

## Disease Threats to Wild Apes

It is customary to divide the diseases that threaten apes into two classes, human introduced and “naturally” occurring. However, the impact of modern humans on tropical forests is now so overwhelming that the distribution and prevalence of many pathogens that are currently viewed as “natural” may actually be indirectly influenced by modern human activities. For instance, patterns of human hunting influence the distribution of both apes and reservoir hosts such as bats and, thereby, influence the propensity towards large, explosive outbreaks of Ebola virus in ape populations. Thus, it is probably more productive to focus on the reservoir species and mode of transmission than on the “naturalness” of disease threats to wild apes.

One of the biggest disease threats to wild apes is the deadly Ebola virus, which appears to circulate persistently in bats (Leroy et al. 2005) then occasionally spill over into gorilla and chimpanzee populations (Leroy et al. 2005). Over the last two decades the Zaire strain of Ebola has caused major gorilla and chimpanzee die-offs in Gabon and Republic of Congo which were home to some of the largest protected great ape populations in the world (Walsh et al. 2003, Bermejo et al. 2007). To date, Ebola Zaire has killed roughly one third of the world gorilla population and large numbers of chimpanzees, leading to the World Conservation Union to upgrade western gorillas to *Critically* Endangered status on its Red List of Threatened Species (Walsh et al. 2007). The Cote d’Ivoire strain of Ebola killed chimpanzees at Tai Forest in 1994 (Formenty et al. 1999) while human outbreaks of the Sudan and newly described Bundibugyo strain of Ebola have occurred within a few tens of kilometers of major gorilla and chimpanzee habituation programs in Uganda and Rwanda (Oware et al. 2002, Towner et al. 2008). Thus, Ebola threatens African apes across their range.

It is has also recently become clear that a major threat to wild apes is posed by a second class of pathogens, human respiratory viruses. It has long been suspected that habituating wild apes to close approach by humans in research and tourism programs increased the probability of respiratory pathogen transmission (Wallis & Lee 1999, Woodford et al. 2002). However, the difficulty of diagnosing disease under field conditions has made it difficult to rigorously quantify how large this risk is. For instance, measles was the suspected cause of a 1988 outbreak killed multiple mountain gorillas but the diagnosis was made largely on the basis of clinical symptoms (Hastings et al. 1991). Respiratory disease symptoms have been observed many other times amongst habituated Virunga gorillas over the years and have also been associated with habituated gorilla mortalities at Mondika, in Republic of Congo (P. Mongo, personal communication). Respiratory disease has also caused a substantial death toll at chimpanzee

habituation sites. For instance, 49% of chimpanzee deaths observed during 47 years of study at Gombe, Tanzania were attributed to respiratory disease (Williams et al. 2008). Although the chimpanzee population size at Gombe has been relatively stable (Pusey et al. 2008), clinically diagnosed respiratory disease outbreaks have associated with population declines amongst chimpanzees at Tai Forest, Cote d'Ivoire (Koengen et al. 2008), Bossou, Guinea (Sugiyama 2004) and Mahale, Tanzania (Hanamura et al. 2008) and amongst bonobos at Wamba, DRC ( T. Furuichi, personal communication).

It was not until 2007 that modern molecular methods allowed the robust diagnosis of the pathogens responsible for respiratory disease mortality in wild apes. Two respiratory viruses, respiratory syncytial virus and human meta-pneumovirus (HMPV), caused a large number of chimpanzee deaths during a series of respiratory outbreaks amongst chimpanzees at Tai Forest in Cote d'Ivoire (Koengen et al. 2008). Furthermore, not only did phylogenetic analysis of viral gene sequences sampled from dead chimpanzees strongly imply that the virus had been introduced by researchers, statistical analyses of chimpanzee demography suggested that chimpanzee mortality from human respiratory pathogens stretched back many years, increased with increasing research pressure, and was the major driver of a large decline in chimpanzee numbers. One of the same viruses, HMPV, was also found to be responsible for chimpanzee respiratory disease mortalities at Mahale, Tanzania, which has also seen a decline in the population size of habituated chimpanzees (Kaur et al. 2008).

Human feces is also another potential source of human pathogen transmission to wild apes. The most spectacular such case may have been the infection of chimpanzees at Gombe by polio virus in 1966 (Goodall 1986). However, a variety of other microscopic (e.g. viruses and bacteria) and macroscopic (e.g. helminthes, mites) parasites are known or suspected to be transmitted from human feces to wild apes. For example, gorillas and chimpanzees in Uganda have recently been shown to be infected by human strains of bacteria (Nizeyi et al 2001, Goldberg et al. 2007). Scabies has infected gorillas (Graczyk et al. 2001) in Uganda and Rwanda (Kalema et al. 2002) and chimpanzees in Tanzania (Williams et al. 2008). Yaws has infected gorillas in Congo (Levrero et al. 2007). These parasites tend not cause the high mortality outbreaks typical of respiratory pathogens. However, their long term effects on fertility and/or survivorship may be equally serious.

Vector borne diseases are another potential threat. Wild apes carry their own strains of malaria (P), although it is not know how pathogenic they are. Also as yet unknown is the extent to which spillover from human malaria poses a threat to wild apes. Another mosquito borne disease that is a potential threat to wild apes is Yellow Fever, which circulates endemically in monkey populations in equatorial Africa (Haddow et al. 1951). Very little is known about the dynamics of

Yellow Fever in African primate populations but it is known to cause large population crashes in South American monkeys (e.g. Rifakis et al. 2006). Equally unknown is the impact on apes of other hemorrhagic fever viruses that circulate in the monkey populations of Equatorial Africa (e.g. Dengue virus, Chikungunya virus).

Pathogens that circulate persistently within ape populations are also a threat. For example, simian immunodeficiency virus, the ancestor of HIV in humans, has recently been shown to cause AIDS-like symptoms as well as reduce birth and survival rates amongst chimpanzees at Gombe (Keele et al. 2009). Wild apes are also known to harbor a number of other persistently circulating viruses such as simian foamy virus (Liu et al. 2008), cytomegalovirus (Leendertz et al. 2009), simian T-lymphotropic virus (Leendertz et al. 2004a) whose effects on the reproduction and survival are yet unknown.

Finally, both gorillas and chimpanzees have been observed to die from previously undescribed strains of Anthrax (Leendertz et al. 2004b, Leendertz et al. 2006). As for many of the pathogens described above, the extent of threat posed by Anthrax is not known.

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