the catarrhine lineage around 30 million years ago. The duplicate genes form an array on the X chromosome, with additional duplicate copies of the M gene common in humans. The array is bounded on the upstream side by a so-called locus control region (LCR), the presence of which is critical for the expression of either gene. The spectral difference between the L and M pigments is largely determined by amino acid changes at only three sites (164, 261 and 269, Figure 1).

Red/green colour vision is much more variable in New World primates. Most New World species exhibit a trichromacy that is based on only two opsin genes, an autosomal SWS1 gene as in Old World primates, and a polymorphic X-linked LWS gene with multiple allelic forms that encode pigments with differing λ_{max} values lying between about 535 and 565 nm. Platyrrhines thus lack the routine trichromacy of Old World primates, as male monkeys can combine the SWS1 gene with just one of the different allelic forms of the LWS X-linked gene and are therefore dichromats. In contrast, those females that inherit a different form of the LWS gene from each parent have the bonus of trichromatic vision, because X-inactivation will ensure that only one allele is expressed per cell.

A major exception to this polymorphism-based trichromacy in New World primates is found in the howler monkey. In this species, separate L and M genes are present (Figure 4), and expressed in separate cone populations with trichromacy present in both males and females. The duplication of the LWS gene differs from that in Old World primates and appears to be limited to the howler monkey, as it is not present in two closely related species, the spider monkey and the woolly monkey, which both possess a polymorphic LWS gene.

Trichromatic colour vision in monkeys probably evolved from an ancestral dichromacy present within the arboreal environment of early primates, where the driving force was the ability to distinguish the redness of ripe fruits or reddish young leaves from a green background of foliage of highly variable luminance.

Nevertheless, the complement of just three cone pigments in Old World monkeys may be considered somewhat limited in comparison to the complexity of cone pigments available to many lower vertebrates. The basic tetrachromatic system that evolved very early in vertebrate evolution has been adapted to a great range of photic environments, perhaps reaching its most advanced forms in diurnal birds and shallow water teleosts. In these species, spectral sensitivities range from the ultraviolet to the far red and in the case of some teleost fish, gene duplications have provided a wide palette of spectrally distinct pigments from which to differentially tune their colour vision.

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Gorilla susceptibility to Ebola virus: The cost of sociality

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Since 1994, there have been nine human Ebola-Zaire virus (EBOV) outbreaks in eastern Gabon and northwestern Congo [1–3]. A majority of them originated from the handling of ape carcasses found by local hunters [4]. The impact of Ebola-Zaire virus on great ape density is suspected to be high [2,5,6], but neither the demographic consequences of outbreaks nor the way the virus spreads within an ape population are well known. The large population of western lowland gorillas, *Gorilla gorilla gorilla*, monitored since 2001 at the Lokoué clearing, Odzala-Kokoua National Park, Congo, was affected in 2004, providing us with the opportunity to address both questions using an original statistical approach mixing capture–recapture and epidemiological models. The social structure of gorillas strongly influenced the spread of EBOV. Individuals living in groups appeared to be more susceptible than solitary males, with respective death rates of 97% and 77%. The outbreak lasted for around a year, during which gorilla social units (group or solitaries) got infected either directly from a reservoir or from contaminated individuals.

The swampy clearing of the Lokoué site (0°54.38N, 15°10.55E) is exceptionally attractive for gorillas. During a 17 month study in 2001–2, 377 gorillas, of which 92% lived in groups and 8% were solitary males, were individually identified [7]. The first evidence for the presence of Ebola among Odzala apes was the discovery of an EBOV-positive gorilla carcass

Figure 1. Schema of the epidemiological models.
Model Spillover2 assumes reservoir-to-social unit transmission of EBOV. Model SEIR2 assumes that the virus spread through ape-to-ape transmission.

in June 2003, 60 km southwest of Lokoué [4]. On October 13, 2003, two villagers from Mbandza hunting at an undetermined site inside the park got contaminated and became index cases of an outbreak that killed 29 people in 7 weeks. Between January and June 2004, 6 ape carcasses were found within a 4 km distance from the Lokoué clearing. Considering the epidemiological context, there is little doubt Ebola virus is responsible for this die-off.

The observation of gorillas in the clearing was maintained during and after the outbreak until the end of June 2005. Overall, 109 distinct gorilla social units visiting Lokoué were reliably identified and monitored during a 1360 day period. We developed two open capture–recapture statistical models in which survival of group-living individuals and solitary individuals was constrained by epidemiological models both to estimate EBOV-induced gorilla mortality and to investigate the transmission of the virus. The first one, model Spillover2, assumed that the outbreak originated in multiple transmissions of the virus from the reservoir to social units [2,4], with ape-to-ape transmission occurring only within groups.

The second one, model SEIR2, assumed by contrast that ape-to-ape transmission of EBOV was prominent (Figure 1 and see Supplemental Data published with this article online). Both models adequately fitted the data, without overdispersion (parametric bootstrap, model Spillover2: $\hat{c}$ = 0.99; model SEIR2: $\hat{c}$ = 1.00) (Figure 2A). Comparison of the models did not reveal any clear differences, precluding the rejection of one of them (model Spillover2: AIC = 3465, model SEIR2 : AIC = 3468, see Supplemental Data).

These analyses reveal that the outbreak started in December 2003 (Figure 2B). The mortality peaked in May 2004. Although the epidemic lasted almost one year, 95% of all affected gorillas had disappeared before late July 2004. Overall, 95% of the gorillas died from Ebola (95% confidence interval (CI): 90–97%). Due to intra-group transmission, the death rate was highest among gorillas living in groups (estimate: 97%, CI: 92–98%). Solitary gorillas were at least two times more resistant to infection (model SEIR2: 2.28 times, X_1^2 = 7.59, P = 0.006; model Spillover2: 2.26 times, X_1^2 = 7.41, P = 0.007), although the virus caused a 77% decrease in their number (CI: 62–87%). Intra-group

spread of EBOV was probably rapid since only one partially affected group was observed during the outbreak. All the other groups disappeared as a unit.

Whether model SEIR2, model Spillover2, or a mix of these two models corresponds to the evidence is a key point of the epidemiology of the disease. According to model SEIR2, inter-unit transmission of the virus would have been possible provided that, at the epidemic peak, the probability for a susceptible unit to get infected, per 10-day period, reached 0.22 (Supplemental data). This low value is realistic, revealing that this model cannot be disregarded, contrary to what is usually stated [2,4]. The contamination of social units could have occurred during dyadic encounters, for example in the vicinity of fruit trees [8] or forest clearings [9], or during contact with infected carcasses. Alternatively, the Lokoué outbreak could also have been driven by a massive spillover from the reservoir host, provided that this phenomenon lasted around 10 months (estimate: 322 days, CI: 130–539 days). This duration exceeds that of dry seasons previously proposed to promote reservoir-to-ape transmission (Figure 2B) [2,4].

These results provide new insights into the epidemiology of a still largely unknown disease. In an evolutionary perspective, this study provides direct evidence that, in hominoids other than humans, group individuals face a higher disease risk. This cost has probably been an important constraint to sociality evolution in early humans [10]. In a conservation perspective, the demographic impact of Ebola virus is dramatically enhanced since it disproportionately affects females and young individuals, which are essential for population recovery (Figure 2C,D). Censuses conducted in 1994–5 revealed that Odzala-Kokoua National Park gorilla density was the highest ever recorded, averaging 5.4 ind/km² [11]. Preliminary surveys we conducted show that EBOV may have affected this population heterogeneously, with some large areas being now almost devoid

Figure 2. Impact of the Lokoué Ebola outbreak on gorillas.

(A) Cumulative survival of gorillas during the study, corresponding to the probability that a gorilla alive in October 2001 is still alive at a given date. Dots are placed according to estimations performed independently for each of 135 10-day intervals (red dots, group-living individuals (Gr); blue dots, solitary males (Sol); sp2: Spillover2). The slow decrease observed before 2004 is due to normal, non-epidemic mortality or definitive emigration. The strong decrease in 2004 corresponds to the outbreak. The solid lines are placed according to the epidemiological models. (B) Instantaneous survival rates, per 10-day period, predicted by the epidemiological models. The epidemic lasted around one year, but comparison with Figure 2A shows that almost all affected gorillas disappeared during the first half of this period. Wet seasons are shown in green. (C) Number of adult males (M), adult females (F) and immatures (I) identified during 30-day periods. Continuous data collection started 2 months before the outbreak and ended 7 months after its end. Note the sex ratio reversal. (D) Number of adult males, adult females and immatures identified during 150 observation days before and after the outbreak.

of gorillas and others seeming intact. Thousands of gorillas have probably disappeared. As the impact of EBOV on apes is still difficult to control, reinforced protection of gorillas and chimpanzees is required throughout their range, especially against poaching and logging, the two major additional threats to these species [6].

Supplemental data
Supplemental data including experimental procedures are available at http://www.current-biology.com/cgi/content/full/16/13/R489/DC1/

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