

Original Contribution

Modeling the Impact of Ebola and Bushmeat Hunting on Western Lowland Gorillas

Carol Rizkalla,^{1,3} Francisco Blanco-Silva,² and Stephanie Gruver²

¹Department of Forestry and Natural Resources, Purdue University, West Lafayette, IN 47907-2033, USA

²Department of Mathematics, Purdue University, West Lafayette, IN 47907-2033, USA

³Disney's Animal Kingdom, P.O. Box 10,000, Lake Buena Vista, FL 32830-1000, USA

Abstract: The 2003 outbreak of Ebola in the Republic of Congo killed 114 people and up to 800 western lowland gorillas. This outbreak and all outbreaks between 2001–2003 began with human handling of infected animal carcasses. Ebola has since spread, putting the entire gorilla population at risk. An epidemiological model is presented to describe the combined effects of Ebola and hunting on persistence of gorillas. The number of infected gorillas also provides a means of assessing the risk of transmission to humans. Under current harvest practices and the estimated annual outbreak rate, the gorilla population is predicted to undergo a 97% decline within 100 years. Controlling bushmeat hunting may not be enough to prevent extinction if frequent outbreaks occur.

Keywords: bushmeat, Ebola, epidemiology, gorilla, SIR model

INTRODUCTION

Ebola hemorrhagic fever was first recognized in 1976 near the Ebola River in Congo (formerly Zaire) as a severe, often fatal disease in human and nonhuman primates (CDC, 2002). In 13 occurrences since its discovery, human mortality has ranged from 50%–100% (average 59.3%). There are four strains of the virus: Ebola-Zaire (which has the highest mortality rate and is the cause of the most recent outbreaks), Ebola-Sudan, Ebola-Ivory Coast, and Ebola-Reston, the latter of which only occurs in monkeys and apes (CDC, 2002). Ebola typically appears in sporadic outbreaks coinciding with the rainy season and is usually spread in humans within a health-care setting. Incubation

is 2–21 days, and transmission occurs through direct contact of bodily secretions or contaminated objects, such as needles or as will be described, through contaminated meat (CDC, 2002).

In 2003, a Congolese outbreak of Ebola-Zaire killed 114 out of the 128 humans who contracted it (Clover, 2003). Around the same time, 600–800 western lowland gorillas (*Gorilla gorilla gorilla*), encompassing two-thirds of the local population, disappeared from the nearby Lossi Gorilla Sanctuary (Aveling, 2003). An unknown number of chimpanzees (*Pan troglodytes*) also disappeared. Ebola virus was confirmed in six gorilla carcasses. Lossi is 15 km from Odzala National Park, home to 20,000 gorillas, and by 2005, the park population had been devastated (Gross, 2005). Congo and neighboring Gabon hold 80% of the world's gorilla population, and all of these individuals appear to be potentially at risk of infection.

Preventative measures for Ebola have been hindered because the natural reservoir of the virus remains unknown. However, the virus is believed to be zoonotic, maintained in a non-primate animal host native to Africa. Fruit bats have been identified as potential carriers (Biek et al., 2006; Leroy et al., 2005). Contact between bats and gorillas may occur as they compete for fruit during the dry season. This fails to explain how other animals, such as duikers, become infected. Duikers may be infected by eating fallen fruit or by licking fresh carcasses (Rouquet et al., 2005).

The 2003 outbreak began with the handling and consumption of an infected carcass (Rouquet et al., 2005). Although wild animals have been hunted and eaten in Africa for years, bushmeat hunting has increased in recent years because roads provide easier access to the forest. Commercial demand for bushmeat also exists in larger cities (Clarke, 2003). Though apes make up only about 1% of the bushmeat trade, approximately 15,000 chimpanzees and 6,000 gorillas are killed each year across Africa. To be hunted sustainably, apes should lose no more than one animal per square kilometer every 20 years (Stein et al., 2002; Clarke, 2003).

The combined threats of Ebola and overhunting prompted us to develop an epidemiological model to describe the persistence of Ebola and its effects on western lowland gorillas concurrently being harvested. The Kermack-McKendrick model, also known as the *SIR* model, consists of three differential equations and has been applied to a variety of infectious diseases (Anderson and May, 1979). When another organism is involved in disease transmission, the *SIR* model is typically coupled with an *SIR* model for the reservoir. For simplicity, we ignore the unknown host here. We have speculated on the annual rate of an outbreak occurring and provide population projections. The model provides the additional value of estimating the risk to humans of harvesting an infected gorilla.

METHODS

To model the epidemiology of Ebola in gorillas, we chose to develop an *SIR* model, where *S* represents the number of susceptible individuals, *I* and *R* represent infected and recovered individuals, respectively. As an instantaneous model, if individuals move between population classes, they do so within one time step, 1 month in this model. With an incubation rate up to 21 days, 1 month represents the maximum time an individual could move between each class.

We have assumed density-dependent population growth with a carrying capacity set at the current population size, approximately 100,000. We also assumed sufficient migration between populations, as reflected by our initial conditions in which we model the entire gorilla population ($N = 100,000$) and not just that of the Congo and Gabon. Given the rapid spread of Ebola (Walsh et al., 2005) and either numerous hosts (Rouquet et al., 2005) or the mobility of bats as the single host (Leroy et al., 2005), there is no reason to believe that neighboring countries are safe from an outbreak. Additionally, inter-group transmission appears to have been important in past outbreaks (Bermejo et al., 2006).

The number of susceptible gorillas that become infected is a function of the probability of transmission and the proportion of infected gorillas in the population. The transmission parameter typically represents the joint probability of contact and infection. Here, we consider transmission of infection alone and treat the contact rate as the proportion of infected individuals. Because transmission occurs within and between both groups and solitary gorillas (Caillaud et al., 2006), if infected individuals constitute a small proportion of the population, it is less likely that susceptible individuals will come into contact with them. Susceptible gorillas can also become infected due to contact with the Ebola host during an outbreak (see below). Infected animals either die or move to the recovered class by the next time step. We assume recovered animals become immune to further infection and, therefore, do not re-enter the susceptible class. In humans, those who die have not developed a significant immune response to the virus (CDC, 2002). Thus, it is possible that those who recover have developed an immunity to subsequent infection. Finally, the probability of being harvested is equal for each population class. Infected animals may have a lower probability of being harvested if they leave their group. Conversely, being ill may make an individual more vulnerable as it is easier to kill.

The following equations are derived from these assumptions:

$$\frac{dS}{dt} = gN \left(1 - \frac{N}{K} \right) - (d + \eta)S - \frac{bSI}{N} \quad (1)$$

$$\frac{dI}{dt} = \frac{bSI}{N} - (d + \eta + a + \gamma)I \quad (2)$$

$$\frac{dR}{dt} = \gamma I - (d + \eta)R \quad (3)$$

Table 1. Description of the Model Parameters and Initial Conditions^a

Symbol	Description	Value
g	Birth rate	0.113
d	Natural death rate	0.066
b	Ebola transmission probability	0.5
a	Additional death rate due to Ebola	0.937
γ	Recovery rate	0.063
η	Harvest probability	0.06, 0.04, 0.02, 0
N	Total population	100,000
S	Susceptible population	99,150
I	Infected population	800
R	Recovered population	50
α	Outbreak rate	0.636, 0.3, 0.1

^aAll rates and probabilities are annual. Where a range of estimates was tested in the model, multiple values are listed.

where $N(t) = S(t) + I(t) + R(t)$. We extracted the annual birth rate (g) and death rate (d) from Alvarez (2000) (Table 1). Based on the statistic that 6,000 gorillas are taken annually from a total population of approximately 100,000 (Clarke, 2003), we assumed a harvest probability (η) of 0.06. There is no data to provide an estimate for the probability of Ebola transmission (b) among gorillas. Thus, we assumed a chance transmission of 0.5. As a highly infectious disease, the transmission probability may be much higher. Sensitivity analyses demonstrated the transmission parameter was not as significant as others (see Results); thus, we did not test alternative values.

We estimated the death rate due to Ebola ($a = 0.937$) and the recovery rate ($\gamma = 1 - a = 0.063$) as the average of three reports: Only 7 of 143 study gorillas in the Lossi sanctuary survived (Kaiser, 2003); there was a 98% decline in gorilla density in the Minkebe forest of Gabon since 1994, likely due to Ebola (Huijbregts et al., 2003); and there was an 88% decline in chimpanzee indices in Lossi (Leroy et al., 2004). We set the initial conditions according to estimates of the 2003 Lossi outbreak: 800 infected animals (Aveling, 2003), 50 recovered (6.3% of 800), and 99,150 susceptible, summing to a total population of 100,000.

Solving the system of equations provides a boundary equilibrium (S^* , I^* , R^*) for disease-free status in the population:

$$E_0 = \left(K \left(1 - \frac{d + \eta}{g} \right), 0, 0 \right)$$

This equilibrium exists when $(d + \eta)/g < 1$. If the birth rate is greater than the combined natural death rate and death due to harvest, the population can sustain itself. An interior equilibrium also exists. It is not presented here due to its complexity, but is available from the authors.

We did not account for demographic stochasticity, but we included environmental stochasticity in the form of future Ebola outbreaks. There have been seven documented outbreaks in great apes in an 11-year period (1992–2003) (Formenty et al., 1999; Leroy et al., 2004). Therefore, we assumed the annual rate for an outbreak occurring as 0.636 (7/11). Based on the initial conditions of 800 infected individuals, we also assumed that each outbreak leads to the additional infection of 0.8% of the susceptible population. Thus in each model run, if the generated random number was less than 0.636, 0.8% of the susceptible class moved to the infected class. Due to uncertainty in the trigger of an outbreak, we also tested two lower outbreak rates (Table 1). Notably, there were five human outbreaks in less than 3 years (Rouquet et al., 2005). If these coincided with undocumented outbreaks among gorillas, the annual outbreak rate could be greater than one. In this light, our results must be viewed conservatively.

We combined the range of outbreak rates with a range of harvest probabilities to determine the combined threat of future outbreaks and hunting. For each set of parameters, we ran 1000 simulations for 100 years. Finally, we assessed sensitivity of the model to a 10% decrease in transmission, death and recovery, and harvest rates and initial number of infected gorillas. All model runs were seeded for outbreak probabilities to allow comparison.

RESULTS

The model run used to test sensitivity had an outbreak occurring every year, such that the population size after 100 years was 2764.23. The model showed no sensitivity to initial conditions, probability of transmission, or death and recovery rates. A 10% decrease in each of these parameters resulted in an 0.03%, 6.4%, and 9.9% increase, respectively, in the final population size. However, the model was highly sensitive to harvest probability, resulting in a 49.5% change in population size.

All further results are reported in terms of the original values (Table 1). We simulated a combination of harvest probabilities ($\eta = 0, 0.02, 0.04$, and the current 0.06) with three annual outbreak rates ($\alpha = 0.1, 0.3$, and the estimated

Table 2. Population Size of Gorillas in 100 Years from 1000 Simulations under Combinations of Four Harvest Probabilities (η) and Three Rates of Ebola Outbreak (α)

	$\alpha = 0.1$	$\alpha = 0.3$	$\alpha = 0.636$
$\eta = 0$	41,379.29 \pm 231.37	40,532.88 \pm 343.72	39,150.69 \pm 341.14
$\eta = 0.02$	24,793.88 \pm 161.69	24,075.77 \pm 236.7	22,905.95 \pm 235.31
$\eta = 0.04$	11,313.12 \pm 9.64	10,837.04 \pm 131.66	10,074.84 \pm 128.99
$\eta = 0.06$	3582.9 \pm 35.77	3381.59 \pm 50.45	3067.37 \pm 47.91

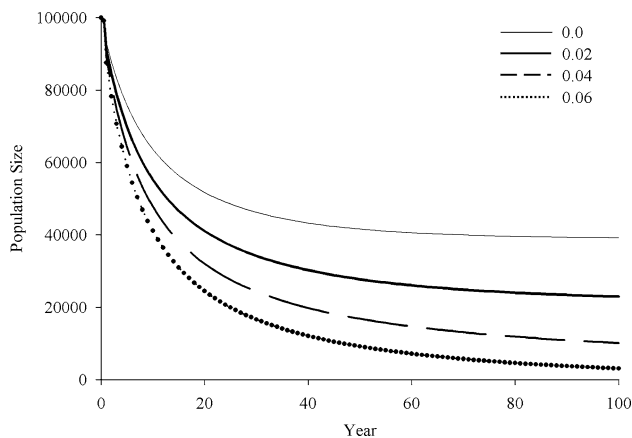


Fig. 1. Mean population size of gorillas under the estimated outbreak rate ($\alpha = 0.636$) with four probabilities of harvest.

0.636). Under current harvest with the estimated outbreak rate, the population size in year 100 was 3067.37 ± 47.91 (Table 2). This increased to $39,150.69 \pm 341.14$ with no harvest (Fig. 1). The population still declined by more than 50% under the most conservative scenario with no harvest and low outbreak probability (Table 2).

According to the model with 6% harvest and the high outbreak rate, the proportion of infected gorillas in the population ranged from 0.9% to 1.25%. This translates to a 0.0058%–0.075% chance that a harvested gorilla is infected. In other words, approximately 1 in every 1500 harvested gorillas would carry Ebola. If 6000 are harvested annually, there could be four cases of gorilla–human transmission each year.

DISCUSSION

We realize our model may be simplistic and contain much uncertainty. Population viability analyses typically contain spatial structure, age structure, and/or historical counts (Reed et al., 2002). However, it illustrates the potential

combined effect of Ebola and harvest on gorillas. Walsh et al. (2003) uses historical surveys to estimate an 80% decline in ape populations within 33 years. Although we did not have historical data to validate our model, the scenario of current harvest probability with any of the three outbreak rates resulted in an 80% decline in 25–26 years on average. Reducing the harvest to 0.04 resulted in an 80% decline in 39–43 years. Thus, a 5% harvest would approximate the findings of Walsh et al. (2003). We believe this adds support to our results.

Efforts to control the bushmeat trade are ongoing, but unfortunately, largely unsuccessful. Walsh et al. (2003) calls for an immediate increase in law enforcement to control the illegal harvest of apes. We echo this sentiment. However, our model suggest that even with no harvest, gorillas will not survive a high frequency of Ebola outbreaks. Two priorities have been proposed to address Ebola: 1) develop a vaccine and 2) determine if natural barriers, such as rivers, can slow the spread of the virus (Tutin et al., 2005). The latter is unlikely if bats are the reservoir, due to their high mobility; however, the predictable spatio-temporal spread of outbreaks (Walsh et al., 2005) may provide focus to such efforts. Increased control of harvesting may be able to buy us the time we need to develop an effective vaccine. Because transmission between gorillas appears to be high, widespread vaccination may be the better option. A hopeful strategy for vaccine development has already been devised (Sullivan et al., 2000; but see Geisbert et al., 2002), and elucidation of the virus’s properties provides greater promise (Yaddanapudi et al., 2006).

Despite efforts to change the eating habits of African villagers, many believe occult forces are behind Ebola. They do not understand that they could limit exposure by avoiding sick or dead animals. The probability of harvesting an infected gorilla (0.0058%–0.075%) appears quite low. Thus to a hunter risking transmission, this may not serve as a deterrent. The model predicted that four cases of gorilla–human transmission could occur annually. Indeed, in four

human outbreaks beginning between October 2001 and December 2002, index patients were infected by gorilla or chimpanzee carcasses (Rouquet et al., 2005). Additionally, Ebola outbreaks occur in localized pockets containing a subset of the gorilla population, and other hunted species such as chimpanzees and duikers would also likely be infected. Therefore, the risk in these areas could be much higher. Alternatives to bushmeat such as livestock and agriculture have been advocated (Brashares et al., 2004). However, such efforts would also result in habitat destruction. Since apes are a small portion of the bushmeat trade, providing alternatives to this subset would be more feasible.

It is quite likely that as gorilla densities decline, so too will the harvest. This would provide a natural termination of Ebola transmission to humans (though not from other sources) and possibly allow gorillas to slowly recover from outbreaks. More likely, at low densities, gorillas might only occur in small pockets that have been isolated from the virus. Before we reach that point in time, if education efforts on the link between bushmeat and Ebola can succeed, the source of one mode of transmission to humans can be controlled, and gorilla populations may continue to persist.

ACKNOWLEDGMENTS

N. Lichti, R. Swihart, W. Karesh, and two anonymous reviewers contributed valuable comments.

REFERENCES

- Alvarez HP (2000) Grandmother hypothesis and primate life histories. *American Journal of Physical Anthropology* 113:435–450
- Anderson RM, May RM (1979) Population biology of infectious diseases: part I. *Nature* 280:361–367
- Aveling C (2003) Massive die-off of great apes reported in Republic of Congo. Conservation et Utilisation Rationnelle des Ecosystèmes Forestiers d’Afrique Centrale. Available: <http://www.bushmeat.org/html/ECOFACTPressRelease2003Feb05.htm> [accessed April 7, 2003]
- Bermejo M, Rodríguez-Teijeiro JD, Illera G, Barroso A, Vilà C, Walsh PD (2006) Ebola outbreak killed 5000 gorillas. *Science* 314:1564
- Biek R, Walsh PD, Leroy EM, Real LA (2006) Recent common ancestry of Ebola Zaire virus found in a bat reservoir. *PLoS Pathogens* 2:e90
- Brashares JS, Arcese P, Sam MK, Coppolillo PB, Sinclair ARE, Balmford A (2004) Bushmeat hunting, wildlife declines, and fish supply in west Africa. *Science* 306:1180–1183
- Caillaud D, Levréro F, Cristescu R, Gatti S, Dewas M, Douadi M, et al. (2006) Gorilla susceptibility to Ebola virus: the cost of sociality. *Current Biology* 16:R489–R491
- Centers for Disease Control and Prevention (CDC) (2002) Ebola hemorrhagic fever information packet. Available: http://www.cdc.gov/ncidod/dvrd/spb/mnpages/dispages/Fact_Sheets/Ebola_Fact_Booklet.pdf [accessed January 10, 2006]
- Clarke P (2003) Wildlife on the menu: the bushmeat crisis is emptying Africa’s forests. Available: http://www.emagazine.com/march-april_2003/0303curr_bushmeat.html [accessed April 7, 2003]
- Clover C (2003) Ebola fever killing lowland gorillas. *The Age*. Available: <http://www.theage.com.au/articles/2003/04/01/1048962755859.html> [accessed April 7, 2003]
- Formenty P, Boesch C, Wyers M, Steiner C, Donati F, Dind F, et al. (1999) Ebola virus outbreak among wild chimpanzees living in a rain forest of Côte d’Ivoire. *The Journal of Infectious Diseases* 179(Suppl 1):S120–S126
- Geisbert TW, Pushko P, Anderson K, Smith J, Davis KJ, Jahrling PB (2002) Evaluation in nonhuman primates of vaccines against Ebola virus. *Emerging Infectious Diseases* 8:503–507
- Gross L (2005) Charting the path of the deadly Ebola virus in central Africa. *PLoS Biology* 3:1846–1847
- Huijbregts B, DeWachter P, Obiang LSN, Akou ME (2003) Ebola and the decline of gorilla *Gorilla gorilla* and chimpanzee *Pan troglodytes* in populations in Minkebe Forest, north-eastern Gabon. *Oryx* 37:437–443
- Kaiser J (2003) Ebola, hunting pushing ape populations to the brink. *Science* 300:232
- Leroy EM, Kumulungui B, Pourrut X, Rouquet P, Hassanin A, Yaba P, et al. (2005) Fruit bats as reservoirs of Ebola virus. *Nature* 438:575–576
- Leroy EM, Rouquet P, Formenty P, Souquière S, Kilbourne A, Froment J, et al. (2004) Multiple Ebola virus transmission events and rapid decline of central African wildlife. *Science* 303:387–390
- Reed JM, Mills LS, Dunning JB Jr, Menges ES, McKelvey KS, Frye R, et al. (2002) Emerging issues in population viability analysis. *Conservation Biology* 16:7–19
- Rouquet P, Froment J, Bermejo M, Kilbourne A, Karesh W, Reed P, et al. (2005) Wild animal mortality monitoring and human Ebola outbreaks, Gabon and Republic of Congo, 2001–2003. *Emerging Infectious Diseases* 11:283–290
- Stein JT, Bailey ND, Wade DL, BCTF (2002) BCTF Fact Sheet: African great apes and the bushmeat trade. Bushmeat Crisis Task Force, Washington, D.C. Available: <http://www.bushmeat.org/cd/fs/FSapesCD.pdf> [accessed March 24, 2003]
- Tutin C, Stokes E, Boesch C, Morgan D, Sanz C, Reed T, et al. (2005) *Regional Action Plan for the Conservation of Chimpanzees and Gorillas in Western Equatorial Africa*. Washington, DC: Conservation International
- Sullivan NJ, Sanches A, Rollin PE, Yang A, Nabel GJ (2000) Development of a preventive vaccine for Ebola virus infection in primates. *Nature* 408:605–609
- Walsh PD, Abernethy KA, Bermejo M, Beyers R, de Wachter P, Akou ME, et al. (2003) Catastrophic ape decline in western equatorial Africa. *Nature* 422:611–614
- Walsh PD, Biek R, Real LA (2005) Wave-like spread of Ebola Zaire. *PLoS Biology* 3:1946–1953
- Yaddanapudi K, Palacios G, Towner JS, Chen I, Sariol CA, Nichol ST, et al. (2006) Implication of a retrovirus-like glycoprotein peptide in the immunopathogenesis of Ebola and Marburg viruses. *FASEB Journal* 20:2519–2530