhibit vertical transmission, with severe consequences for offspring health. In humans, for example, syphilis causes congenital defects that are likely to reduce the survival and reproductive success of offspring (Radolf *et al.*, 1999). Similarly detrimental effects are found in infants that contract papilloma virus, gonorrhoea, herpes and HIV from their infected mothers (see chapters in Holmes *et al.*, 1999). (3) Relative to infectious disease transmitted by non-sexual contact, STDs commonly exhibit long infectious periods with low host recovery, failure to clear infectious organisms following recovery, or limited immunity to reinfection. This pattern arises because many sexually transmitted parasites (like some non-STDs) have mechanisms of 'hiding' from host immune defences (e.g. persisting in neural ganglia; Hoeprich *et al.*, 1994). In fact, many STDs are impossible to eradicate from the body through the immune response alone, resulting in lifetime infections.

Many negative consequences of STD infection probably provide benefits to the parasites themselves, increasing the likelihood of invasion, transmission and persistence (see Lockhart *et al.*, 1996). In mammals, for example, host infertility is likely to result in repeated cycling by females and may consequently increase their number of sexual contacts. Primates offer an important opportunity to test this hypothesis, because the frequency of infertile females within wild groups may exceed 10 per cent (Anderson, 1986). Similarly,

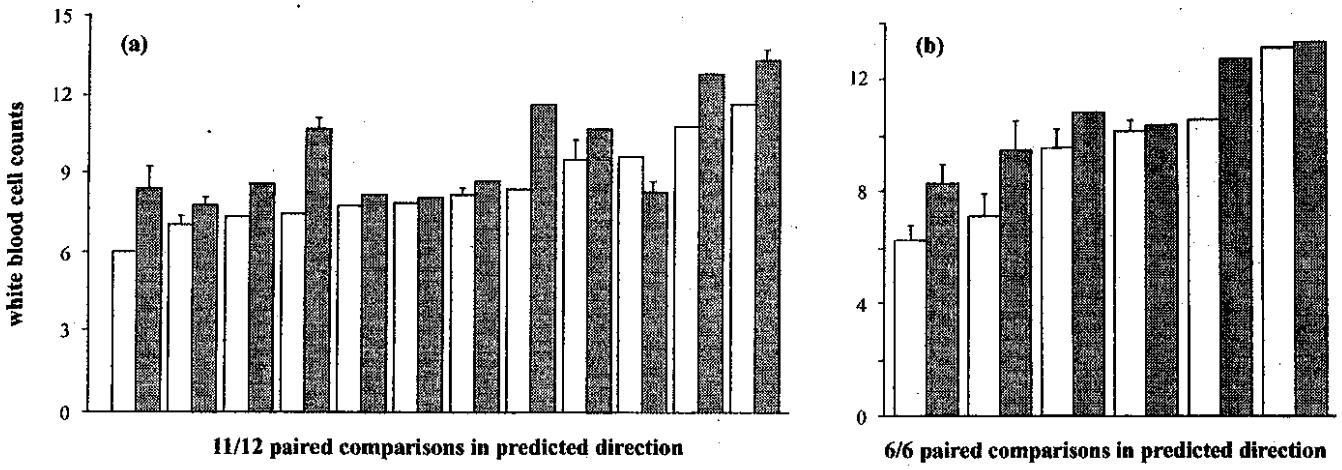

Fig. 7.1 White blood cell counts in primates (a) and carnivores (b). Bars show phylogenetically independent comparisons of less promiscuous (open bars) and more promiscuous (filled bars) species. Error bars are provided when comparisons involve phylogenetically weighted mean values for more than one species. The prediction to test is whether evolutionary transitions to increased promiscuity show increases in white blood cell counts. Results are significant at $P < 0.05$ (Nunn, 2002; Nunn *et al.*, 2003), with comparisons calculated using the BRUNCH method in the computer program CAIC (Purvis & Rambaut, 1995).

STDs that increase host mortality or possess short infectious periods are less likely to survive until the next breeding season, when contact is established with new, uninfected hosts (e.g. Thrall *et al.*, 1993). Thus, in addition to long infectious periods, STDs tend to produce less disease-induced mortality relative to other infectious diseases (Lockhart *et al.*, 1996).

Another approach to assessing the costs of STDs is to examine their effects on host defences. Nunn *et al.* (2000; Nunn, 2002) conducted comparative tests across a diverse assemblage of primates to assess whether baseline white blood cell (WBC) counts are associated with risk of STD infection. White blood cell counts among healthy, captive animals may indicate the capacity of innate immune cells (monocytes, granulocytes or natural killer cells) to respond quickly to parasite infection. Such generalised defences could be critical to STD prevention because antibody-mediated immune responses are unlikely to be effective in eradicating venereal diseases following establishment. Consistent with predictions that more promiscuous species of primates should experience increased risk of STD infection, species with greater promiscuity exhibited higher baseline WBC counts in phylogenetic comparative tests (Nunn *et al.*, 2000; Fig. 7.1). These results were upheld when using different measures of promiscuity, after controlling for other sources of infection, such as terrestrial locomotion and sociality, when limiting the analysis to adult females or males only, and in an independent data set on WBC counts (M. J. Anderson, J. Hessel & A. F. Dixson, unpublished). Moreover, analyses were repeated in carnivores and found to be consistent with the patterns in primates, although it proved difficult to rule out confounding effects of sociality, life history and body size in carnivores (Nunn *et al.*, 2003, in review).

One mechanistic explanation for the association between promiscuity and primate immune defence involves the interaction between WBCs and sperm in the female reproductive tract. Immediately following copulation, massive numbers of WBCs flood the female reproductive tract and actively engulf sperm and seminal fluid (e.g. Phillips & Mahler, 1977; Pandya & Cohen, 1985; Barratt *et al.*, 1990). Neutrophils are a primary phagocytic WBC central to this process, a fact that is relevant because analysis of neutrophil counts provided the most consistent results in phylogenetic comparative tests in primates and carnivores (Nunn *et al.*, 2000; Nunn, 2002). Given that STDs are present in seminal fluid (Holmes *et al.*, 1999), a plausible interpretation is that active and immediate phagocytosis of ejaculate functions to reduce the risk of STD infection. It is important to note, however, that sperm destruction may involve other functions, including cryptic female choice (Eberhard, 1985).

Further research is needed to document the occurrence and epidemiology of STDs in wild animal populations. Information is also needed on the costs of infection for different STDs in their natural hosts. The limited information that is available demonstrates that STDs are present in non-human primates and can have a considerable impact on host fitness, thus representing an important selective force in wild populations.

SEXUAL SELECTION AND PATTERNS OF STD SPREAD

As noted above, STDs spread through populations via networks of sexual contacts. By affecting mating patterns within populations, sexual selection should be a major determinant of how STDs spread through animal populations (Thrall *et al.*, 2000). The spread of STDs will be influenced by either male–male competition or female choice, with the critical variable being the variance in mating contacts in the two sexes. The following questions therefore arise. Does sexual selection affect the prevalence of STDs within populations? Do males and females exhibit different infection rates? How do other host characteristics, such as dispersal and life-history parameters, influence the prevalence of STDs?

Several investigators have addressed these questions using epidemiological models. For example, a recent paper by Thrall *et al.* (2000) examined disease spread in the context of a polygynous mating system applicable to mammals. The authors used a simulation model to investigate the spread of STDs in males and females with respect to variance in male mating success, transfer of females among groups, and adult mortality rates. Variance in male mating success simply represents sexual selection, which is the focus of this chapter, but dispersal and mortality also are expected to modify the distribution of infections within and among groups. Dispersal is a key predictor of disease spread that is particularly relevant to primates because most (but not all) primate matings take place within social groups. Thus, greater dispersal increases the prevalence of infection in the entire population because this allows greater mixing among infected and uninfected sub-populations. Host mortality is important because it influences the duration of infectiousness: when an infected host dies, the parasite dies with it. Thus, higher host mortality is expected to reduce prevalence.

It is important to note that data required to estimate key variables in the model by Thrall *et al.* (2000) – reproductive skew among males, female dispersal and adult mortality – are available for many free-living animal populations. As applied to primates, however, the assumptions of the model do not fit all types of mating systems, with polygynandrous groups noticeably absent. To the extent that variance in mating success determines the spread of STDs, the model makes some useful predictions for primate groups in initial tests. This is because high variance in male mating success within polygynandrous groups may yield transmission dynamics similar to the polygamous systems described below.

Simulations by Thrall *et al.* (2000) were conducted by randomly assigning 'attractivity scores' to each of 250 males in a simulated population. These values were drawn from a log-normal distribution, with increasing variance in this distribution, reflecting increasing sexual selection. The same total number of females was assigned to these males in proportion to each male's attractivity code. Dispersal was accomplished by drawing a random subset of females to transfer to other groups, with assignment to groups proportional to male attractivity scores. Thus, female dispersal provides a means for the STD to spread outside polygynous groups and infect males in other groups. Mortality was simulated by eliminating a set number of males and females and replacing them with healthy individuals of the same sex. If a male died, his group of females was dissolved and reassigned to other males. Disease transmission was assumed to be a function of mating probabilities between healthy and infected hosts, number of copulations, and per-mating transmission probabilities. Thus, by systematically varying key parameters representing sexual selection, dispersal and life history, Thrall *et al.* (2000) examined the spread of STDs within and among mating groups.

Model simulations showed that increasing variance in male mating success resulted in higher prevalence for both males and females. In addition, STD prevalence tended to be higher in females than males, and this difference increased with greater variance in male attractiveness. An intuitive explanation for this outcome is that as sexual selection increases, a smaller percentage of males in the population actually mate, generating lower prevalence among males than females. The simulations also revealed that greater among-group dispersal increased population-wide prevalence, whereas increased mortality reduced overall prevalence. These simulation results make predictions that can be tested comparatively. We acquired published data on STD prevalence in wild primates, using only studies with prevalence data separated by sex among adult individuals (Table 7.1). Because quantitative data were not available on dispersal or adult mortality for each population, we focus here on the key prediction, namely that when variance in male mating opportunities is greater than variance in female mating opportunities (i.e. sexual selection on males), the prevalence of STDs should be higher in females than in males. The detailed information that we required was available for only two STDs, both of which are retroviruses: SIV and simian T-cell lymphoma/leukaemia virus (STLV).

Table 7.1 *Prevalence of two retroviruses, SIV and STLV-1, in free-living primates.*[a]

Species	Locality–group	Parasite	Prevalence (males)	Number of males	Prevalence (females)	Number of females	Reference
Cercopithecus aethiops	Saloum Delta National Forest, Senegal	SIV	0.64	14	0.95	21	Bibollet-Ruche *et al.*, 1996
Cercopithecus aethiops	Samburu, Kenya	STLV	0.69	26	0.68	38	Drapcoli *et al.*, 1986
Cercopithecus aethiops	Mosiro, Kenya	STLV	0.6	5	1.00	13	Drapcoli *et al.*, 1986
Cercopithecus aethiops	Naivasha, Kenya	STLV	0.45	11	0.61	33	Drapcoli *et al.*, 1986
Cercopithecus aethiops	Kimana, Kenya	STLV	0.78	18	0.67	15	Drapcoli *et al.*, 1986
Cercopithecus aethiops	Awash National Park, Ethiopia	STLV	0.12	26	0	35	Drapcoli *et al.*, 1986
Cercopithecus aethiops	Fathala Forest, Senegal, Group P	SIV	0.71	7	0.90	10	Galat-Luong *et al.*, 1994b
Cercopithecus aethiops	Fathala Forest, Senegal, Group G	SIV	0.33	3	1.00	5	Galat-Luong *et al.*, 1994b
Cercopithecus aethiops	Awash National Park, Ethiopia, 1990–93	SIV	0.76	37	0.97	33	Jolly *et al.*, 1996
Cercopithecus aethiops	Awash National Park, Ethiopia, 1973	SIV	0.61	18	0.79	29	Jolly *et al.*, 1996
Colobus guereza	Cameroon	SIV	0.27	11	0.44	9	Courgnaud *et al.*, 2001
Erythrocebus patas	Saloum Delta National Forest, Senegal	SIV	0.14	7	0.07	14	Bibollet-Ruche *et al.*, 1996
Erythrocebus patas	Fathala Forest, Senegal	SIV	0.17	6	0.09	11	Galat-Luong *et al.*, 1994a
Macaca fascicularis	Indonesia	STLV	0.36	28	0.14	36	Ishikawa *et al.*, 1987
Macaca fuscata	Japan	STLV	0.35	314	0.43	719	Hayami *et al.*, 1984
Macaca fuşçata	Nagano Prefecture, Japan	STLV	0.23	13	0.55	20	Ishida *et al.*, 1983
Macaca maurus	Indonesia	STLV	0.28	18	0.43	14	Ishikawa *et al.*, 1987
Papio ursinus	Northern and Eastern Transvaal	STLV	0.30	56	0.25	52	Botha *et al.*, 1985
Macaca nemestrina	Southern Sumatra, Indonesia	STLV-1	0	5	0.15	55	Richards *et al.*, 1998

[a] Information was found on two viruses that are known to exhibit some degree of sexual transmission. Prevalence and sample sizes for males and females in wild populations are shown separately. Many studies reported estimates of both seroprevalence (the presence of anti-viral antibodies as determined by enzyme-linked immunosorbent assay (ELISA) and western blot analysis) and the presence of virus using polymerase chain reaction (PCR) methods with virus-specific primers. ELISA and PCR analyses provided consistent results (e.g. Richards *et al.*, 1998), probably because animals remained infected with these retroviruses for life (Lockhart *et al.*, 1996). Although existing evidence strongly suggests that SIV and STLV are sexually transmitted in wild and captive primates, other close-contact transmission (allowing exchange of bodily fluids) is possible and the degree of sexual transmission may vary among host species (e.g. Georges-Courbet *et al.*, 1996).

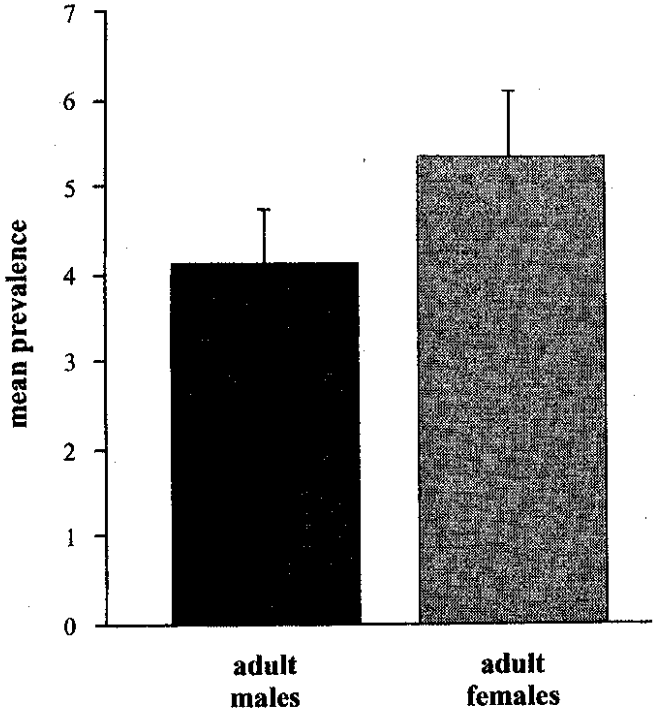

Fig. 7.2 Sex differences in prevalence of STDs in wild primates. Bars represent mean prevalence of two STDs in males and females, +1 *SE*. Data are from Table 7.1, and statistical results are provided in the text.

First, we tested whether prevalence was higher among adult than immature animals, an obvious expectation if the parasite is transmitted sexually. This prediction was consistently supported by all studies with necessary information (matched pairs $t_{17} = 7.95$, $P < 0.0001$, one-tailed; one study provided no data on immature animals and was therefore excluded). We then tested whether STD prevalence was greater in females than males, with analyses restricted to data from sexually mature adults. Again, the pattern was statistically significant (Fig. 7.2, matched pairs $t_{18} = 2.49$, $P = 0.011$, one-tailed). Twelve of the 19 studies showed the predicted pattern regarding sex differences.

These analyses represent an initial test of predictions, and prior to conducting more detailed analyses it is important to examine critically the assumptions and consider alternative explanations for our findings. First, we assumed that both SIV and STLV are transmitted sexually, but they also may be transmitted from infected mothers to infants (vertical transmission) or through aggressive interactions, such as biting (Nerrienet *et al.*, 1998). However, greater competition among males means that transmission via aggressive contacts is expected to bias patterns opposite to our findings, suggesting that this is not an alternative explanation for the results. Second, we assumed that transmission probabilities are equal in males and females. Currently we lack quantitative data on the mechanisms of transmission for these STDs in their natural hosts, but retroviruses in humans often show higher transmission rates from males to females than vice versa (Alexander, 1990; Padian *et al.*, 1997). Differential transmission probabilities could be incorporated in future simulation studies to refine the comparative predictions. Finally, the predictions that we tested were generated from simulations of disease spread in polygynous mating systems, whereas many of the species in Table 7.1 are polygynandrous, in which more than one male breeds with a group of females. Violations of this assumption are likely to have minor consequences in the present case, however, because males within multi-male groups also show striking variance in mating success, limiting transmission opportunities to one or a few dominant males (Cowlishaw & Dunbar, 1991).

CAN STDs BE AVOIDED THROUGH MATE CHOICE?

One expected behavioural consequence of infection by an STD would involve increased host sexual activity or attractiveness of infected hosts. Although such modification of host sexual behaviour would increase STD transmission, few examples involving direct manipulation by parasites have been documented (but see Møller, 1993). Dourine, a sexually transmitted trypanosome of horses, is one parasite thought to increase sexual activity of stallions (Thrall *et al.*, 1997), thus increasing the probability of sexual transmission. Other evidence suggests that STD infection increases the duration of oestrus in cows (bovine genital campylobacteriosis; Roberts, 1979). However, examples are fragmentary and inconsistent (Webberley *et al.*, 2002), and more detailed studies are needed.

Because sexual reproduction offers an important mechanism for disease spread and may even be influenced by infection status, it is pertinent to ask whether animals can identify infected individuals and avoid mating with them. Symptoms such as visible lesions, sores, discharge around the genitalia or olfactory cues may provide evidence of infection. Infection cues might work both ways, with females inspecting males and males inspecting females. If STDs influence the expression of secondary sexual characteristics, then parasite-mediated sexual selection may play a role in female avoidance of infected partners (Hamilton & Zuk, 1982), in addition to more general contagion-avoidance mechanisms (Able, 1996).

Application of either framework requires that STDs produce reliable indicators of infection so that potential partners can identify infected individuals. We know of no studies that have examined mate choice in relation to STD infections in vertebrates. In two species of beetle, however, potential partners showed no evasion of mates that were infected with sexually transmitted mites (Abott & Dill, 2001; Webberley *et al.*, 2002).

In humans, STDs (such as genital herpes) are notorious for producing unpleasant symptoms that could be detected by potential mating partners. Despite this dogma, many human STDs are more frequently characterised by limited symptoms or, in the case of viruses, asymptomatic shedding (Holmes *et al.*, 1999). The most prominent example of this is HIV, in which infection status (prior to advanced stages) can be determined only through medical tests that detect the virus or host antibodies in blood or other bodily fluids. The same absence of obvious symptoms is likely to characterise many non-human STDs (Lockhart *et al.*, 1996).

It might seem puzzling that STDs produce few severe or notable signs of infection, as compared to other diseases. A recent theoretical model by Knell (1999) sheds light on this issue by considering how STD virulence interacts with host mating success. Virulence has many definitions (Bull, 1994; Ebert, 1994; Ewald, 1994; Read, 1994; Herre, 1995). In the context of Knell's (1999) model, virulence reflects the degree to which the parasite produces symptoms in infected hosts. This is a reasonable assumption, given that increased virulence is likely to drain resources from the host, which then becomes unavailable for investment in sexual ornamentation and courtship displays. Knell's (1999) model shows that mate choice is unlikely to evolve as an effective mechanism for avoiding STDs, making parasite avoidance through secondary sexual characteristics or contagion-avoidance mechanisms less likely (Hamilton & Zuk, 1982; Able, 1996). The reason for this is that reproductive success of an STD is correlated with partner exchange and successful matings of infected hosts. Therefore, virulent parasites that produce outward signs of infection will experience decreased transmission because they provide conspicuous cues for choosy members of the opposite sex to avoid infected mates. Thus, Knell's (1999) model predicts that STDs will be less virulent, which is a general pattern that emerges when comparing STDs to non-sexually transmitted parasites (see Lockhart *et al.*, 1996). To state the conclusion differently, a host and its sexually transmitted parasites have congruent interests in facilitating host mating success, favouring low virulence among STDs.

BEHAVIOURAL COUNTER-STRATEGIES TO STDs

The above discussion raises the issue of behavioural counter-strategies more generally. Based on Knell's (1999) model and the evolutionary importance of mating success, we propose that pre-copulatory behaviours that limit exposure to STD infection will arise less frequently in natural populations than behaviours performed after copulation. In this section, we review recent comparative results (Nunn, 2003) that examine a range of STD-avoidance behaviours available to primates before and after mating.

Figure 7.3 illustrates the basic process of dispersal and invasion for any kind of parasite. A parasite faces two main barriers, or defences, imposed by the host: behavioural counter-strategies to avoid exposure, and physical or immune defences (including both innate and acquired immunity). The order of events can vary, but behavioural mechanisms commonly are viewed as the first line of defence. An important point we wish to emphasise is that host behaviour to avoid exposure prior to mating is likely to have other reproductive costs, and these costs may outweigh their benefits. Three examples support this point. First, individuals of either sex could reduce STD risk by limiting their number of mating partners (Loehle, 1995; Thrall *et al.*, 1997), but this is likely to be costly in terms of reproductive success. For a female, failure to mate multiply may increase the risk of infanticide committed by males with whom she did not mate while fertile (Hrdy, 1979; Hausfater & Hrdy, 1984; van Schaik & Janson, 2000). For males, missed mating opportunities directly impact reproductive success by reducing the number of offspring potentially sired. Second, individuals could reduce the number and duration of copulatory bouts with each mating partner (e.g. Hooper *et al.*, 1978; Sheldon, 1993; Thrall *et al.*, 1997). However, copulatory patterns in primates are likely to have been shaped by selection to increase fertilisation success, for example by sperm competition (Dixson, 1998); hence, manipulating these parameters is likely to be costly. Finally, simulations by Thrall *et al.* (2000) show that males who are more successful in sexual selection are more likely to be infected with an STD. Thus, females could avoid mating with successful males to reduce STD risk (Graves & Duvall, 1995). However, these females would be sacrificing the direct and indirect benefits of mating with successful males.

In addition to pre-copulatory behaviours, animals possess post-copulatory anti-parasite behaviours that may exhibit fewer reproductive trade-offs in the context of sexual

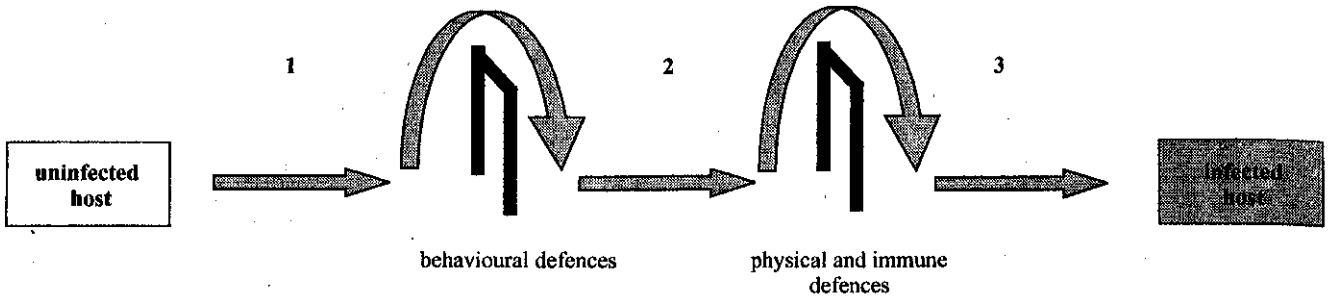

Fig. 7.3 Parasite transmission. Generalised steps required for successful infection of susceptible hosts: (1) encounter with infected host or infectious material, (2) exposure of susceptible tissues to parasites; and (3) successful invasion of host and evasion of immune defences. A parasite must therefore overcome two main lines of host defences in transmission and invasion: behavioural defences that involve avoiding contagion or overcoming infection (e.g. Richards *et al.*, 1998), probably because animals remained infected with these retroviruses for life (Lockhart *et al.*, 1996); and physical and immune defences to prevent and eliminate infections. Although existing evidence strongly suggests that SIV and STLV are sexually transmitted in wild and captive primates, other close-contact transmission (allowing exchange of bodily fluids) is possible and the degree of sexual transmission may vary among host species (e.g. Georges-Courbot *et al.*, 1996), overcoming infection, and defences that provide physical barriers to infection and the immune response.

selection. In what follows, we consider two post-copulatory behaviours: oral self-grooming of the genitalia and post-copulatory urination. Oral self-grooming of the genitalia is common in many mammals after mating, and has long been known to occur in prosimians (e.g. *Lemur catta*: Jolly, 1966). It has now been established that saliva has anti-bacterial and anti-viral properties (Baron *et al.*, 2000), and grooming in rats has been shown to reduce transmission of STDs (Hart *et al.*, 1987). In many human societies, genital washing is practised before and after sex (Donovan, 2000a, b). Urination also is a human folk remedy for prevention of STDs (Donovan, 2000b) and is commonly practised after 'risky' sex (Hooper *et al.*, 1978). This behaviour may be more effective for males than females because the urethra is the primary site of infection for some STDs in males (Holmes *et al.*, 1999), whereas females have a larger mucosal area for STD infection that cannot be as effectively 'flushed' by urinating.

Because data on oral–genital grooming and urination among primate species are not readily available from the literature, Nunn (2003) compiled data using an email survey of primatologists. These data represent the best available information at this time and are unlikely to be systematically biased. Replies were received from 77 primatologists, including individuals that work with wild, zoo and laboratory populations of primates. Many respondents provided information on more than one species, and for 21 primate species, two to four responses were available, allowing assessment of data quality. Because inconsistencies were found for some questions, analyses were conducted first with variable responses coded as behaviour absent for a species and, second, as the behaviour present.

We predicted that post-copulatory oral–genital grooming would be more common among promiscuous species of primates. Surprisingly, this prediction was not supported. We estimated promiscuity in two ways – as relative testes mass and the duration of oestrus (Nunn *et al.*, 2000). In phylogenetic analyses of male and female oral–genital grooming, grooming was unrelated to either variable after incorporating variation among survey respondents (i.e. we found a nearly even mixture of positive and negative results, $P = 0.04$ to 0.78 in sensitivity tests; no results were significant after correcting for multiple comparisons; Nunn, 2003). It is unlikely that the data are too fragmentary or variable for testing the hypothesis, because further analysis revealed a clear phylogenetic signal in the frequency of post-copulatory grooming among species (Fig. 7.4). Most prosimians show very stereotyped genital grooming after mating, whereas this stereotyped behaviour is largely absent in anthropoids. The other major phylogenetic group showing oral–genital grooming is the callitrichids, where both sexes of all species in the database were reported to groom their genitals orally after mating, in at least one response to the survey. While several other species were reported to exhibit the behaviour, not all replies were consistent within species or genera (see Fig. 7.4). These results highlight two points. First, prosimians and callitrichids may be key clades for testing the hypothesis observationally or experimentally. Interestingly, both clades exhibit marked variation in mating behaviour and flexible mating systems (Goldizen, 1988; Kappeler, 2000). Second, these analyses suggest that body size may be a correlate of oral-grooming behaviour because callitrichids and

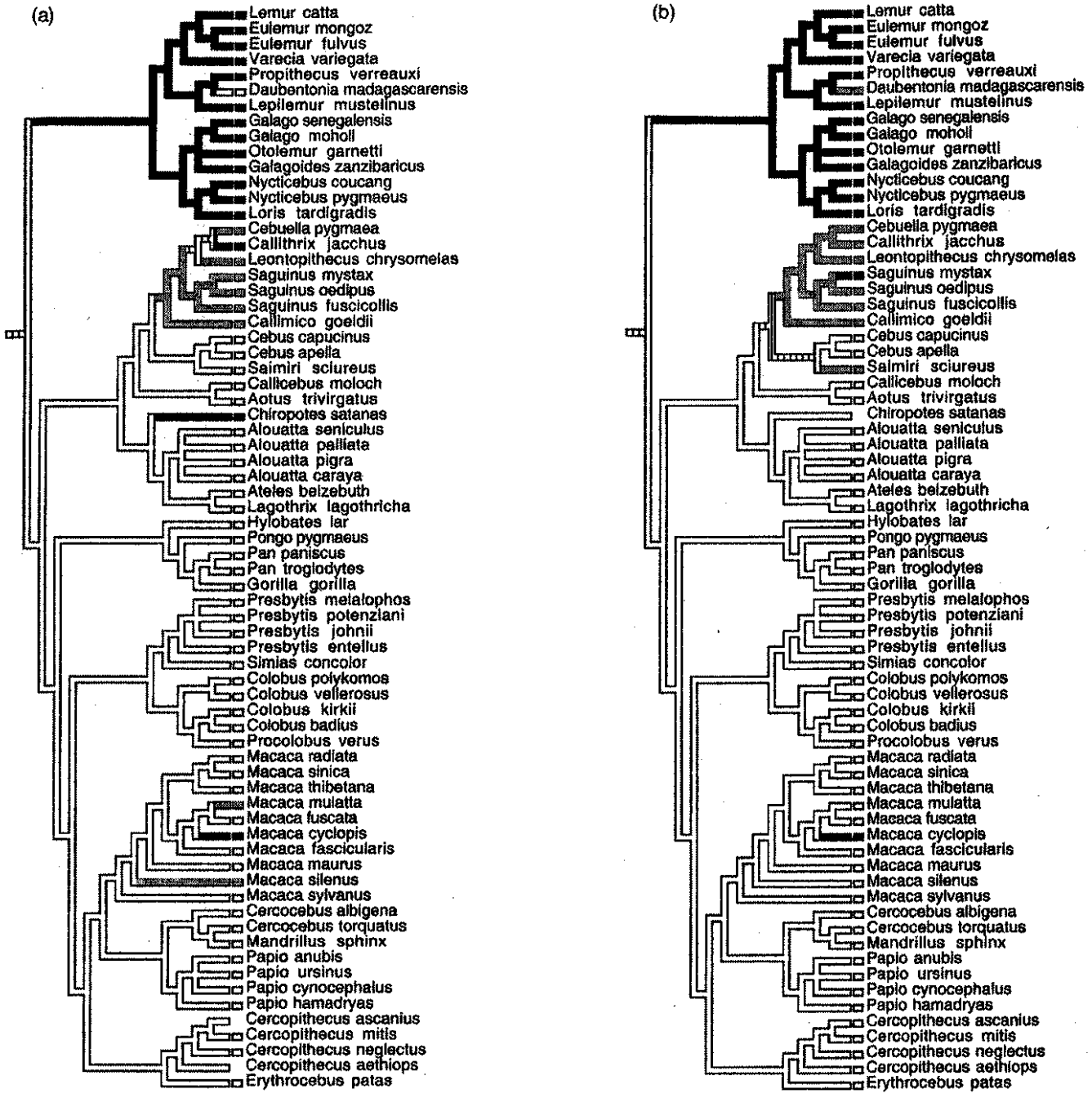

Fig. 7.4 Phylogenetic distribution of oral–genital self-grooming in male (a) and female (b) primates following copulation. Dark shading indicates presence of the trait, white shading indicates absence, and grey shading indicates conflicting responses or intermediate values of the trait (see Nunn, 2003). Species with no boxes indicate that no information was available. The phylogeny was taken from Purvis (1995).

prosimians are among the smallest-bodied primates. One possible explanation is that larger-bodied species experience physical limitations in reaching their genitalia for oral grooming.

Comparative tests using data on the occurrence of post-copulatory urination also failed to support predictions that this behaviour is associated with the risk of STD infection. It should be noted, however, that many respondents expressed uncertainty in their answers to these questions, reflecting that few observers systematically record urination in relation to mating behaviour. With this limitation in mind, survey responses indicated that very few species exhibit the behaviour, with only 2–6 per cent of species ($n = 53$) exhibiting post-copulatory urination by males, and only 5–13 per cent of species ($n = 55$) exhibiting post-copulatory

urination by females. Rather than being involved in STD prevention, many respondents noted that urination was part of scent-marking behaviour, as reported for some species (e.g. Robinson, 1979; Boinski, 1992). Moreover, post-copulatory urination was not significantly related to quantitative measures of promiscuity involving relative testes mass or the duration of oestrus (Nunn, 2003). A secondary prediction from models of disease risk is that post-copulatory urination should be more common in males than females (see above). In fact, the behaviour was slightly more common in females than males.

These results must be considered preliminary, but thus far it appears that primates fail to exhibit post-copulatory behaviours that would prevent STD transmission. Nunn (2003) also examined patterns of genital inspection prior to copulation, but found no correlations between inspection and STD risk factors. Despite these negative results, some variables, such as oral–genital grooming, showed strong phylogenetic signals (Fig. 7.4), suggesting that the data are not so 'noisy' that patterns are undetectable. Further research is needed to identify the causes of variation within particular primate clades and to understand the absence of behavioural defences to STDs in other species.

SUMMARY AND CONCLUSIONS

STDs clearly exist in free-living primate populations and are likely to be costly to infected hosts. Although STDs have been relatively under-studied in comparison to other parasites, many examples of primate STDs exist, and a growing number of studies have monitored prevalence in wild primate populations. From what we know of STDs in humans and domesticated animals, there is good reason to expect that STDs impact reproductive success in wild primates. This is borne out in comparative studies of immune-system parameters in relation to host promiscuity and infection risk (Nunn *et al.*, 2000, in review; Nunn, 2002). Moreover, our analysis of prevalence indicates that STDs are distributed in primate populations as expected from theoretical models of parasite transmission and sexual selection (Thrall *et al.*, 2000).

Based on these lines of evidence, it is surprising that animals have not been reported to show behavioural defences to STDs, including mate choice to avoid infected partners and post-copulatory grooming and urination. As illustrated by Knell's (1999) model and primate options to reduce STD risk, pre-copulatory behavioural defences are expected to show trade-offs with other reproductive activities. This is made clear by the case of monogamy in primates, which is expected to be an effective defence against STDs in humans (Immerman, 1986) and other animals (Loehle, 1995; Thrall *et al.*, 2000). In many of these 'monogamous' species, however, extra-pair matings have been observed (*Callicebus moloch*: Mason, 1966; *Hylobates syndactylus*: Palombit, 1994; *Hylobates lar*: Reichard, 1995). One interpretation of these observations is that immediate reproductive benefits from multiple mating outweigh the risks of STD infection, even in presumably monogamous species.

More generally, male and female behaviour indicates that STD risk is of secondary importance relative to other selective pressures operating on mating success. Females mate polyandrously to reduce infanticide risk (van Schaik & Janson, 2000) and, for similar reasons, they prefer novel males, though risking infection with STDs acquired from other social groups. Males prefer females of intermediate age that have already produced offspring, as these females have high reproductive value (see Anderson, 1986). Both sets of decisions by males and females are expected to increase exposure to STDs by increasing the number of partners and mating events.

These results represent an initial exploration of the consequences of sexual selection for the spread of STDs, and the distribution of behavioural defences across primate species. Many important questions remain to be tested both within and among natural populations of primates, including:

(1) Is male mating success associated with the risk of acquiring STDs?
(2) How does STD infection influence the expression of sexually selected traits?
(3) How do age, sex and social status correlate with STD risk?
(4) What symptoms do STDs cause in wild primates, and to what extent do STDs impact host reproductive success?
(5) Are there medicinal plants that reduce the risk of acquiring sexually transmitted diseases, as suggested for non-sexually transmitted parasites (e.g. Huffman *et al.*, 1997)?

Finally, sexual transmission is only one transmission route for parasites, and it may not be equally effective in all mating systems (Thrall *et al.*, 1997, 2000). We therefore need to investigate the ecological and evolutionary effects of other parasite transmission modes, including trade-offs between sexual and non-sexual transmission (Thrall *et al.*, 1998). It seems likely that variation in host behaviour affects the selective advantages conferred to parasites with different transmission strategies, although very few studies have quantified

transmission in free-living populations. With increasing data on parasites and phylogenies, it will become increasingly possible to answer these broad evolutionary questions and link them to the behavioural ecology of hosts.

ACKNOWLEDGEMENTS

For helpful discussion or comments on the manuscript, we thank Sarah Hrdy, Peter Kappeler, Monique Borgerhoff Mulder, Mary Poss, Carel van Schaik and two anonymous reviewers.

C. Nunn thanks the respondents to the email survey, whose names can be found at http://faculty.virginia.edu/charlienunn. This research was supported through a grant from the National Center for Ecological Analysis and Synthesis (NCEAS) to S. Altizer and C. Nunn, and a National Science Foundation (NSF) Postdoctoral Research Fellowship in Biological Informatics to C. Nunn.

REFERENCES

Abbot, P. & Dill, L. M. 2001. Sexually transmitted parasites and sexual selection in the milkweed leaf beetle, *Labidomera clivicollis*. *Oikos*, **92**, 91–100.

Able, D. J. 1996. The contagion indicator hypothesis for parasite-mediated sexual selection. *Proceedings of the National Academy of Sciences, USA*, **93**, 2229–33.

Alexander, N. J. 1990. Sexual transmission of human immunodeficiency virus: virus entry into the male and female genital tract. *Fertility and Sterility*, **54**, 1–18.

Anderson, C. M. 1986. Female age: male preference and reproductive success in primates. *International Journal of Primatology*, **7**, 305–26.

Anderson, R. M. & May, R. M. 1991. *Infectious Diseases of Humans: Dynamics and Control*. Oxford: Oxford University Press.

Baron, S., Singh, I., Chopra, A., Coppenhaver, D. & Pan, J. Z. 2000. Innate antiviral defenses in body fluids and tissues. *Antiviral Research*, **48**, 71–89.

Barratt, C. L. R., Bolton, A. E. & Cooke, I. D. 1990. Functional significance of white blood cells in the male and female reproductive tract. *Human Reproduction*, **5**, 639–48.

Bibollet-Ruche, F., Galat-Luong, A., Cuny, G. *et al.* 1996. Simian immunodeficiency virus infection in a patas monkey (*Erythrocebus patas*): evidence for cross-species transmission from African green monkeys (*Cercopithecus aethiops sabaeus*) in the wild. *Journal of General Virology*, **77**, 773–81.

Blower, S. M. & McLean, A. R. 1991. Mixing ecology and epidemiology. *Proceedings of the Royal Society of London, Series B*, **245**, 187–92.

Boinski, S. 1992. Olfactory communication among Costa Rican squirrel monkeys: a field study. *Folia Primatologica*, **59**, 127–36.

Botha, M. C., Jones, M., Deklerk, W. A. & Yamamoto, N. 1985. Spread and distribution of human T-cell leukemia-virus type-I-reactive antibody among baboons and monkeys in the northern and eastern Transvaal. *South African Medical Journal*, **67**, 665–8.

Bull, J. J. 1994. Virulence. *Evolution*, **48**, 1423–37.

Courgnaud, V., Pourrut, X., Bibollet-Ruche, F. *et al.* 2001. Characterization of a novel simian immunodeficiency virus from guereza colobus monkeys (*Colobus guereza*) in Cameroon: a new lineage in the nonhuman primate lentivirus family. *Journal of Virology*, **75**, 857–66.

Cowlishaw, G. & Dunbar, R. I. M. 1991. Dominance rank and mating success in male primates. *Animal Behavior*, **41**, 1045–56.

Dixson, A. F. 1998. *Primate Sexuality: Comparative Studies of the Prosimians, Monkeys, Apes, and Humans*. Oxford: Oxford University Press.

Donovan, B. 2000a. The repertoire of human efforts to avoid sexually transmissible diseases: past and present. Part 1: strategies used before or instead of sex. *Sexually Transmitted Infections*, **76**, 7–12.

2000b. The repertoire of human efforts to avoid sexually transmissible diseases: past and present. Part 2: strategies used during or after sex. *Sexually Transmitted Infections*, **76**, 88–93.

Dracopoli, N. C., Turner, T. R., Else, J. G. *et al.* 1986. STLV-I antibodies in feral populations of east-african vervet monkeys (*Cercopithecus aethiops*). *International Journal of Cancer*, **38**, 523–9.

Eberhard, W. G. 1985. *Sexual Selection and Animal Genitalia*. Cambridge, MA: Harvard University Press.

Ebert, D. 1994. Virulence and local adaptation of a horizontally transmitted parasite. *Science*, **265**, 1084–6.

Ewald, P. W. 1994. *Evolution of Infectious Disease*. Oxford: Oxford University Press.

Galat-Luong, A., Bibollet-Ruche, F., Pourrut, X. *et al.* 1994a. Social organization and SIV seroepidemiology of a patas monkey population in Senegal. *Folia Primatologica*, **63**, 226–8.

Galat-Luong, A., Galat, G., Bibollet-Ruche, F. *et al.* 1994b. Social structure and SIVagm prevalence in two groups of green monkeys, *Cercopithecus aethiops sabaeus*, in Senegal.

In *Current Primatology*. Vol. 3: *Behavioral Neuroscience, Physiology and Reproduction*, ed. J. R. Anderson, J. J. Roeder, B. Thierry & N. Herrenschmidt. Strasbourg: Université Louis Pasteur, pp. 259–62.

Garnett, G. P. & Anderson, R. M. 1993. No reason for complacency about the potential demographic impact of AIDS in Africa. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, **87**, 19–22.

Georges-Courbot, M. C., Moisson, P., Leroy, E. *et al.* 1996. Occurrence and frequency of transmission of naturally occurring simian retroviral infections (SIV, STLV, and SRV) at the CIRMF Primate Center, Gabon. *Journal of Medical Primatology*, **25**, 313–26.

Getz, W. M. & Pickering, J. 1983. Epidemic models: thresholds and population regulation. *American Naturalist*, **121**, 892–8.

Goldizen, A. W. 1988. Tamarin and marmoset mating systems: unusual flexibility. *Trends in Ecology and Evolution*, **3**, 36–40.

Graves, B. M. & Duvall, D. 1995. Effects of sexually-transmitted diseases on heritable variation in sexually selected systems. *Animal Behaviour*, **50**, 1129–31.

Hahn, B. H., Shaw, G. M., de Cock, K. M. & Sharp, P. M. 2000. AIDS as a zoonosis: scientific and public health implications. *Science*, **287**, 607–14.

Hamilton, W. D. & Zuk, M. 1982. Heritable true fitness and bright birds: a role for parasites? *Science*, **218**, 384–7.

Hart, B. J., Korinek, E. & Brennan, P. 1987. Postcopulatory genital grooming in male rats: prevention of sexually transmitted infections. *Physiology and Behavior*, **41**, 321–5.

Hausfater, G. & Hrdy, S. B. 1984. *Infanticide: Comparative and Evolutionary Perspectives*. New York, NY: Aldine de Gruyter.

Hayami, M., Komuro, A., Nozawa, K. *et al.* 1984. Prevalence of antibody to adult T-cell leukemia virus-associated antigens (ATLA) in Japanese monkeys and other non-human primates. *International Journal of Cancer*, **33**, 179–83.

Herre, E. A. 1995. Factors affecting the evolution of virulence: nematode parasites of fig wasps as a case study. *Parasitology*, **111**, S179–91.

Hoeprich, P. D., Jordan, M. C. & Ronald, A. R. 1994. *Infectious Diseases: A Treatise of Infectious Processes*. Philadelphia, PA: J. B. Lippincott.

Holmes, K. K., Sparling, P. F., Mardh, P.-A. *et al.* 1999. *Sexually Transmitted Diseases*. New York, NY: McGraw Hill.

Hooper, R. R., Reynolds, G. H., Jones, O. G. *et al.* 1978. Cohort study of venereal disease. I: The risk of gonorrhea transmission from infected women to men. *American Journal of Epidemiology*, **108**, 136–44.

Hrdy, S. B. 1979. Infanticide among animals: a review, classification, and examination of the implications for the reproductive strategies of females. *Ethology and Sociobiology*, **1**, 13–40.

Huffman, M. A., Gotoh, S., Turner, L. A., Hamai, M. & Yoshida, K. 1997. Seasonal trends in intestinal nematode infection and medicinal plant use among chimpanzees in the Mahale Mountains, Tanzania. *Primates*, **38**, 111–25.

Immerman, R. S. 1986. Sexually transmitted disease and human evolution: survival of the ugliest? *Human Ethology Newsletter*, **4**, 6–7.

Ishida, T., Yamamoto, K., Kaneko, R., Tokita, E. & Hinuma, Y. 1983. Seroepidemiological study of antibodies to adult T-cell leukemia virus-associated antigen (ATLA) in free-ranging Japanese monkeys (*Macaca fuscata*). *Microbiology and Immunology*, **27**, 297–301.

Ishikawa, K., Fukasawa, M., Tsujimoto, H. *et al.* 1987. Serological survey and virus isolation of simian T-cell leukemia/lymphotropic virus Type-I (STLV-I) in nonhuman primates in their native countries. *International Journal of Cancer*, **40**, 233–9.

Jolly, A. 1966. *Lemur Behavior*. Chicago, IL: University of Chicago Press.

Jolly, C. J., Phillips-Conroy, J. E., Turner, T. R., Broussard, S. & Allan, J. S. 1996. SIVagm incidence over two decades in a natural population of Ethiopian grivet monkeys (*Cercopithecus aethiops aethiops*). *Journal of Medical Primatology*, **25**, 78–83.

Kappeler, P. M. 2000. Causes and consequences of unusual sex ratios among lemurs. In *Primate Males: Causes and Consequences of Variation in Group Composition*, ed. P. M. Kappeler. Cambridge: Cambridge University Press, pp. 55–63.

Knell, R. J. 1999. Sexually transmitted disease and parasite-mediated sexual selection. *Evolution*, **53**, 957–61.

Lockhart, A. B., Thrall, P. H. & Antonovics, J. 1996. Sexually transmitted diseases in animals: ecological and evolutionary implications. *Biological Reviews*, **71**, 415–71.

Loehle, C. 1995. Social barriers to pathogen transmission in wild animal populations. *Ecology*, **76**, 326–35.

Mason, W. A. 1966. Social organization of the South American monkey, *Callicebus moloch*: a preliminary report. *Tulane Studies in Zoology*, **13**, 23–8.

Møller, A. P. 1993. A fungus infecting domestic flies manipulates sexual behavior of its host. *Behavioral Ecology and Sociobiology*, **33**, 403–7.

Nerrienet, E., Amouretti, X., Mullertrutwin, M. C. *et al.* 1998. Phylogenetic analysis of SIV and STLV Type I in mandrills (*Mandrillus sphinx*): indications that intracolony transmissions are predominantly the result of male-to-male aggressive contacts. *AIDS Research and Human Retroviruses*, **14**, 785–96.

Norley, S., Beer B., Holzammer S., Zur Megede, J. & Kurth, R. 1999. Why are natural hosts of SIV resistant to AIDS? *Immunology Letters*, **66**, 47–52.

Nunn, C. L. 2002. A comparative study of leukocyte counts and disease risk in primates. *Evolution*, **56**, 177–90.

2003. Behavioural defences against sexually transmitted diseases in primates. *Animal Behaviour*, in press.

Nunn, C. L., Gittleman, J. L. & Antonovics, J. 2000. Promiscuity and the primate immune system. *Science*, **290**, 1168–70.

2003. A comparative study of white blood cell counts and disease risk in carnivores. *Proceedings of the Royal Society of London, Series B. Biological Sciences*, **270**, 347–56.

Padian, N. S., Shiboski, S. C., Glass, S. O. & Vittinghoff, E. 1997. Heterosexual transmission of human immunodeficiency virus (HIV) in Northern California: results from a ten-year study. *American Journal of Epidemiology*, **146**, 350–7.

Palombit, R. A. 1994. Extra-pair copulations in a monogamous ape. *Animal Behaviour*, **47**, 721–3.

Pandya, I. J. & Cohen, J. 1985. The leukocytic reaction of the human uterine cervix to spermatozoa. *Fertility and Sterility*, **43**, 417–21.

Phillips, D. M. & Mahler, S. 1977. Leukocyte emigration and migration in the vagina following mating in the rabbit. *Anatomical Record*, **189**, 45–60.

Phillips-Conroy, J. E., Jolly, C. J., Petros, B., Allan, J. S. & Desrosiers, R. C. 1994. Sexual transmission of SIV(agm) in wild grivet monkeys. *Journal of Medical Primatology*, **23**, 1–7.

Purvis, A. 1995. A composite estimate of primate phylogeny. *Philosophical Transactions of the Royal Society of London, Series B*, **348**, 405–21.

Purvis, A. & Rambaut, A. 1995. Comparative analysis by independent contrasts (CAIC): an Apple Macintosh application for analysing comparative data. *Computer Applications in the Biosciences*, **11**, 247–51.

Radolf, J. D., Sanchez, P. J., Schulz, K. F. & Murphy, F. K. 1999. Congenital syphilis. In *Sexually Transmitted Diseases*, ed. K. K. Holmes, P. F. Sparling, P.-A. Mardh, S. M. Lemon, W. E. Stamm, P. Piot & J. N. Wasserheit. New York, NY: McGraw-Hill, pp. 1165–89.

Read, A. F. 1994. The evolution of virulence. *Trends in Microbiology*, **2**, 73–6.

Reichard, U. 1995. Extra-pair copulation in a monogamous gibbon (*Hylobates lar*). *Ethology*, **100**, 99–112.

Richards, A. L., Giri, A., Iskandriati, D. *et al.* 1998. Simian T-lymphotrophic virus type-I infection among wild-caught Indonesian pig-tailed macaques (*Macaca nemestrina*). *Journal of Acquired Immune Deficiency Syndromes and Human Retrovirology*, **19**, 542–5.

Roberts, L. 1979. Bovine venereal campylobacteriosis (vibriosis) in north east Scotland. *Veterinary Record*, **105**, 295–6.

Robinson, J. G. 1979. Correlates of urine washing in the wedge-capped capuchin *Cebus nigrivittatus*. In *Vertebrate Ecology in the Northern Neotropics*, ed. J. F. Eisenberg. Washington, DC: Smithsonian Institution Press, pp. 137–43.

Santiago, M. L., Rodenburg, C. M., Kamenya, S. *et al.* 2002. SIVcpz in wild chimpanzees. *Science*, **295**, 465.

Schwartländer, B., Garnett, G., Walker, N. & Anderson, R. 2000. AIDS in a new millennium. *Science*, **289**, 64–7.

Sheldon, B. C. 1993. Sexually-transmitted disease in birds: occurrence and evolutionary significance. *Philosophical Transactions of the Royal Society of London, Series B*, **339**, 491–7.

Smith, G. & Dobson, A. P. 1992. Sexually transmitted diseases in animals. *Parasitology Today*, **8**, 159–66.

Sorci, G., Møller, A. P & Boulinier, T. 1997. Genetics of host–parasite interactions. *Trends in Ecology and Evolution*, **12**, 196–200.

Swanstrom, R. & Wehbie, R. 1999. The biology of HIV, SIV and other lentiviruses. In *Sexually Transmitted Diseases*, ed. K. K. Holmes, P. F. Sparling, P.-A. Mardh, S. M. Lemon, W. E. Stamm, P. Piot & J. N. Wasserheit. New York, NY: McGraw-Hill, pp. 215–29.

Thrall, P. H., Antonovics, J. & Bever, J. D. 1997. Sexual transmission of disease and host mating systems: within-season reproductive success. *American Naturalist*, **149**, 485–506.

Thrall, P. H., Antonovics, J. & Dobson, A. P. 2000. Sexually transmitted diseases in polygynous mating systems: prevalence and impact on reproductive success. *Proceeding of the Royal Society of London, Series B*, **267**, 1555–63.

Thrall, P. H., Antonovics, J. & Hall, D. W. 1993. Host and pathogen coexistence in vector-borne and venereal diseases characterized by frequency-dependent disease transmission. *American Naturalist*, **142**, 543–52.

Thrall, P. H., Antonovics, J. & Wilson, W. G. 1998. Allocation to sexual vs. non-sexual disease transmission. *American Naturalist*, **151**, 29–45.

van Schaik, C. P. & Janson, C. H. 2000. *Infanticide by Males and Its Implications*. Cambridge: Cambridge University Press.

Webberley, K. M., Hurst, G. D. D., Buszko, J. & Majerus, M. E. N. 2002. Lack of parasite-mediated sexual selection in a ladybird/sexually transmitted disease system. *Animal Behaviour*, **63**, 131–41.