

COMMENTARY

Why Do Chimpanzees Die in the Forest? The Challenges of Understanding and Controlling for Wild Ape Health

CHRISTOPHE BOESCH*

Department of Primatology, Max Planck Institute for Evolutionary Anthropology, Leipzig, Germany

Tourists from all over the world travel to different African countries to see the great apes in their natural environment. This is a booming component of the ecotourism market. But what is the risk of disease transmission when humans are in close proximity to the apes [Butynski & Kalina, 1998; Wallis & Lee, 1999]? Our recent publication provided the first clear evidence of transmission of respiratory disease from humans to chimpanzees [Köndgen et al., 2008]. At the same time many remain skeptical of the magnitude of such a risk or may think we cannot ask tourists to wear surgical masks. First, I want to provide an account of how we were forced to become convinced that disease is a central issue to ape survival. Second, I want to address some of the important challenges that we have to resolve in order to understand the natural causes of mortality in wild chimpanzees and how we can protect them from disease risks.

FIRST ENCOUNTERS WITH A DEADLY DISEASE

On 16th November 1992, while following a group of chimpanzees, the observers saw Ondine, the old dominant female of the North Group, lying on stomach, dead. Her face was covered with maggots and no injury was visible. Her 1-year-old son lay dead close nearby. She must have been dead for over 10 hr because her skin had started to crack. The other chimpanzees were giving alarm calls and repeatedly dragged the body over short distances. The next morning, when the observers arrived they realized that her body was almost totally covered by leafy branches, presumably by chimpanzees. After some days, we realized that six more chimpanzees were missing. Eight disappearances out of 74 individuals within 12 days! Only three bodies were found in the following days. What could have produced these sudden disappearances of seemingly healthy individuals? We first thought about an “encelo-myocardium” that is known to produce sudden death in animals such as pigs and rodents, but then also considered anthrax or poisoning.

Without any fresh tissue samples it was impossible to solve the case.

Two years later, starting on 24th October 1994, we lost another 10 individuals out of 82 within 2 weeks. Six corpses were discovered by the chimpanzees, of which two were fresh enough to take tissue samples. After 3 months of analysis and one student being infected, we were able to identify the source of death: a new strain of the now too famous Ebola virus, Ebola Côte d’Ivoire [Formenty et al., 1999; Le Guenno et al., 1995]. Sixteen years after its first appearance in Kikwit, RDC, the Ebola virus reappeared in the Tai chimpanzee study group located over 2,000 km away!

This was not the first time individuals of our study group disappeared, but it was the first time so many disappeared at roughly the same time. Were previous disappearance also attributable to disease? Chimpanzees live in fission–fusion societies in which only a small number of group members are seen every day. So it is only after weeks of absence that we start to wonder if an individual might have disappeared, at which point it becomes difficult to find clues about what happened. Additionally, it is very rare to see chimpanzees showing symptoms of disease. In a few cases, we have seen some individuals isolating themselves from the rest of the group soon after we noticed the first symptoms of illness. However, these individuals were not seen again until they fully recovered. Field researchers typically attributed sudden disappearances of healthy individuals either to emigration, if it is a young maturing female, or to intergroup violence or predation pressure [Boesch, 1991; Boesch & Boesch-Achermann, 2000]. Disease outbreaks were known from Gombe chimpanzees, but we thought that the artificial feeding with bananas, unique to Gombe,

*Correspondence to: Christophe Boesch, Department of Primatology, Max Planck Institute for Evolutionary Anthropology, Deutscher Platz 6, 04103 Leipzig, Germany.
E-mail: boesch@eva.mpg.de

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might be responsible. Furthermore, we knew from National Geographic articles that both Jane Goodall and Dian Fossey had played with gorilla and chimpanzee infants and we believed that disease transmission would not occur if we avoided direct physical contact with the chimpanzees.

In 1979, when my wife Hedwige Boesch-Achermann and I started the research project on the wild chimpanzees in the Tai National Park of Côte d'Ivoire, we wanted to contribute to the protection of the chimpanzees and therefore decided not to provision the animals and accept the additional effort required to habituate them. Therefore, the Ebola outbreak came as a shock to us, as diseases remained an issue despite avoiding close contact with the chimpanzees.

Where did the chimpanzees get the disease? Ebola was not the easiest disease to start investigating transmission routes, as no reservoir species is known for it [Le Guenno et al., 1995]. However, four different transmission routes were theoretically possible. The first is chimpanzee-specific pathogens that are naturally circulating and are not influenced by other species (Hypothesis 1). An alternative route would be if the chimpanzees were infected by another species naturally occurring in the forest (Hypothesis 2). A third less reassuring route could be that ecological changes prevailing in the region owing to climatic changes resulted in the emergence of new pathogens within the forest environment (Hypothesis 3). The last, and the most disturbing, route would be pathogens introduced by humans in the forest (Hypothesis 4), either directly by the researchers following the chimpanzees in the forest, or by poachers from the local populations entering the park or indirectly by animals that live commensally with humans and come into contact with wild animal species (mice and rats are well-known vectors).

CHALLENGE ONE: DISCOVERING THE CAUSE OF MORTALITY IN WILD CHIMPANZEES

In May 1999, 3 days after the first chimpanzee was observed with a runny nose, all group members were found to exhibit the same condition. However, some became very weak immediately and could barely move. This was the first time in 20 years of observation that we saw such a rapid deterioration. It took us over 1 week to realize that some individuals had already died within the first 5 days. This outbreak killed 5 adult chimpanzees and 1 juvenile out of 32 individuals, whereas 4 infants died of starvation after their mothers disappeared [Leendertz et al., 2006a]. We first thought that it could be measles, but laboratory tests excluded measles and other respiratory human viruses as the cause of death [Formenty et al., 2003]

This illustrates one of the major challenges that we face when trying to understand what killed the chimpanzees, namely that chimpanzees might have pathogens that are not yet known to science. It was only after Fabian Leendertz developed a technique to test for new strains of known pathogens that we could start to understand what happened in the forest [Leendertz et al., 2004a, 2006a]. To make a long story short, it took us 9 years of constant work to determine the pathogens that were responsible for the death of the chimpanzees in the May 1999 outbreak. For later respiratory outbreaks it only took a few weeks as analyses could be based on previous experience.

With this new technique, we discovered new chimpanzee-specific *Simian T-lymphotropic virus* (STLV) type 1 in the individuals that died from this outbreak [Leendertz et al., 2003, 2004a]. Sadly, this did not seem to provide us with a satisfactory explanation for the deaths we observed: STLV is known in human to have an immuno-depressive effect but does not result in high mortality. However, it certainly prepared us to look for new unknown strains of known pathogens. The next one we uncovered was a new strain of *Bacillus anthracis* [Leendertz et al., 2004b]. This clearly could explain more recent deaths of some individuals, but it was not found in individuals that died during the May 1999 outbreak. The search continued and after a few more years, we identified two distinct and new strain types of *Streptococcus pneumoniae* in those dead individuals [Chi et al., 2007]. They were sufficiently different from known human strains so that we could exclude an infection by humans.

CHALLENGE TWO: UNDERSTANDING THE DYNAMICS OF MULTIPATHOGEN INFECTIONS IN WILD APES

While trying to clarify what happened in the May 1999 outbreak, we had another outbreak in 2004 in the South Group. We found that different combinations of bacteria all involved in respiratory infections occurred within a same chimpanzee [Table I; see also Fang et al., 2007; Köndgen et al., 2008]. Interestingly, the mix of bacteria seemed to be distinct according to which community the chimpanzees belonged. These were interesting results, but they did not solve our mystery, as classically these respiratory bacteria are only secondary infections and not the primary cause of the disease. Therefore, we turned our attention back to the viruses, although we did not find any during our first analysis [Formenty et al., 2003]. Eventually, we isolated two typical human respiratory paramyxoviruses that infected the dead chimpanzees, respiratory syncytial virus (RSV) and

TABLE I. Preliminary List of Pathogens Detected in Dead Tai Chimpanzees for Which we Could Obtain Good Tissue Samples

| Name | Lou | Léo | Lef | Dor | Gar | Gis | Noa | Léo | Kad | Old | Vir | Oph | Ore | Iba | Raf | Tit ^a | Can | Vas | |
|------------------------------|--------|--------|--------|---------|---------|---------|--------|--------|---------|--------|--------|--------|--------|--------|--------|------------------|--------|--------|----|
| Group ID | NG | NG | NG | NG | NG | NG | MG | MG | MG | SG | SG | SG | SG | SG | SG | SG | EG | EG | |
| Death date | 5-1999 | 5-1999 | 5-1999 | 10-2001 | 10-2001 | 10-2001 | 2-2002 | 2-2002 | 11-2001 | 6-2002 | 3-2004 | 3-2004 | 3-2004 | 2-2006 | 2-2006 | 9-2000 | 2-2006 | 2-2006 | |
| Virus | | | | | | | | | | | | | | | | | | | |
| Retrovirus | | | | | | | | | | | | | | | | | | | |
| STLVcpz* | + | - | - | + | | | - | + | + | - | | | | | + | | | | |
| Foamy* | - | - | + | + | | | + | | | + | | | | | + | | | | |
| Herpesvirus | | | | | | | | | | | | | | | | | | | |
| LCV1* | + | + | + | + | | | + | + | + | + | | | | | - | + | | | |
| Paramyxovirus | | | | | | | | | | | | | | | | | | | |
| RSV | ++ | - | ++ | - | | | - | - | - | - | - | - | - | ++ | | | ++ | | - |
| HMPV | - | - | - | - | | | - | - | - | - | + | + | + | | | | - | | - |
| Bacteria | | | | | | | | | | | | | | | | | | | |
| <i>Bacillus anthracis</i> * | - | - | - | ++ | ++ | ++ | ++ | ++ | - | - | | | | | - | | | | |
| <i>Strep. pneumonia A</i> * | ++ | ++ | ++ | - | - | - | - | - | - | - | - | - | - | - | | | ++ | | ++ |
| <i>Strep. pneumonia B</i> * | - | - | - | - | - | - | - | - | - | - | ++ | ++ | ++ | ++ | | | - | | - |
| <i>Pasteurella multocida</i> | - | - | - | - | - | - | - | - | - | - | + | + | + | - | + | | - | | + |
| <i>Klebsiella pneumonia</i> | + | | | | | | | | | | + | | + | | | | | | |
| <i>Hemophiles influenzae</i> | - | + | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | | - |

NG, North Group; MG, Middle Group; SG, South Group; EG, East Group; STL, Simian T-lymphotropic virus; RSV, respiratory syncytial virus; HMPV, human metapneumovirus; LCV1, lymphocryptovirus type 1.

^aTita died from a violent death and not from a disease.

*All pathogens marked with a star were new strains first described in our study (Ehlers et al., 2003; Leendertz et al., 2008).

human metapneumovirus (HMPV), the last one discovered only in 2004 [Köndgen et al., 2008].

Thus, currently most likely scenario seems to be that the deaths we observed in the Tai chimpanzees resulted from a mix of both a virus transmission from humans to chimpanzees for the primary infection (Hypothesis 4), combined with an increased sensitivity to bacteria that are most likely chimpanzee-specific for the secondary and deadly infection (Hypothesis 1). At the same time, we now know that transmission of STLV viruses from red colobus monkey to chimpanzee occurs in the Tai forest (Hypothesis 2), and that chimpanzees might get infected from deadly bacteria occurring naturally in the forest, such as anthrax (Hypothesis 2). Multiple infections have now been documented in almost all individuals that were tested from Tai (see Table I), and it stresses the challenge of trying to implement effective prevention from disease for the wild chimpanzee populations.

CHALLENGE THREE: IMPLEMENT PREVENTIVE MEASURES TO PROTECT WILD CHIMPANZEES

As we observed some of the chimpanzees from the habituated groups dying from disease, we immediately reacted by trying to prevent further illnesses and deaths. Although still not knowing the cause of mortality, we adopted a precautionary approach and implemented health rules as our knowledge increased. Our strategy was that as long as we did not know otherwise, we would assume that humans were responsible for some of the diseases and therefore try to prevent further transmission. Table II illustrates the chronology of the different rules we have had to implement in order to limit such risks. We have tried to limit the risks by having rules working at three levels:

1. Decrease human presence in the forest: by reducing the presence of humans in the research

TABLE II. Development Over Time of the Hygienic Rules Implemented by the Tai Chimpanzee Project

| Date of implementation | Health and hygienic rules | Epidemiologic rationale | Effect |
|------------------------|---|---|---|
| Up to October 1992 | No loud talking, no smoking | Only poachers talk loud | ↓ Stress of human presence |
| | Never go in the forest when ill | Ill persons present higher infection risk | ↓ Introduction pathogens |
| | Max. two family members in camps | Decrease pathogen introduction in camps | ↓ Introduction pathogens |
| | Garbage well protected | Prevent animal contact with human rests | ↑ Hygiene in camps |
| October 1994 | Bring back all food remains | Infant chimpanzee curious of any remains | ↓ Respiratory pathogens |
| | Human feces buried in the forest | Intestinal worms survive in the forest | ↓ Intestinal pathogens |
| | Collaboration with veterinary | Guidance about disease risk and prevention | Elucidating disease identity |
| May 1999 | Vaccinations for everyone ^a | For known chimpanzee costly pathogens | ↓ Viral infection |
| | Keep a 5 m distance | Aerosol carries 3 m away | ↓ Direct aerosol infection |
| Early 2000 | Constant veterinary presence | Guarantee good sample collection | ↑ Identification pathogens |
| June 2002 | “Hygienic barrier” out of camp ^b | Prevent camp/village introduction | ↓ Introduction pathogens |
| | Carry human feces back to camp | Worms in feces should be buried 50 cm deep | ↓ Intestinal pathogens |
| March 2004 | Surgical masks when with chimpanzee | Sneezing can carry ~10m away | ↓ Direct aerosol infection |
| | No spitting in the forest | Saliva very rich in respiratory pathogens | ↓ Respiratory pathogens |
| | No family member in camps | Children are main carriers of pathogens | ↓ Introduction pathogens |
| February 2008 | 8-Day quarantine ^c | Most viral infections disappear within 1 week | ↓ Healthy carriers in forest ^d |

^aYellow fever, tuberculosis, measles, and poliomyelitis. In 2006, we added meningitis.

^bClothes and shoes disinfected whenever entering or leaving the forest.

^cFor international travelers arriving in the country with the last 3 days to stay in the camps of the project.

^dHealthy carriers are humans without symptoms but who carry potential infectious pathogens.

camps particularly family members, wives and children of the field assistants and nonscientific visitors.

2. Decrease introduction of pathogens into the forest: by making hygienic barriers in which clothes worn in the forest are not taken to camp and boots are washed before entering the forest.
3. Limit the risk of contamination to the chimpanzees: by making it obligatory to wear masks that block most of the aerosol transmission, and by not leaving any human remains in the forest.

We have not yet had sufficient time to know if these measures are effective. However, a 100% risk-free solution is probably never going to be possible, and we have tried to implement measures that allow research to continue while at the same time increase the protection of the apes. This will require a constant evaluation of our hygienic rules and an important collaboration with veterinarians to identify the pathogens and risks [Leendertz et al., 2006a,b].

FINAL CONSIDERATIONS

Detailed data are scanty concerning what pathogens are affecting wild apes. Respiratory diseases have been reported from most wild populations and this should be a concern owing to the close proximity of humans and apes in research and tourist projects. Furthermore, new data show that, similar to the Tai chimpanzees, other wild ape populations are in contact with unknown pathogens, such as the Ivorian anthrax strain killing gorillas and chimpanzees in Cameroon [Leendertz et al., 2006a,b] or the different foamy virus strains [Calattini et al., 2007]. We should expect more of such discoveries as we more closely examine wild ape health. Owing to the threats that apes face, we all should take every precaution and make all possible efforts to reduce the risk to them. In particular, ape-viewing tourist projects in Africa and Asia have an obligation to explain to the tourists the reality of the threat that human disease transmission represents and require hygienic measures to alleviate these risks.

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