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Simian retroviruses in African apes

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Abstract

It is now well established that simian immunodeficiency viruses (SIVs) from chimpanzees (SIVcpz) and gorillas (SIVgor) from west Central Africa are at the origin of HIV-1/AIDS. Apes are also infected with other retroviruses, notably simian T-cell lymphotropic viruses (STLVs) and simian foamy viruses (SFVs), that can be transmitted to humans. We discuss the actual knowledge on SIV, STLV and SFV infections in chimpanzees, gorillas, and bonobos. We especially elaborate on how the recent development of non-invasive methods has allowed us to identify the reservoirs of the HIV-1 ancestors in chimpanzees and gorillas, and increased our knowledge of the natural history of SIV infections in chimpanzees. Multiple cross-species events with retroviruses from apes to humans have occurred, but only one transmission of SIVcpz from chimpanzees in south-eastern Cameroon spread worldwide, and is responsible for the actual HIV pandemic. Frequent SFV transmissions have been recently reported, but no human-to-human transmission has been documented yet. Because humans are still in contact with apes, identification of pathogens in wild ape populations can signal which pathogens may be cause risk for humans, and allow the development of serological and molecular assays with which to detect transmissions to humans. Finally, non-invasive sampling also allows the study of the impact of retroviruses and other pathogens on the health and survival of endangered species such as chimpanzees, gorillas, and bonobos.

Keywords: Africa, bonobo, chimpanzee, gorilla, SFV, SIV, STLV
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Introduction

HIV/AIDS is the most devastating infectious disease to have emerged in the 20th century. Like the majority of emerging infectious disease events, HIV has a zoonotic origin in wildlife [1]. It is now well established that simian immunodeficiency viruses (SIVs) from chimpanzees and gorillas from west Central Africa are at the origin of HIV-1/AIDS [2]. Apes are also infected with other retroviruses, notably simian T-cell lymphotropic viruses (STLVs) and simian foamy viruses (SFV). Here, we review the current knowledge on retroviral infections in African apes.

SIVs

SIV infection in chimpanzees

According to mitochondrial DNA sequences, chimpanzees can be divided into four distinct subspecies ([3]) that are also geographically separated across western and equatorial Africa (Fig. 1). The first SIV strains from chimpanzees (SIVcpz) were isolated from two captive wild-born *Pan troglodytes troglodytes* chimpanzees in Gabon almost 25 years ago [4]. Sequence and phylogenetic analysis showed that SIVcpz was closely related to HIV-1, and cross-species transmission of SIVs from chimpanzees was suspected to be at the origin of the HIV-1 epidemic [5]. In order to better define the origin of HIV-1, studies to further document SIV prevalence and genetic diversity were initially performed on blood samples drawn from captive animals. These studies revealed low prevalences, and resulted in the isolation of only a handful of SIVcpz strains. However, phylogenetic analysis of the few available SIVcpz strains suggested two distinct SIVcpz lineages, according to the host subspecies: SIVcpz*Ptt* from central chimpanzees (*P. t. troglodytes*) and SIVcpz*Pts* from eastern chimpanzees (*P. t. schweinfurthii*) [6–9]. The majority of these chimpanzees were captured as infants, so the results for prevalence and genetic diversity do not reflect the situation

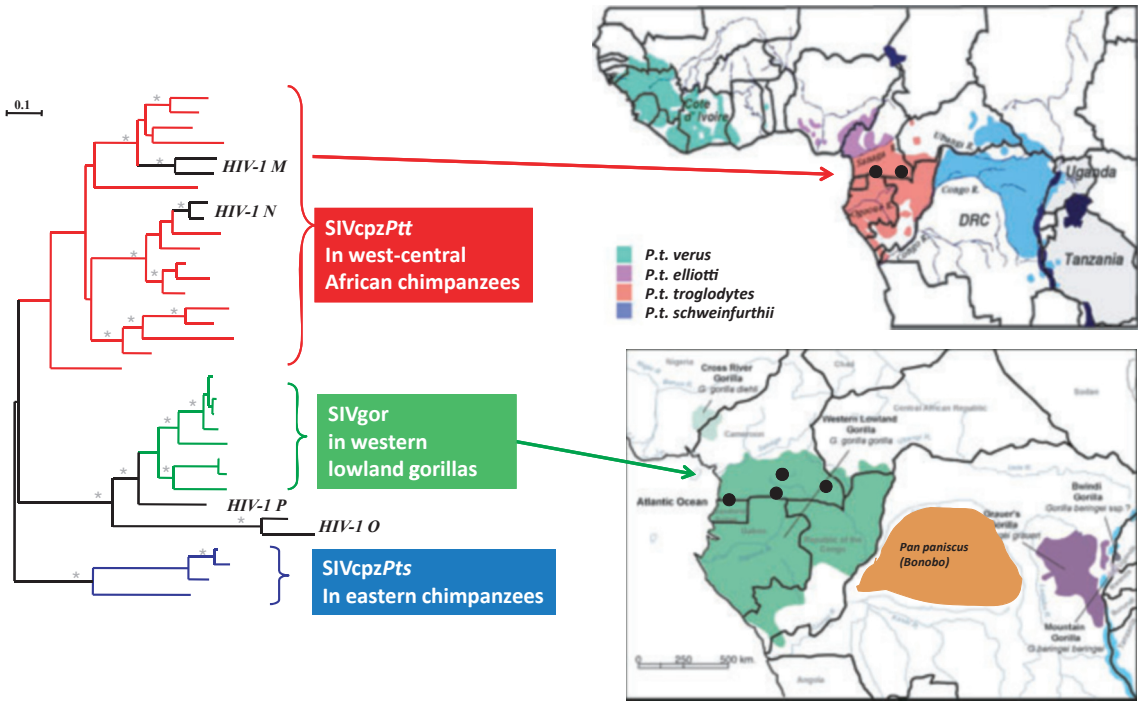

FIG. 1. HIV-1 is derived from simian immunodeficiency viruses (SIVs) circulating in chimpanzees and/or gorillas from west Central Africa. Evolutionary relationships of SIVcpz*Pts* infecting eastern chimpanzees (*Pan troglodytes schweinfurthii*) are in blue, those of SIVcpz*Ptt* infecting central chimpanzees (*Pan troglodytes troglodytes*) are in red, those of SIVgor infecting western lowland gorillas (*Gorilla gorilla gorilla*) are in green, and those of HIV-1 group M, N, O and P strains in humans are in black, based on maximum likelihood phylogenetic analysis of partial Env (gp41) sequences. Horizontal branch lengths are drawn to scale (the scale bar indicates 0.1 substitutions per site), and bootstrap values >80% are represented by grey asterisks. The geographical range of the four chimpanzee subspecies are shown in the upper right panel. The geographical ranges of gorilla species and bonobos are shown in the lower right panel. The arrows between the phylogenetic tree and the map indicate the ape reservoirs with the ancestors of or most closely related strains to the different HIV-1 groups. DRC, Democratic Republic of Congo.

in wild populations. The major issues in studying SIVcpz infection in wild chimpanzees are their endangered status and the fact that they live in isolated forest regions. The development of non-invasive methods for the detection and characterization of SIVcpz in faecal and urine samples boosted the search for new SIVcpz strains in wild ape populations in the vast tropical forest of Central Africa [10,11]. Moreover, faecal samples can also be used to confirm the species and subspecies of the host, and individuals and their sex can be identified by analysis of DNA short tandem repeats (Fig. 2).

To date, more than 7000 faecal samples from nearly 90 field sites across the entire chimpanzee home range have been analysed. These studies revealed SIVcpz infection at multiple sites across Central Africa, but SIVcpz seroprevalence rates varied widely, from 30–50% in some chimpanzee communities from west Central and East Africa, to rare cases or absence of infection in others [2,12,13] (Li *et al.*, 17th Conference on Retroviruses and Opportunistic Infections, 2010, Abstract 440). These studies also confirmed subspecies-specific clustering, and showed that SIVcpz*Ptt* strains from *P. t. troglodytes* are significantly more closely related to HIV-1 strains from humans. Moreover, the ancestors of HIV-1 M and HIV-1 N could be traced to distinct chimpanzee communities in south-eastern and south-central Cameroon (Fig. 1) [2]. Despite testing of numerous samples of wild and captive *P. t. verus*, largely exported from West Africa to primate centres in Europe and the USA, no SIV infection has been detected in this subspecies [14,15]. Also, all *P. t. ellioti* samples were SIV-negative [2,12].

These data suggest that chimpanzees acquired SIVcpz after the evolutionary divergence and geographical separation of the West African and Nigerian subspecies from the Central and East African subspecies [16]. Detailed analysis of the SIVcpz genome revealed that SIVcpz is a recombinant virus, resulting from cross-species transmission events followed by recombination between SIVrcm from red-capped mangabeys and a strain from the SIVgsn/mus/mon lineage infecting greater-spot-nosed, mustached and mona monkeys [17]. Chimpanzees were most probably infected with these viruses

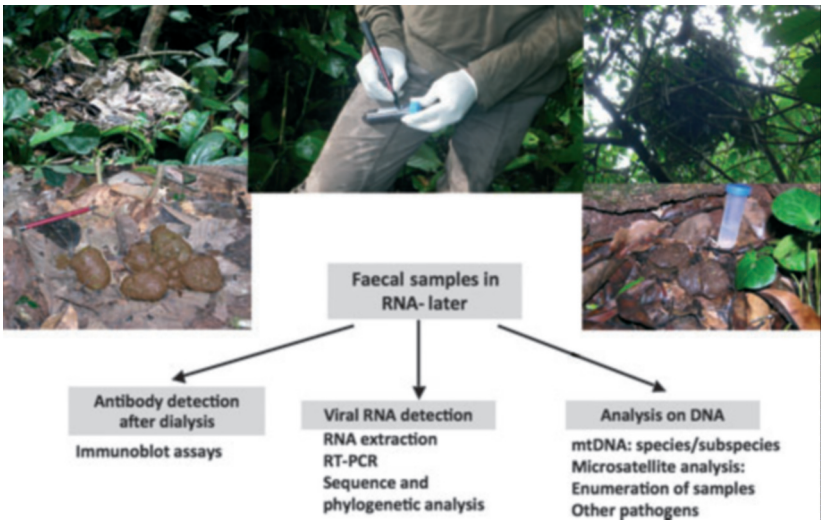

FIG. 2. Non-invasive methods for the detection of retroviral infections in faeces. Collection of faecal samples at nesting sites of gorillas (left upper panel) and chimpanzees (right upper panel) and subsequent storage in RNA later (middle). Photographs by Sabrina Locatelli and Florian Liegeois. mtDNA, mitochondrial DNA.

while hunting and consuming monkeys [18]. The current range of the central chimpanzee overlaps with the ranges of red-capped mangabeys and the various *Cercopithecus* species infected with SIVgsn/mus/mon. The recombination therefore most likely occurred in a chimpanzee from west Central Africa, and this new virus became the common ancestor of today's SIVcpz lineages. Despite the fact that chimpanzees can be frequently exposed to infected monkeys, no other SIV has been observed among them [19,20].

Pathogenicity of SIVcpz in chimpanzees

Initially, like other SIVs in their natural hosts, SIVcpz was also thought to be non-pathogenic for chimpanzees. This has been challenged recently by a study initiated in 2001 on two habituated populations of chimpanzees (*P. t. schweinfurthii*) at Gombe National Park in Tanzania [21]. The SIV status was assessed by analysing faecal samples at regular time intervals for more than 10 years. SIV infection was associated with a 10-fold to a 16-fold increase in age-corrected risk of death. Fertility was significantly reduced in SIV-positive females, in terms of both birth rate and survival of offspring. Immunohistochemistry and *in situ* hybridization of post-mortem spleen and lymph node samples showed lower $CD4^+$ T-cell counts in SIV-positive than in SIV-negative individuals. $CD4^+$ T-cell counts and tissue samples strongly resembled the histopathology of human end-stage AIDS patients. SIVcpz could thus be causing the decline of chimpanzee communities, and could also explain the uneven distribution of SIVcpz throughout Central Africa [22]. No data are currently available on wild *P. t. troglodytes* chimpanzees. However, we recently described a progressive SIV infection similar to HIV infection in humans in a naturally SIV-infected *P. t. troglodytes* chimpanzee. This animal had low CD4 counts, severe thrombocytopenia, weight loss, and unusually frequent periods of infection with diverse pathogens [23]. Overall, SIVcpz most likely has a negative impact on the health, reproduction and survival of both chimpanzee subspecies in Central Africa.

SIV infection in gorillas

Gorillas are classified into two species, the western *Gorilla gorilla* and eastern *Gorilla beringei*, which in turn are subdivided into four subspecies (*G. g. gorilla*, *G. g. diehli*, *G. b. beringei*, and *G. b. graueri*) [24]. As shown in Fig. 1, the range of gorillas overlaps with that of chimpanzees, especially in west Central Africa. Analysis of about 200 faecal samples from southern Cameroon showed that gorillas are also infected with an SIV, termed SIVgor [25]. Surprisingly, this lineage fell within the radiation of SIVcpz, and was closely related to HIV-1 groups P and O (Fig. 1). The phylogenetic relationships between SIVcpz, SIVgor and HIV-1 show that chimpanzees were the original reservoir of SIVs found in gorillas and humans, and that *P. t. troglodytes* apes were most likely the original source of SIVgor [25–27]. In addition, an ancestral SIVcpz*Ptt* lineage from which SIVgor and HIV-1 group O viruses are derived has been identified in the form of mosaic *pol* fragments in present-day SIVcpz*Ptt* recombinants [26].

The methods of transmission and the exact origin of SIVgor infection in gorillas are not yet resolved. Because of the extensive overlap in habitat and diet [28–31], direct encounters between gorillas and chimpanzees seem inevitable, but they have rarely been observed, and have been described as primarily non-aggressive [32]. However, it cannot be excluded that physical encounters between chimpanzees and gorillas, possibly involving biting or other forms of aggression, occurred in the past. Moreover, sharing the same habitat could also lead to indirect contact with the virus of the other species (e.g. disease outbreaks such as anthrax, Ebola, etc.) [33].

Subsequent screening of faecal gorilla samples from 30 field sites, mainly from western lowland gorillas (*G. g. gorilla*) in southern Cameroon and the Central African Republic, but also from eastern gorillas (*G. b. graueri*), revealed an overall seroprevalence of SIVgor of 1.6%. SIVgor appears to be much less common in gorillas than SIVcpz is in chimpanzees; SIVgor was only documented in western gorillas, and at only four field sites [34]. At two of these sites, however, the SIVgor seroprevalence was close to 5%, indicating efficient virus spread within and between different communities. It remains possible that SIVgor is even more prevalent in other parts of the home range of western lowland gorillas. Similarly, as observed for SIVcpz, SIVgor strains cluster according to their collection site of origin.

As gorillas may have acquired SIV only recently from chimpanzees, it is possible that SIVgor is also pathogenic for its host. Actually, there are no captive gorillas naturally infected with SIVgor and no habituated gorilla groups infected with SIVgor. The impact of such infection on the survival of gorillas can only be determined by a non-invasive approach in the wild.

SIVs from chimpanzee and gorillas, and the origin of HIV-1

It is now clear that central chimpanzees (*P. t. troglodytes*) are the reservoirs for the pandemic HIV-1 group M strain and also for HIV-1 N [2]. HIV-1 P is most likely of gorilla origin, but the reservoirs of the direct ancestors of HIV-1 O have not been identified yet; moreover the current data do not allow differentiation between a chimpanzee or a gorilla reservoir [25–27,34]. More studies are therefore still needed to identify the direct ancestors of HIV-1 O and P in gorillas and/or chimpanzees. The cross-species transmissions giving rise to HIV-1 most likely occurred in western equatorial Africa, within the geographical ranges of central chimpanzees and western gorillas. This coincides with the geographical area of HIV-1 N, O and P infections, which are also mainly restricted to Cameroon [35]. Only, HIV-1 M has spread across Africa and all of the other continents, but the reservoir of the ancestors of HIV-1 M does not coincide with the epicentre of the HIV-1 epidemic [2,36]. The latter has been identified around Kinshasa, the capital city of the Democratic Republic of Congo (DRC), at a distance of almost 1000 km. HIV-1 M (subtypes A and D) was already circulating in humans in Kinshasa 20 years before the AIDS epidemic was recognized in the USA; HIV-1 was identified in a serum sample from 1959 and a biopsy sample from 1960 [37,38]. Molecular clock analyses estimated the date of the most recent common ancestor of HIV-1 M to be around 1908 (1884–1924), and the date for the origin of the HIV-1 O radiation to be around 1920 (1890–1940) [39–41]. HIV-1 N seems to be a more recent cross-species transmission, the date of the most recent common ancestor being estimated to be around 1963 (1948–1977) [39], and, finally, HIV-1 P was only discovered in 2009 [27]. A combination of several factors (viral, host, socio-economic, demographic, etc.) are thus most likely to have been involved in the subsequent efficient spread of HIV-1 M [42,43].

STLV in Apes

Human T-lymphotropic viruses (HTLVs) and STLVs are collectively called primate T-lymphotrophic viruses. In contrast to HIV, the majority of HTLV infections remain asymptomatic, but approximately 5% of them are associated with severe diseases such as adult T-cell leukaemia/lymphoma or an inflammatory disease of the central nervous system called HTLV-1-associated myelopathy/tropical spastic paraparesis [44,45]. Simian counterparts, STLV-1 to STLV-3, have been described for HTLV-1 to HTLV-3 in humans [46]. No simian analogue of the recently discovered HTLV-4 has been identified yet, and no human analogue has been reported to date for the tentatively identified STLV-5 in a macaque species from Asia [47]. STLV has been documented in more than 30 non-human primate (NHP) species from sub-Saharan Africa and Asia, but few data are available on STLV infection in wild apes [43]. Phylogenetic analysis shows that primate T-lymphotrophic viruss cluster by geography, rather than by host species, suggesting multiple cross-species transmission events between NHPs, and also from NHPs to humans [47–49].

STLV-1 belonging to the African subtype B has been documented in captive but wild-caught chimpanzees (*P. t. ellioti* and *P. t. troglodytes*) and gorillas from west Central Africa [50]. Approximately 70% of wild chimpanzees in Ivory Coast are infected with STLV-1, and, in addition to subtype B, a variant closely related to STLVs observed in the western red colobus was identified, suggesting cross-species transmission by predation [51]. STLV-2 has only been documented in bonobos, an ape species endemic to DRC [52]. The initial strains were isolated in captive animals, and STLV-2 DNA was also recently identified in a faecal sample from a wild bonobo in DRC [53]. No data are currently available on STLV pathogenicity in wild apes; however, STLV-1 has occasionally been associated with lymphoma or chronic fatal disease in captive gorillas [54,55].

Because many other monkey species are also infected with STLV strains closely related to those that circulate among chimpanzees and gorillas, it is not easy to determine which primate species is at the origin of today's HTLV-1 B subtypes in humans.

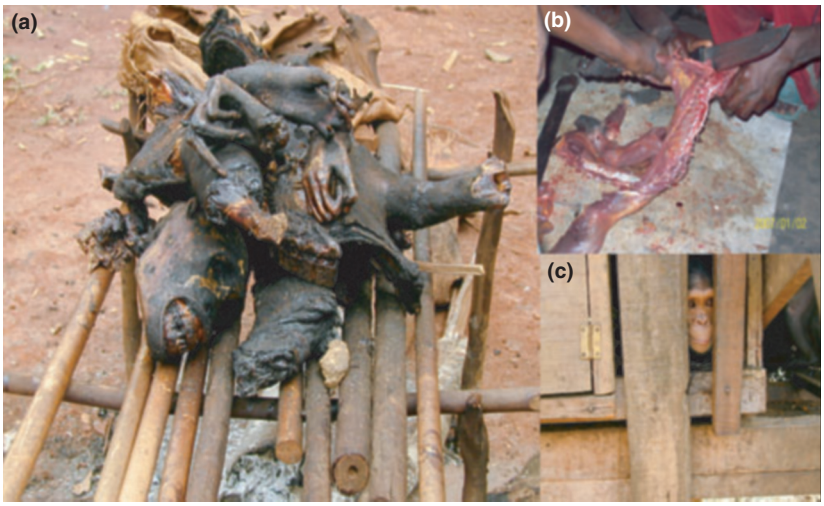

FIG. 3. Human exposure to retroviruses. Pictures taken between 2001 and 2009 in Cameroon and Democratic Republic of Congo, illustrating bushmeat (smoked (a), butchering (b)) and pet chimpanzee as bushmeat byproducts (c). Photographs by Bernadette Abela (a, c), and Steve Ahuka-Mundeke (b).

SFVs

SFVs have been identified with a high prevalence in a wide variety of primates, including prosimians, and New and Old World NHPs; each species has been shown to harbour a unique (species-specific) SFV strain, and no disease is associated with SFV infection in their natural hosts [56]. SFVs have co-evolved with their NHP hosts for more than 30 million years [56]. SFVs have been described in captive apes, and a recent serological and molecular survey on faecal samples showed a high SFV infection rate (44–100%) in wild chimpanzees from West, Central and East Africa [57–59]. Remarkably, SFVcpz strains clustered according to their subspecies of origin [59]. No large-scale studies have yet been performed on wild gorillas, but species-specific SFV infection has been identified in wild-caught captive gorillas in Cameroon [60].

There is no human foamy virus, but SFV infections have been reported in persons occupationally exposed to NHPs and in a few hunters in Central Africa, with no consequences for human health [61]. Efficient transmission and high levels of cross-species transmission of SFVs to humans after ape bites, followed by viral persistence, have been reported in Gabon and Cameroon [62,63]. No SFV epidemic has been documented yet, and the apparent lack of human-to-human SFV transmission represents an informative marker of contact between human and NHPs and of transmission of simian retroviruses to the human population.

Conclusion

Although the conditions and circumstances determining SIV, STLV and SFV transmission from NHPs to humans are not completely elucidated, exposure to blood or other secretions of infected animals, through hunting and butchering of bushmeat, or through bites and scratches from pet NHPs, represent the most plausible source for human infection (Fig. 3). Direct evidence for such events has been reported recently for SFV in primate hunters in independent studies in Cameroon and Gabon. The recent development of non-invasive methods for the detection of retroviral infections has allowed us to define the origin of HIV-1, and has significantly increased our knowledge of the natural history of SIV infections in chimpanzees. However, our knowledge of retroviral infections in wild apes is still incomplete with regard to geographical areas and certain species. Currently, almost no data are available for bonobos, or eastern lowland and mountain gorillas. Importantly, despite their highly endangered status, apes continue to be hunted, and remain a potential source of human infections. SIVs from apes have crossed the species barrier to humans on at least four occasions in west Central Africa. Given the recent discovery of the HIV-1 group P lineage, it would not be surprising if additional cases of SIVcpz or SIVgor cross-species transmission had occurred.

Finally, non-invasive sampling also allows large-scale studies on wild apes to prevent or trace the origin of other epidemics, e.g. the origin of human *Plasmodium falciparum* in gorillas in west Central Africa [64]. Non-invasive studies also allow studies on the impact of retroviruses and other pathogens on the health and survival of endangered species.

Transparency Declaration

The authors declare they have no conflicts of interest.

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