Ebola Virus Outbreak among Wild Chimpanzees Living in a Rain Forest of Côte d'Ivoire

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An outbreak of Ebola in nature is described for the first time. During a few weeks in November 1994, ~25% of 43 members of a wild chimpanzee community disappeared or were found dead in the Taï National Park, Côte d'Ivoire. A retrospective cohort study was done on the chimpanzee community. Laboratory procedures included histology, immunohistochemistry, bacteriology, and serology. Ebola-specific immunohistochemical staining was positive for autopsy tissue sections from 1 chimpanzee. Demographic, epidemiologic, and ecologic investigations were compatible with a point-source epidemic. Contact activities associated with a case (e.g., touching dead bodies or grooming) did not constitute significant risk factors, whereas consumption of meat did. The relative risk of meat consumption was 5.2 (95% confidence interval, 1.3–21.1). A similar outbreak occurred in November 1992 among the same community. A high mortality rate among apes tends to indicate that they are not the reservoir for the disease causing the illness. These points will have to be investigated by additional studies.

The earliest described outbreaks of a filovirus (Marburg [MBG] virus) were in 1967 in Germany and Yugoslavia [1]. Cases of MBG virus infection occurred in South Africa in 1975 [2], in Kenya in 1980, and in Kenya again in 1987 [3]. Epidemiologic surveys did not identify a reservoir; however, a biting insect was suspected in South Africa.

Ebola (EBO) epidemics were recorded in the Democratic Republic of the Congo (DRC) [4] and Sudan [5] in 1976; investigations did not discover the virus in insects or mammals [6, 7]. EBO reemerged with a single lethal case in Tandala, DRC, in 1977 [8] and a new outbreak in Sudan in 1979 [9]. An outbreak due to a new subtype of the virus, EBO (subtype Reston [EBO-R]), occurred in a colony of cynomolgus monkeys *(Macaca fascicularis)* in a quarantine facility in Reston, Virginia, in 1989 [10]. The same virus was responsible for three further epizootics among monkeys in the United States in 1990, as well as one outbreak in Italy in 1992. Investigations traced the source of all EBO-R outbreaks to a primate export facility in the Philippines [11], but the mode of contamination of this facility was not determined. Although African green monkeys *(Cercopithecus aethiops)* from Uganda were the first animals known to be infected with filovirus [1], the cycle of these viruses in nature remains a mystery.

In November 1994, ethologists studying the behavior of a community of chimpanzees *(Pan troglodytes verus)* in the Taï National Park, Côte d'Ivoire, found 8 dead chimpanzees and noted the absence of other individuals. An epidemiologic survey was done to elucidate the cause of these deaths. Herein, we report the results of investigations that led to the identification of a new subtype of the virus, EBO (subtype Côte d'Ivoire [EBO-CI]), in the blood of a researcher who was probably infected during a chimpanzee necropsy [12].

Materials and Methods

Background. Taï National Park (436,000 ha) represents the last and largest remains of the tropical rain forest belt in West Africa. The park is located near the Liberian border in southwest Côte d'Ivoire (figure 1). The forest in this area is a moist tropical forest of fundamental type, including typical flora, such as *Eremospatha macrocarpa* (Palmaceae), and *Diospyros mannii* (Ebenaceae). Canopy trees average 30 m in height and are dominated by emergent trees of 40–60 m. The average rainfall is 1900 mm per year, and the average temperature is 26°C [13].

Since 1979, a community of wild chimpanzees has been studied in Taï National Park [14]. The home range of the chimpanzees is ~27 km^2 and is situated in the western part of the park (between lat. 5°51′ to 5°54′ N and long. 7°22′ to 7°19′ W), about 10 km from the village of Taï. The closest traditional plantations are located ~2 km from the area (figure 1). The long-term study of the chimpanzees has centered on behavior and ecologic questions, and noninvasive observational methods have been used to follow target individuals on a daily basis from dawn to dusk [15].

Outbreak investigation. A case was defined as any chimpanzee from this community who was missing or found dead during Octo-
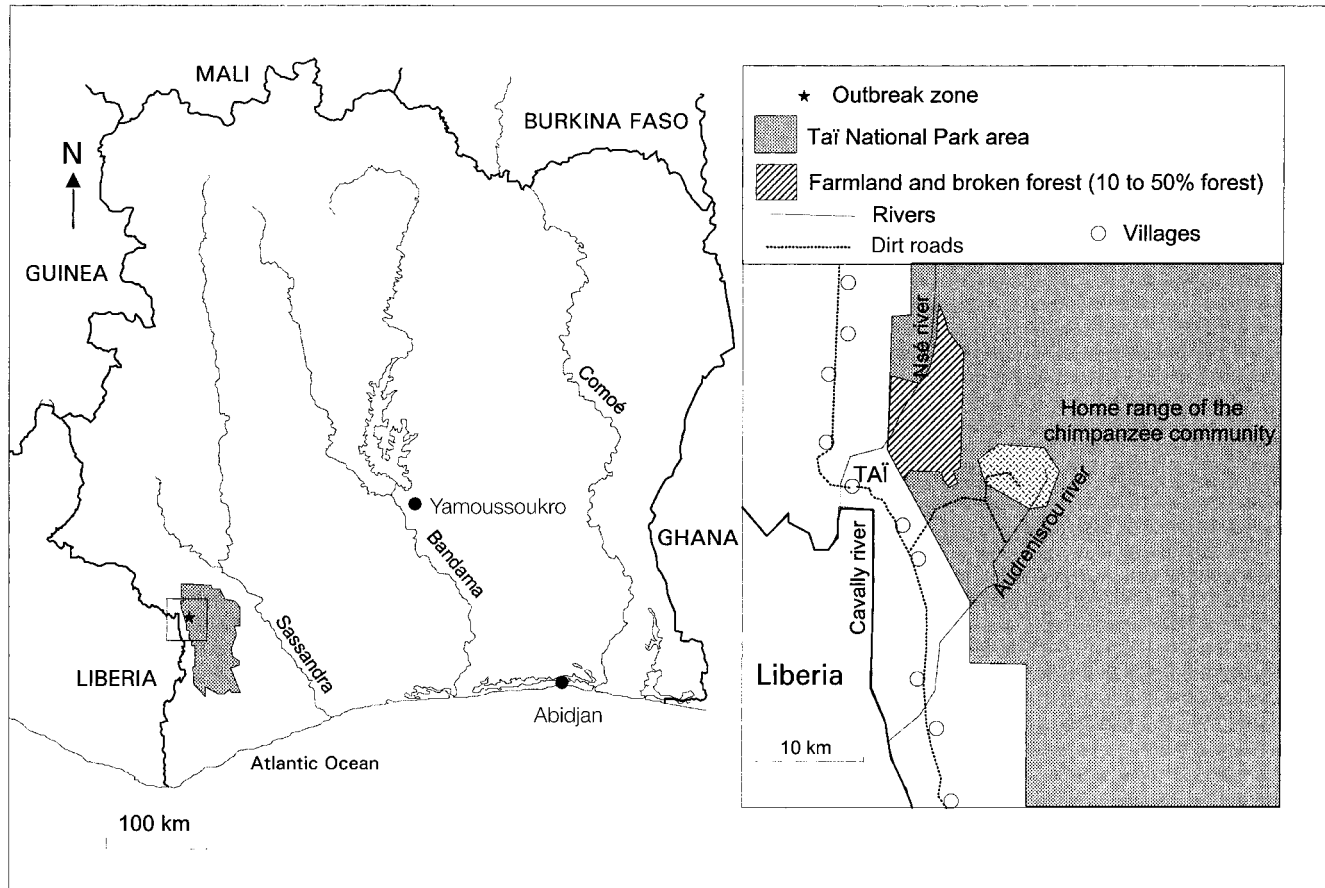
Figure 1. Location of October–November 1994 Ebola outbreak among wild chimpanzee community living in Taï National Park, Côte d'Ivoire. Dotted area indicates home range of chimpanzees.

ber, November, or December of 1994. A definite case was a dead chimpanzee in whom the presence of EBO virus was confirmed by laboratory tests. A probable case was a chimpanzee whose dead body was found. A possible case was a chimpanzee who was missing and whose prolonged disappearance could not be explained by the natural dispersion of individuals.

Cases were identified from the daily records of the investigators. The date of onset was defined as the first day clinical signs were recorded in the chimpanzees (2 cases) or the day of disappearance (10 cases), on the presumption that chimpanzees isolate themselves when they feel sick. The date of death was evaluated from the condition of the bodies. The duration of disease was evaluated by the difference between the date (or probable date) of onset and the probable date of death.

Epidemiologic investigations. A retrospective cohort study was done, using data available from surveys held since 1979, to identify risk factors for cases. The cohort study took place from October to December 1994. The presence and physical status of all chimpanzees that were seen by an investigator were recorded on a daily basis. The sexual activity of females (it was assumed that all adult males were sexually active), the duration of meat consumption (in minutes) during the hunting season, the general situation of the group, and all social interactions with cases (e.g., grooming, behavior while discovering dead bodies, behavior with sick animals) were also noted.

A contact chimpanzee was defined as any chimpanzee observed having direct contact (sexual contact, grooming, touching the body of a dead chimpanzee, caring for a sick animal) with a case in the period between 2 days before the onset of symptoms and the death of the case. This was the same definition as the one used during the 1976 EBO outbreak in DRC [4].

Statistical analysis. Data were analyzed using Epi Info software (version 5.0) [16]. χ^2, Mantel-Haenszel, and Fisher's exact tests were used as appropriate. Relative risk (RR) was calculated with a 95% confidence interval (CI).

Specimen collection. Two necropsies were conducted in the field in November 1994. The first was done by investigators who were not aware of proper sampling methods, and the samples collected were not usable for classical microbiologic investigation. During the second necropsy, samples of kidney, spleen, lung, liver, lymph nodes, and intestinal tissue were collected from a 45-month-old female for histologic examination and bacteriologic studies [12]. Samples were not submitted for virus studies.

Blood specimens were obtained from live chimpanzees during the first week of December 1994 for complete blood cell counts and for serologic studies. Three adult chimpanzees (2 males, 1 female) were tranquilized by use of a dart with a mixture of ketamine and diazepam (Zoletil; Virbac, Carros, France). Blood smears were prepared from peripheral blood to detect infection with malaria or trypanosome parasites.

Laboratory studies. Tissues were fixed in 10% neutral buffered formalin and embedded in paraffin wax. Sections (4 μm) were stained with hematoxylin, eosin, and saffron. Immunohistochemistry studies were done using a pool of monoclonal antibodies known to cross-react with different subtypes of EBO [10]. Additional immunohistochemistry studies were developed using 2 mouse polyclonal antibodies (Institut Pasteur), one of which was prepared with EBO-CI and the other with a recombinant N protein from EBO (subtype Zaire), which was isolated in Gabon [17].

Part of each tissue sample was used for bacteriologic investigations. Hemoglobin and hematocrit levels and platelet, leukocyte, and red blood cell counts were determined by use of an automated analyzer. Differential white blood cell counts were determined manually.

ELISAs were done to determine the presence of IgG and IgM antibodies to Rift Valley and Crimean-Congo hemorrhagic fever viruses, hantaviruses, and chikungunya, yellow fever, and dengue viruses. IFAs were done for Lassa, EBO, and MBG viruses. We also used an ELISA to test for IgG and IgM antibodies against the new subtype, EBO-CI [12].

Results

Epidemiologic investigation. At the beginning of October 1994, the chimpanzee community included 43 individuals: 13 infants (0–5 years old), 4 young adults (6–9 years old), and 26 adults (≥10 years old). Between October and December 1994, 12 community members died or were missing: 1 definite EBO case, 7 probable cases, and 4 possible cases. None of the 4 missing animals have been seen in or outside the studied group of chimpanzees since the outbreak.

The age- and sex-specific attack rates (ARs) during the outbreak are presented in table 1. The 12 cases included 2 infants (AR = 15%) and 10 adults (AR = 38%). There were no statistically significant differences in ARs between males and females; however, ARs were highest among adults (Fisher's exact test, $P < .05$). The mothers of both infants who were

Table 1. Ebola virus attack rate by age and sex among a chimpanzee community in Taï forest, Côte d'Ivoire, November 1994.

Sex, age (years)	No. exposed	No. of cases	%
Male			
0–5	5	0	0
6–9	1	0	0
≥10	7	4	57
All ages	13	4	31
Female			
0–5	8	2	25
6–9	3	0	0
≥10	19	6	32
All ages	30	8	27
Both sexes			
0–5	13	2	15
6–9	4	0	0
≥10	26	10	38
All ages	43	12	28

cases also died or disappeared during the same period; the mother of 1 45-month-old female infant who was a definite case had disappeared 1 week earlier. The other infant, a 26-month-old female, disappeared at the same time as her mother.

The first case was recorded on 25 October and the last on 27 November. The shape of the epidemic curve (figure 2) indicates a point-source or an intermittent point-source outbreak with or without secondary cases.

The geographic distribution of the dead chimpanzees shows a clustered distribution within a radius of 1.5 km within the home range; that zone also corresponded to the most commonly used area of the territory.

Activities associated with a case-contact, such as touching a dead chimpanzee or grooming a case-patient before or during illness, were not significant risk factors (table 2). For infants, having a mother who was a case was a very high risk factor (RR, indefinite; $P < .05$, Fisher's exact test). Animals who engaged in sexual activity anytime from October through November had an RR of 2.5 (95% CI, 0.9–7.1), and those who consumed meat had an RR of 5.2 (95% CI, 1.3–21.1). A stratified analysis was done to examine the effects of the quantity of meat eaten on the occurrence of cases: Among meat consumers, the risk increased with the quantity of meat ingested (χ^2 for linear trend = 14.8; $df = 1$; $P < .001$) (table 3). The 2 cases who did not eat meat were infants.

Clinical investigation. Most of the chimpanzees who disappeared did so without first showing clinical signs of illness. Six days before disappearing, a 24-year-old male showed signs of abdominal pain, lethargy, and anorexia for 1 day. The day before his disappearance, he was observed in apparently fair condition. One day before her death, a 45-month-old female ate throughout the day (43% of the time) but looked very tired and spent 40% of her time resting. For 8 cases (1 definite and 7 probable), we found a body that was identifiable. The length of disease (mean, 5.5 days; range, 2–14 days) could be evaluated for 6 cases.

The bodies of 2 dead chimpanzees had only minimal autolysis. A 13-year-old female was found in the fetal position on the forest floor. Necropsy revealed that the intracardial blood was brown and not coagulated. No gross lesions were observed on the viscera. The 45-month-old female mentioned above was found lying on her side; her rib cage was full of blood, and her lungs were dark red. Tissues from multiple organs were collected from this individual.

Other observations. Before the outbreak, from 10 to 19 October, the chimpanzee group fed on one fig tree *(Ficus goliath)*. The tree was full of fruit, and everyday, pigeons were seen feeding on the figs. Two times during October and November, chimpanzees were seen hunting: The first hunting event took place on 19 October, 7 days before the beginning of the outbreak, and a young red colobus monkey was killed and eaten. The 2 main consumers on 19 October were among the early cases. The last 2 cases fed on an adult red colobus on 17 November, 11 days before disappearing.

For the period 1991–1994 (figure 3), demographic analysis revealed that the annual mortality rate had two peaks: 27% for
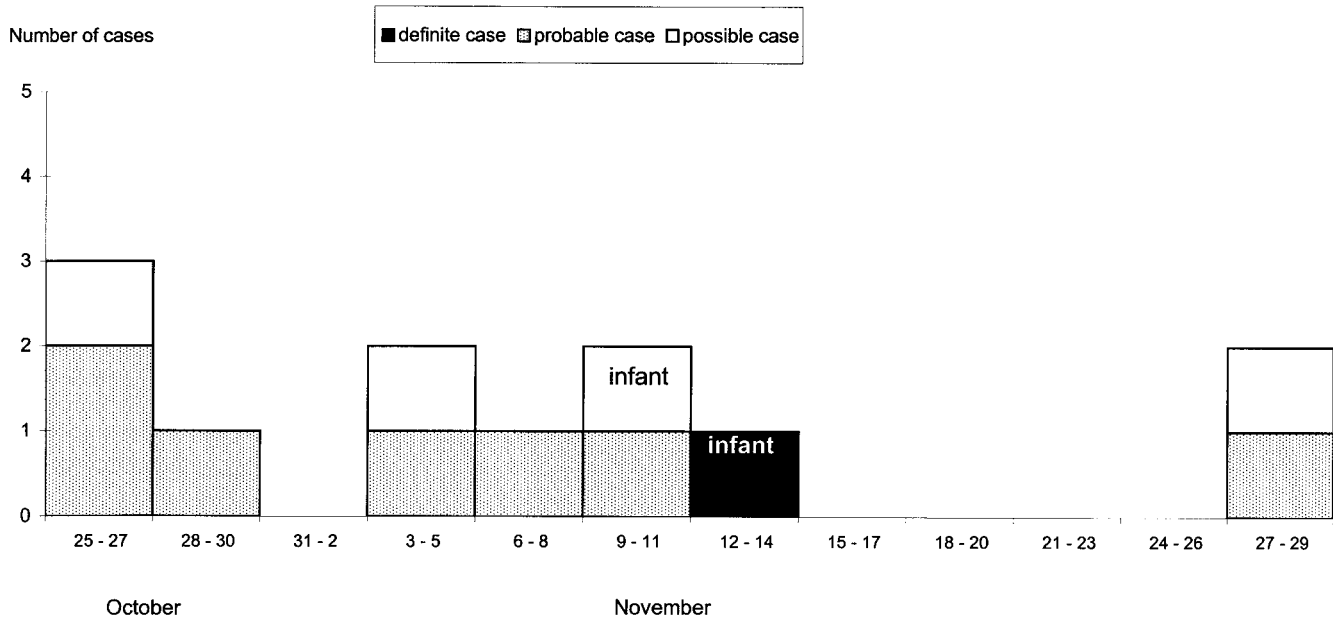
Figure 2. Distribution of cases by day of onset (or probable day of onset; see definition in text) during outbreak among chimpanzee community in Taï forest, Côte d'Ivoire, October–November 1994.

1992 and 37% for 1994. For 1991 and 1993, the respective annual mortality rates were only 4% and 9%. During November 1992, 8 chimpanzees died or were missing (AR = 17%), and 12 more were dead or missing in October and November 1994 (AR = 28%).

Histopathology. Liver lesions consisted of numerous small foci of necrosis. The spleen presented extensive areas of necrosis of the red pulp. There were single, large, amorphous, acidophilic inclusion bodies in the cytoplasm of splenic macrophages of the red pulp, some hepatic Kupffer's cells, and rare hepatocytes. In a mesenteric lymph node, the cortical pulp exhibited pyknosis and necrosis of the centrofollicular areas. Several macrophages contained big acidophilic inclusion bodies. Inclusion bodies were compatible with viral inclusions.

The results of immunohistochemistry using monoclonal antibodies have been previously described [12]. A detailed description of the histopathology and immunohistochemistry studies done with polyclonal mouse sera are reported separately [17]. EBO-specific immunohistochemistry of the liver, spleen, lymph nodes, and lung were positive, demonstrating a large distribution of the virus in all organs. Macrophages, mostly the vascular ones in the spleen and liver, were the immunopositive cells.

Other laboratory results. Culture results were negative. Because of the hemorrhagic lesions, we attempted to identify *Bacillus anthracis* in tissues from the 2 animals on which necropsies were done, and tests were negative.

Serologic tests were negative for the following viruses: Rift Valley fever, Crimean-Congo hemorrhagic fever, chikungunya,

Table 2. Risk factors during Ebola outbreak among chimpanzees, according to a retrospective cohort study, Côte d'Ivoire, 1994.

Risk factor	Cases		Non-cases		RR	95% CI	P*
	Exposed	Not exposed	Exposed	Not exposed			
Engaged in sexual activity in October and November	8	4	11	20	2.5	0.9–7.1	.07
Ate meat in September and October	10	2	11	20	5.2	1.3–21.1	.005
For infants (age <5 years) whose mothers were cases	2	0	0	15	Indefinite	—	.007
Contact with a case	3	9	10	21	0.8	0.2–2.4	.73

NOTE. RR = relative risk: [a/(a + b)]/[c/(c + d)]. 95% CI = confidence interval of RR with α = 5%.
* By Fisher's exact test.

Table 3. Relationship between meat consumption and cases of Ebola infection among a chimpanzee community, Taï forest, Côte d'Ivoire, September and October 1994.

Meat consumption	No. exposed/ no. of cases (%)	OR (95% CI)
No. of minutes spent consuming meat		
0	22/2 (9)	1 (reference)
0–100	11/2 (18)	2.2 (0.3–18.4)
>100	10/8 (80)	40.0 (4.8–334.8)
Total no. of meat consumers	21/10 (48)	5.2 (1.3–21.1)
Total no. of cohort members	43/12 (28)	

NOTE. OR = odds ratio; CI = confidence interval. χ^2 for linear trend = 14.8; df = 1; P = .0001.

Lassa, EBO, MBG viruses, and hantaviruses. The 3 chimpanzees had IgG but not IgM antibodies against yellow fever and dengue viruses. In addition, the 3 chimpanzees sampled were negative for antibodies against the new subtype of EBO, EBO-CI.

Hematology test results were normal. No hemoparasites were found on blood smears.

Discussion

Immunohistochemistry techniques clearly established a diagnosis of EBO infection in 1 individual who died in November 1994. During this epidemic, the other dead and missing chimpanzees most likely died from the same pathogen. Clinical findings indicate a short illness followed by a sudden death. Three surviving chimpanzees with high risk factors for EBO (they ate large amounts of meat) were seronegative for EBO-CI. This indicates that they probably were never infected, which is compatible with the hypothesis that the case fatality rate

was probably near 100% among the infected animals. Lesions described in this report are similar to those observed in experimentally infected monkeys [18]. The most severe histologic lesions were present in the liver, spleen, and lymph nodes. Contrary to previous reports in naturally infected humans or experimentally infected monkeys, hemorrhagic, thrombotic, and vascular lesions were not observed and neither were any aspects suggesting the evolution of disseminated intravascular coagulation. However, the pathologic features of EBO-CI could be different from those in other EBO infections, which raises questions regarding the physiopathology of this new subtype and of the natural disease in chimpanzees. This is the first time that a forest cycle for a filovirus in Africa has been confirmed. The short duration of disease and the high mortality rate for chimpanzees indicate that these animals would be a very poor reservoir for this virus.

The epidemic curve tends to indicate a point-source or an intermittent point-source contamination. Six dead chimpanzees were discovered by the community, and of them, only 2 (1 of whom was a definite case) were touched by a total of 10 community members. Chimpanzees usually do not touch the dead bodies of other chimpanzees.

Contacts, such as taking care of a sick animal, touching a corpse, or grooming a case, were not risk factors for EBO-CI; however, very close contact with cases, such as sexual activity (low risk) or the contact between mothers and infants (high risk), were risk factors. Infants <5 years of age are usually breast-fed by their mothers and spend most of their time clinging to their mothers. The 1 definite EBO-CI case (an infant) appeared to be a secondary case; the infant's mother disappeared a week earlier. The other infant disappeared with its mother and possibly died from starvation or from the virus infection. These results are compatible with dissemination patterns observed in human EBO outbreaks. The immunohistochemistry studies showed that pulmonary parenchyma was affected, but in view of the epidemi-
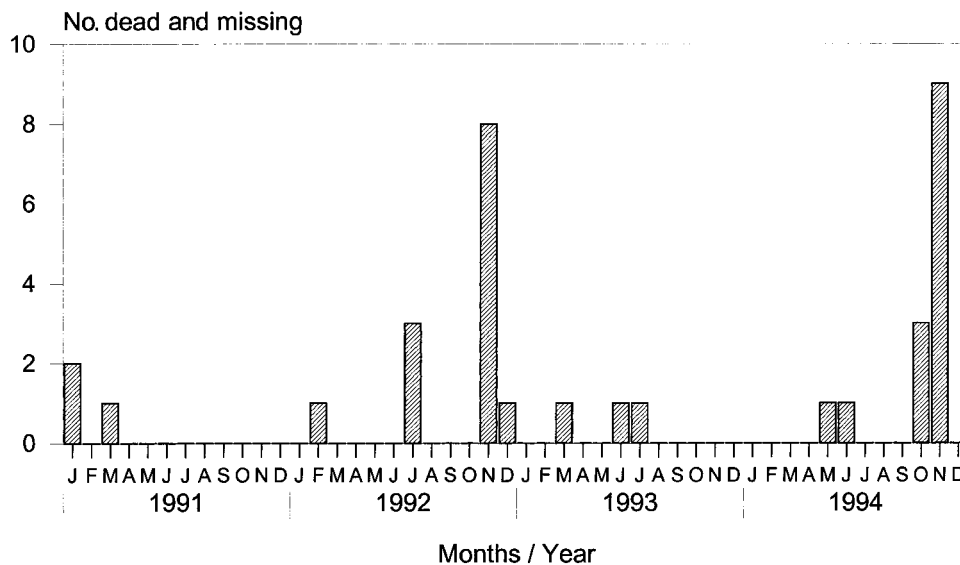

Figure 3. Fluctuation of monthly mortality rate among chimpanzee community studied in Taï forest, Côte d'Ivoire, 1991–1994.

ologic results, aerosol transmission appeared to be very unlikely in this outbreak. During this natural epidemic, EBO-CI infection appeared to have originated from an intermittent point source and to have spread in the chimpanzee community through very close contact.

Our data show that the consumption of meat during September and October was the highest risk factor for becoming infected with EBO virus and that the risk increased with the quantity of meat eaten (tables 2, 3). Chimpanzees may become infected from prey that they eat. In Taï forest, red colobus *(Colobus badius badius)* represents 81% of the monkeys hunted by chimpanzees [19].

The red colobus that was hunted on 19 October might have been infected with EBO and thus been responsible for the first cases of EBO, and the colobus hunted on 17 November might have been responsible for the last 2 cases. However, the last 2 cases were also contacts with the definite case and therefore might have been secondary cases; in this scenario, the incubation period would have been 12 days. During the latter part of October, we mainly followed females or selected individuals who hunted less frequently than males; therefore, we missed following some hunts, among which may have been a hunting party responsible for the second epidemic wave (3–14 November).

Samples from 3 chimpanzees were tested and found to be negative for EBO. Of the 3 chimpanzees, 2 were involved in the recorded hunting parties (1 on 19 October and 1 on 17 November). Both were observed eating small quantities of meat. These data are consistent with those gathered during an EBO outbreak that occurred among humans in Gabon during February 1996 [20]. A chimpanzee suspected of being infected with EBO was at the origin of this outbreak; at least 20 people were identified as having had contact with the chimpanzee meat, but only 18 became infected.

Colobus monkeys may have been the EBO source for these chimpanzees; however, if red colobus were EBO carriers, epidemics would be expected to occur throughout the year or throughout the hunting season, and such was not the case. Red colobus are widely distributed around the Taï National Park and represent one-third of the monkeys in the park. If they were commonly infected, a rather different picture of EBO infection would prevail. Colobus monkeys may be an intermediate host, itself contaminated by the true reservoir during October and November, the end of the hunting season. In the Taï forest, red colobus monkeys (*C. badius badius*) are strictly vegetarian (33% leaves, 33% unripe fruits, 33% flowers) [20a]. Their home range area is ~1 km², and they live in multi-male groups of 60–100 individuals, spending most of their time in the canopy and emergent trees [21]. Their ecologic niche could be the place where the reservoir of EBO virus goes into hiding. They could be contaminated from food (perhaps a plant virus), from a specific arthropod of the upper strata, or through contact with virus via small mammal secretions. Ecologic and behavior studies of colobus monkeys could help identify which animal species they are in contact with that may be a candidate for EBO reservoir.

Before the beginning of the outbreak, the chimpanzee community spent a long time in a fig tree *(F. goliath)* that was full of fruits. We observed many birds in the tree during the day, and no doubt rodents, fruit bats, and other species were feeding on the tree during the night. The *F. goliath* could have played a role in the outbreak, being a focal point for different species, allowing vectors a cycle, and putting different species in contact with one another. We have no history of such a phenomenon during the 1992 outbreak for the chimpanzees.

In the 1992 and 1994 outbreaks, deaths peaked in November, the end of the rainy season. EBO virus has emerged during recent years in the chimpanzee community and twice at the end of the second rainy season. The last 20 years have been characterized by a drought in all of West Africa. The Guinean forest zone has also suffered from an important deforestation. These factors have led to major perturbations of the rainfall [22], seasons, and temperature. In Taï, we noted that the long dry season (December to February), the long rainy season (March to June), and the short dry season (July and August) have been much drier in recent years. Conversely, the short rainy season (September to November) has had more abundant precipitation. During 1992 and 1994, this phenomenon was particularly marked.

The habitat in the region has also been modified constantly by human migration from regions north of the forest belt [23]. This process has sharply increased in the last 6 years since the start of the civil war in Liberia; the influx of refugees doubled the local population between early 1992 and early 1993 and again in the summer of 1994. Massive migration of humans with their domestic animals and other commensal organisms could explain environmental perturbation near the forest and consequently in the forest. The increased deforestation pressure resulting from this influx would also result in perturbation of the habitat. Crop activities have developed on the edge of the park and in the park itself. Illegal plantations and poaching into the Taï National Park have increased from 1985 to 1995 and led to the existence of a large area of farmland and broken forest [24]. This area was only 2 km from the home range of the chimpanzees that were studied (figure 1).

The emergence of infectious diseases has often been linked to ecologic changes. The environment and climatologic perturbations recorded in Taï could have combined to change the demographic parameters of the EBO reservoir or some aspect of its behavior. The seasonal character of the two epidemics could indicate a demographic or a behavioral aspect in the vector or the reservoir species. November is the end of the rainy season in Taï, and increased numbers of some insects or small mammal species might explain the outbreak of infections at that time.

Further studies must test alternative explanations and investigate the natural reservoir of the EBO virus, which is still unknown. Such a study would allow a better understanding of some of the transmission mechanisms of EBO within and between species. Both active searching for cases of hemorrhagic fevers and serologic surveys should be done in the region's health centers. In particular, more research is needed to evaluate

the capability of EBO to leave its forest cycle and to emerge in an urban environment and in human populations. Studies in Côte d'Ivoire should provide a unique opportunity to trace EBO to its origins.

This paper describes for the first time an EBO outbreak in nature: A high mortality rate was recorded among chimpanzees in Taï National Park. Twice in 1996 in Gabon, dead chimpanzees were linked with EBO outbreaks, and mortalities among chimpanzees and gorillas have been recorded in the forest of Minkouka, Gabon, in November 1994 [25]. The possibility exists that larger numbers of wildlife (e.g., nonhuman primates, predators, scavengers) in other parts of the rain forest could also be affected by the virus; thus, EBO appears to be a threat to the conservation of fauna in the rain forest of Africa.

Acknowledgments

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