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8 *Disease avoidance and the evolution of primate social connectivity: Ebola, bats, gorillas, and chimpanzees*

PETER D. WALSH, MAGDALENA BERMEJO, AND JOSÉ DOMINGO RODRÍGUEZ-TEIJEIRO

Introduction

During the 1970s and '80s ecology experienced a vigorous, one might even say raucous, debate over whether biological communities were structured by competition or predation (Connell, 1975; Menge & Sutherland, 1976). The answer that emerged may have been predictable . . . "both, of course" . . . but the

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debate energized the field. Spurred on by an almost apostolic vigor, ecologists cranked out a raft of exciting new theories and boatloads of great empirical work to test these theories (reviewed in Sih *et al*., 1985; Gurevitch *et al*. 2000).

At about the same time, there were signs that a similar debate might take hold in primatology. The melding of ethology and ecology brought a wave of new ideas on how behavior might interact with ecological factors and, in particular, on the way in which competition for resources might influence social organization. Factors such as territoriality, social group size, and rates of association within groups were all viewed in terms of the way resource distribution influenced competition (e.g. Eisenberg *et al*., 1972; Mitani & Rodman, 1979; van Schaik & van Hooff, 1983; Terborgh & Janson, 1986). At about the same time, William J. Freeland wrote a seminal paper discussing the influence of disease on the evolution of primate social structure (Freeland, 1976, see discussion in Chapman *et al*., Chapter 21, this volume). Freeland's central insight was that the risk of disease transmission was proportional to the number of individuals one interacted with and, therefore, that natural selection should promote behavioral mechanisms that limited social contact. He looked at the same phenomena as the competitionistas . . . territoriality, group size, and rates of association within groups . . . and came to very different conclusions about their evolutionary origin.

Unfortunately, the debate never materialized. Since the 1980s primatology has danced to the beat of one hand clapping, developing an almost religious devotion to the idea that resource distribution and abundance is the (only) important evolutionary driver of social structure. Territoriality, social group size, rates of association within groups, and other attributes of social organization are now almost universally viewed as adaptive consequences of competition for food and/or mates. A few devoted acolytes of the disease cult have labored diligently to keep Freeland's candle burning (Nunn & ~~Alizer~~, 2006). But even they have tended to treat disease transmission as an emergent consequence of primate social structure rather than a selection pressure driving its evolution (e.g., Nunn & Dokey 2006).

This paper is an attempt to stir the pot: to rekindle wider interest in Freeland's ideas by presenting empirical data that illustrate just how strong a selective pressure disease can be and how much primate social structure can influence disease impact. To that end we present data on western gorilla (*Gorilla gorilla*) and chimpanzee (*Pan troglodytes*) mortality from Ebola virus at our study site in and around Lossi Sanctuary in northwest Republic of Congo. Over the last 15 years Ebola has caused massive gorilla and chimpanzee population declines in Congo and neighboring Gabon (Huijbregts *et al*., 2003; Walsh *et al*., 2003; Caillaud *et al*., 2006; Bermejo *et al*., 2006), killing about one third of the world's protected area gorilla population (IUCN, 2007). Ebola makes a nice

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case study in that gorillas and chimpanzees differ both in their social structure and in the extent of their contact with the putative reservoir hosts for Ebola, bats (Leroy *et al*., 2005). Conveniently, the bat and social contact patterns make opposing predictions about which species, gorillas or chimpanzees, should be more susceptible to Ebola infection.

Here we first outline the differences between gorillas and chimpanzees in social structure and bat contact rates and make predictions about how these differences should translate into different rates of Ebola infection. We then present survey data on patterns of Ebola survivorship collected in and around Lossi after Ebola outbreaks in 2002–2003 and evaluate different scenarios for the Ebola transmission dynamics underlying both the survey data and auxiliary data on bats. Finally, we discuss the implications of our results for hypotheses on the evolution of social organization in primates, with a particular focus on how body size affects disease exposure risk.

Social contact structures and spillover rates

Western gorilla and chimpanzee social organization differ in three ways that have direct implications for disease transmission. First, neighboring western gorilla social groups show substantial ranging overlap. They have direct social encounters about every 2 weeks (Bermejo, 2004; Doran-Sheehy *et al*., 2004) and more often visit fruiting trees and clearings visited by other groups on the same day (Walsh *et al*., 2007). Interactions between groups are often tolerant if not affiliative. Juveniles from different groups occasionally play together and neighboring groups have even been observed to nest together (Bermejo, 2004). In stark contrast, chimpanzee communities vigorously defend territories to the point of killing intruders. Second, western gorillas live in much smaller social groups than chimpanzees. Fourteen gorilla groups at Lossi averaged 14 individuals (Bermejo *et al*., 2006; see also Robbins *et al*., 2004), while chimpanzee communities typically contain about 50–75 individuals (Wrangham, 2000). Third, gorillas live in cohesive groups which forage together each day and sleep together every night. Chimpanzees spend much of the year in smaller parties, with sub-group composition turning over on a daily basis (Chapman *et al*., 1995).

Exposure to disease spillover from bats also differs between the two ape species as a function of three major differences in their diets. The first involves the rate at which they consume the fruit of trees from the genus *Ficus*. Rates of *Ficus* consumption are probably good indicators of spillover rates because the three bat species implicated as reservoirs for Ebola are obligate frugivores which tend to specialize on small seeded fruit, particularly *Ficus*. For example, the

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Table 8.1. *Minimum convex polygon estimates of Apollo group home range size*

Year	MCP area	Obs days
1996	6.05	64
1997	5.99	68
1998	5.31	128
2000	5.84	37
2002	6.17	86
Mean	5.87	

MCP = minimum convex polygon.
Observation days = observation days per year.

only systematic study of *Hypsygnathus monstrosus*, the largest of the putative Ebola reservoirs, was conducted about 200 km west of our study site at Lossi and showed that *Ficus* seeds were present in 85% of *H. monstrosus* dung (Bradbury 1977).

Ficus fruit are a prominent component of chimpanzee diet, with *Ficus* seeds appearing in 50% or more of chimpanzee dung piles (Morgan & Sanz, 2007). In fact, feeding at a *Ficus* tree was one of the risk factors observed during an Ebola outbreak amongst habituated chimpanzees in Cote d'Ivoire (Formenty *et al*., 1999). In contrast, during 440 days of all-day follows at Lossi, gorillas fed on *Ficus* trees on only 38 days or one out every 11.6 days. If we interpret the 50% of chimpanzee dung piles containing *Ficus* seeds to mean that chimpanzees eat *Ficus* every other day, these results imply a nearly sixfold greater rate of chimpanzee feeding overlap with bats at *Ficus* trees than experienced by Lossi gorillas. This difference may stem from a specific preference of chimps for *Ficus*, or it may, in part, reflect the fact that chimpanzee community territories typically cover a larger area than gorilla home ranges and, therefore, contain more *Ficus* trees. For instance, chimpanzee communities typically cover about 20 km^2 (Herbinger *et al*., 2001), while annual minimum convex polygon estimates of home range size for the Apollo gorilla group averaged only 5.87 km^2 (Table 8.1). The ratio of home range sizes ($\frac{20\,km^2}{5.87\,km^2} = 3.45$) is only about half the ratio of *Ficus* feeding rates ($\frac{1\,event/2\,days}{1\,event/11.6\,days} = 5.8$), suggesting that home range size may explain some, but not all, of the difference in *Ficus* feeding rates.

The second major dietary difference is that gorillas consume substantially more plant vegetative material than chimpanzees. This does not result in a substantive decrease in the diversity of fruit species eaten by gorillas, as sympatric

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populations of chimpanzees and gorillas show a very high overlap in which species they consume (Tutin & Fernandez, 1993). However, it does result in a reduction in the number of different trees visited per day. All-day follows showed that the Apollo gorilla group fed on fruit from an average of 1.92 trees/day (n = 199 days, SE = 0.06) and had a mean day path length of only 1.31 km, an estimate comparable to other western gorilla studies (Cipolletta, 2004). This compares to a typical day path length of more than 2 km for chimpanzees (Pontzer & Wrangham, 2006). Thus, even if spillover from bats occurs at fruit trees other than *Ficus*, chimpanzees should have a substantially higher rate of exposure than gorillas.

The third dietary difference between gorillas and chimpanzees is in their propensity to hunt other vertebrates. Chimpanzees do. Gorillas do not. The tendency for chimpanzees to hunt other primates is pertinent to Ebola transmission because, in addition to being reservoir hosts in their own right (Courgnaud *et al*., 2003), other frugivorous primates may be intermediate hosts for bat viruses. In fact, consumption of monkey meat was a second risk factor during the Ebola outbreak amongst chimpanzees in the Cote d'Ivoire outbreak (Formenty *et al*., 1999).

These differences between gorillas and chimpanzees in social contact structure and spillover risk provide one very clear prediction about the expected impact of Ebola. If Ebola outbreaks are caused by "massive spillover" in which each ape is infected directly from the reservoir host (Leroy *et al*., 2004, 2005), then chimpanzees should suffer higher rates of mortality than gorillas as a consequence of their greater dietary overlap with bats. On the other hand, if spillover is rare and most apes are infected through subsequent ape-to-ape transmission, then gorillas should suffer higher mortality rates as a consequence of their higher rates of social contact.

Ape mortality at Lossi

One pattern that is immediately obvious in the nest survey data from Lossi is that gorillas were almost totally extirpated from 2700 km^2 of survey zone lying west of 14.55 degrees. Comparisons of nest encounter rates east and west of 14.55 suggest gorilla mortality rates of about 96% (Bermejo *et al*., 2006). This estimate is consistent with the mortality rate estimated from 14 known social groups (243 individuals) in our primary study area in the Lossi Sanctuary (Bermejo *et al*., 2006) and the rate estimated for more than 350 known gorillas at Lokoue clearing in nearby Odzala National Park (Caillaud *et al*., 2006). Although chimpanzees also suffered very high mortality rates over much of

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the western part of the survey zone, there were regions of chimpanzee survival along the northern and southern borders. Consequently, chimpanzee mortality rates averaged about 86% in the western zone.

Given the greater exposure of chimpanzees to bats, higher rates of chimpanzee survival are not consistent with a "massive spillover" scenario in which most apes are infected directly from the reservoir host. Rather, higher rates of survival in chimpanzees seem more likely a consequence of lower rates of social contact amongst chimpanzees than gorillas. High rates of transmission within gorilla social groups is suggested by observations both at Lokoue (Caillaud *et al*., 2006), where adult males (silverbacks) suffered higher mortality rates than solitary males and at Lossi, where deaths within groups were not confined to a single event, but spread out over 6 weeks or more (Bermejo *et al*. 2006). At both sites, all affected groups lost a large proportion of group members. Patterns of chimpanzee mortality at Taï forest, Cote d'Ivoire were also suggestive of chains of transmission involving multiple individuals, but mortality rates were lower than for gorillas: only 28% (12 of 43) of habituated chimpanzees died (Formenty *et al*., 1999).

The difference between ape species in population impact is not well explained in terms of differences in the virulence of Ebola infection to individual animals, as over large swathes of the western survey zone chimpanzees suffered population declines just as extreme as gorillas. Furthermore, the surviving habituated chimpanzees at Taï showed no symptoms of Ebola infection. Thus, the population impact differences between chimpanzees and gorillas appear to be due to differences in exposure rate rather than differences in infection virulence.

Based only on mean rates, it is difficult to discern whether transmission between social groups contributed to the observed mortality rate difference between Lossi gorillas and chimpanzees. For instance, one can imagine two contrasting scenarios that might produce higher survival in chimpanzees. In the first, spillover rates are high enough to ensure that all social groups are infected, but not high enough to kill all members within each social group. In this case, higher contact rates within gorilla groups would lead to higher gorilla mortality rates. An alternative scenario is that spillover rates are so low that only a small proportion of ape social groups are infected directly from the reservoir. Ebola then spreads laterally amongst apes groups, with gorillas infected at a higher rate because of their greater ranging overlap. Cross-species transmission from gorillas to chimpanzees is even plausible in that chimpanzees have very high rates of ranging and feeding overlap with sympatric gorillas. For instance, a study at Nouabale-Ndoki National Park in Northeast Congo found that gorillas and chimpanzees fed simultaneously in the same tree at least once every 15 days (Walsh *et al*., 2007).

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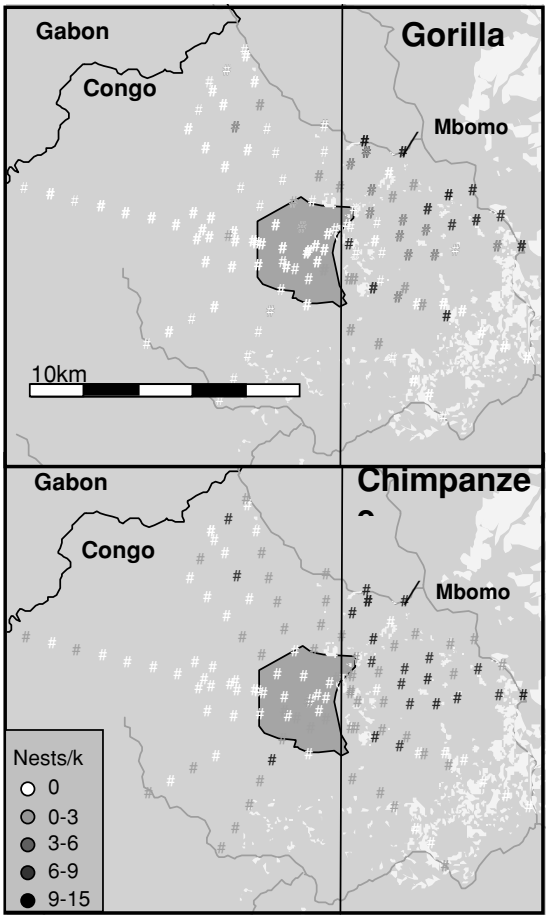

Figure 8.1. Gorilla and chimpanzee nest encounter rates in Lossi survey zone. Each hash mark represents ape nest encounter rate for 5 km segment of reconnaissance survey. Lossi Sanctuary at center and roads (light gray lines) on periphery of survey zone. Region is fully forested except for savanna patches (light areas). Vertical line at 14.55 degrees east longitude.

Although mean mortality rates do not provide information for discriminating between these hypotheses, the spatial pattern of mortality does. In particular, post-Ebola densities of both chimpanzees and gorillas at Lossi showed a strong negative correlation with distance from roads (Figure 8.1, 8.2A). This pattern was likely a consequence of a gradient in hunting intensity, which is evident in an increase in the density of elephants and duiker with increasing distance

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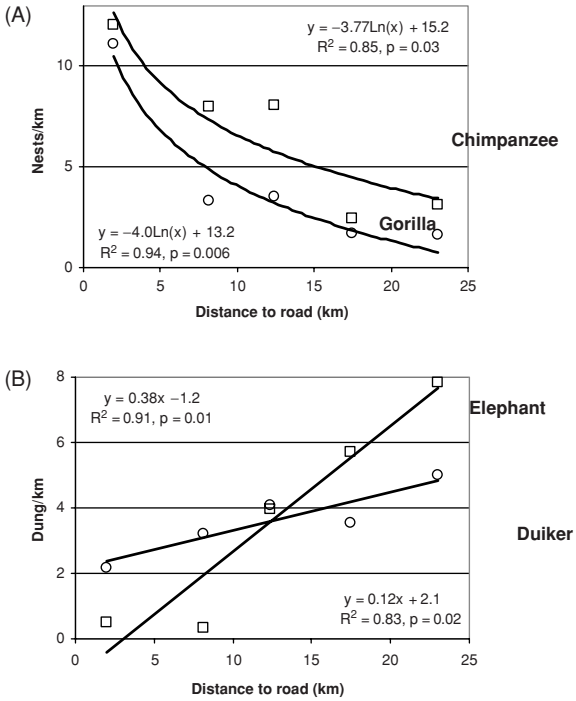

Figure 8.2. Ebola and hunting induced gradients in large mammal density. (A) Decreasing gorilla and chimpanzee nest encounter rates decrease with increasing distance to road suggest density dependent Ebola transmission. (B) Increasing elephant and duiker dung encounter rates increase illustrate impact of hunting. Data are for 137 5-km survey segments pooled into 5-km distance-to-road classes. Linear regression analyses without pooling into distance classes give similar results.

from roads (Figure 8.2B). More apes likely survived near roads because the lower ape densities reduced rates of Ebola transmission.

In principle, the observed gradient in ape mortality might be explained in terms of a gradient in bat density causing a gradient in spillover rates. In practice, this hypothesis is not tenable because larger game have yet to be depleted in the Lossi region (Walsh *et al*., in review). Although bats are taken opportunistically, hunting rates are not high enough to deplete bat densities over the spatial scale on which the ape mortality gradient was observed, particularly given the resilience to offtake afforded by high bat reproductive rates (Langevin & Barclay, 1990). Consequently, there is no gradient in bat density on a comparable scale as the gradients in gorilla and chimpanzee density (Walsh *et al*., in review).

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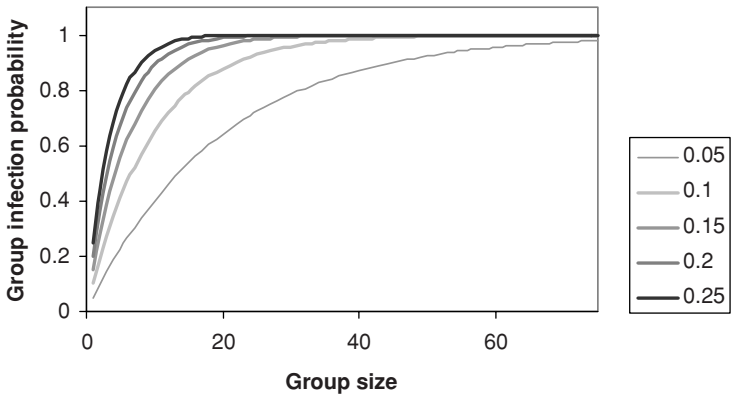

Figure 8.3. Effect of group size on probability that at least one individual in a group is infected by reservoir spillover. Model assumes each individual in a group suffers independent risk of spillover from reservoir host.

Another alternative form of the spillover hypothesis is that hunting induced a pre-Ebola gradient not just in ape densities, but also in ape group sizes. Because the probability that at least one group member is infected by reservoir spillover increases with group size (assuming independent, random infection of individual apes), the proportion of groups in which at least one individual is infected by reservoir spillover increases along the gradient in group size. There are two problems with this hypothesis. The first is that the effect of group size on the probability that at least one individual in a group is infected by reservoir spillover should be highly non-linear (Figure 8.3). This non-linearity makes it hard to maintain between-group heterogeneity in infection probability at both the group sizes typical of gorillas and the community sizes typical of chimpanzees. For example, an individual spillover risk that is high enough to ensure that at least one individual is infected in most larger, but not smaller, gorilla groups will result in infection of virtually all chimpanzee communities, regardless of their sizes. However, lowering the spillover rate to allow heterogeneity amongst chimpanzee communities in the probability that at least one individual is infected will drop the infection probability for large gorilla groups far below the level observed in the high impact zone at Lossi. Keeping spillover probability high but assuming that individuals are infected in clusters (rather than independently) stretches heterogeneity in chimpanzee community infection probability over a wider range of community sizes. However, increasing spillover cluster size makes it more likely that a substantial proportion of larger gorilla groups (i.e. groups far from the road) would escape infection entirely: a result not observed at Lossi. Thus, this version of the spillover

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hypothesis cannot explain the observed gradients in both gorilla and chimpanzee density.

The second problem with this form of the spillover hypothesis is that our survey data showed no correlation between distance from roads and the size of gorilla social groups. Combined with the observation of strong correlation between gorilla nest density and distance from roads, this lack of a correlation between group size and distance from roads implies that the mechanism underlying the mortality gradient was not a group size dependent gradient in the probability that each group was infected, but rather a gradient in the rate of transmission amongst groups, presumably caused by a gradient in group density.

A further problem with the massive spillover hypothesis is that genetic testing of bats captured at Mbomo, on the edge of our study area, revealed that bats already showed a 22.6% prevalence of Ebola infection in March 2003 (Leroy *et al*., 2005). This is a problem for the spillover hypothesis because ape densities in the zone immediately surrounding Mbomo showed no sign of Ebola impact despite the high prevalence of infection in sympatric bats. This might be explained away in terms of the lack of permissive spillover conditions (e.g. weather or fruit availability) in March 2003 except for the fact that gorillas were dying 15 km to the southwest at Lossi, both 1 month earlier and 6 months later (Bermejo *et al*., 2006). There were also ape deaths (and human spillover) 3 months later near Mbandza, 15 km to the north (Leroy *et al*., 2004). It seems more than slightly implausible that in this extremely flat and floristically homogenous region, weather or fruiting phenology would be unsuitable for spillover in an area covering hundreds of square kilometers, but suitable for massive spillover events in areas covering thousands of square kilometers only 15 km to the north and south.

A more likely explanation for the observed spatial gap in ape mortality is that Ebola spillover from bats is not common enough to infect every single ape social group. Rather, spillover is a relatively rare event, with the great majority of ape deaths resulting from transmission within and between ape social groups. This conclusion is supported by observations on known gorilla groups during the 2003 outbreak at Lossi, where a series of groups showed a sequence of mortality clearly suggestive of transmission between groups, with lags in mortality onset between neighboring groups comparable to the infection cycle length for Ebola (Bermejo *et al*., 2006). The distance separating neighboring home range centers, about 2 km, was also much smaller than the typical night range distance of *H. monstrosus*: about 8 km (Bradbury, 1977). It seems unlikely that spillover events could follow such a tight spatial progression if the reservoir host was visiting transmission foci (e.g. fig trees) distributed over a much larger scale.

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Furthermore, pulses of spillover from bats have been proposed to result from the high viral titers that develop as a consequence of maternal immunosuppression around the time of parturition (Leroy *et al*., 2005). However, the two annual bat birthing seasons are in July/August and January/February (Bradbury, 1977), while gorilla deaths at Lossi were detected from September through January (Bermejo *et al*., 2006). Thus, although maternal immunosuppression might be a reasonable explanation for initial spillover into apes, it should not result in sustained spillover for several following months. In this context, it is worth mentioning that viral titers in the body fluids of symptomatic primates are always extremely high as a consequence of the great virulence of Ebola infection in primates (Geisbert *et al*., 2003). In fact, contact with carcasses found in the forest has been the primary mode of human outbreak initiation in Gabon and Congo, while touching of corpses at funerals is a major mechanism of human outbreak amplification (Roels *et al*., 1999). Thus, contact with carcasses covered in or surrounded by infective body fluids is one plausible mechanism of transmission within or between gorillas and chimpanzees. Both ape species are curious about conspecific carcasses and will closely inspect and even groom them (Walsh *et al*., 2007).

Evolutionary implications

Much of the proceeding discussion has involved the role of reservoir spillover in Ebola transmission. However, rather than focusing on the minutiae of feeding overlap between chimpanzees and bats it may ultimately be more instructive to cast the more general issue of disease transmission in primates as a body size scaling problem. As a consequence of body size effects on factors such as thermal efficiency, home range size within a given taxonomic group tends to scale roughly with the 3/4; power of body size (Peters, 1983). Thus, the number of fruit trees falling within a given primate's home range should also scale approximately with the 3/4; power of body size. For instance, the home range of a 45 kg chimpanzee should contain roughly seven times more fruit trees than the home range of an 3.5 kg cephus monkey (*Cercopithecus cephus*) ($45^{3/4}/3.5^{3/4} = 6.8$). Now, each fruit tree is a potential focal point for contact with infective body fluids, deposited by conspecifics but also by other primates and by other frugivorous reservoir hosts such as bats, rodents, or birds. Consequently, to the extent that infection risk is linearly proportional to the number of focal points visited, each chimpanzee should suffer an infection risk that is roughly seven times higher than each cephus monkey.

Of course infection risk will tend to vary with factors other than the number of focal points in a given primate's home range. But the point is that ranging

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over larger scales inherently increases infection risk. Thus, the strength of selection on behavioral traits that limit potential for disease transmission from conspecifics should be stronger for large-bodied chimpanzees than for smaller monkeys. In principle, larger body size should make such selection even stronger for gorillas. However, in practice, the greater rate at which gorillas exploit more uniformly distributed, non-fruit resources (another body size effect) appears to have resulted in smaller day range and home range sizes for gorillas. Thus, the selection pressure for behavioral mechanisms to avoid disease transmission from conspecifics may have been weaker than would be predicted by their body size.

Although our observations at Lossi illustrate how massive a selection pressure disease can be and how social structure can influence disease transmission dynamics, they are clearly very far from "proving" that the territoriality and fission-fusion structure of chimpanzees evolved specifically to minimize disease transmission. They do, however, merit a more critical attitude towards the knee jerk assumption that primate social structure is driven entirely by feeding and mate competition. The challenge ahead will be to devise empirical tests that discriminate between competition and disease avoidance as evolutionary drivers of social structure, as both hypotheses make similar predictions. For instance, under the "Resource Defensibility" hypothesis a correlation between territoriality and the ratio of day range length to home range size is interpreted as evidence that resource defense is the primary modulator of territoriality (Mitani & Rodman, 1979). However, such a correlation is equally consistent with a hypothesis of territoriality as a means of disease avoidance in that species that "trapline" between high yield fruit trees should have longer day ranges and higher disease exposure risk than species that exploit more uniformly distributed resources.

Finally, we suspect that the answer to the question "competition or disease?" will ultimately prove to be "both." We also suspect that resolving the relative importance of competition and disease avoidance in structuring primate social systems will require an expansion beyond the traditional primatological focus on the behavior of individuals and their response to ecological conditions. The lesson of apes and Ebola is that interactions between disease and social structure can only be fully understood by integrating behavior at the individual and social group levels with constraints and pressures imposed by processes at the population and community levels.

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