

## Short Communication

# Origin of HIV Type 1 in Colonial French Equatorial Africa?

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### ABSTRACT

Sociocultural factors during the postcolonial period have been implicated as paramount in generating conditions that promoted both the origin and subsequent epidemic spread of HIV-1 in Africa. We suggest, however, that the origin of the disease may lie in the interaction between colonial practices (e.g., labor camps, nonsterile vaccination campaigns) and traditional bushmeat hunting in French Equatorial Africa. Both the epidemiology of HIV-2 and the colonial history of West Africa appear more complex, but similar conditions existed there and may have contributed to the origin of HIV-2. Focusing the search for the origins of HIV-1 and HIV-2 on this earlier time period may contribute to understanding the evolution of the HIV viruses and the dynamics of emerging diseases.

**H**UMAN IMMUNODEFICIENCY VIRUS TYPE 1 (HIV-1) seems to have originated in at least three zoonotic transmissions of SIVcpz from chimpanzees (*Pan troglodytes troglodytes*) to humans,<sup>1</sup> placing the origin of the disease in Central Africa (Cameroon, Gabon, R.P. Congo, lower Central African Republic, and Río Muni [Equatorial Guinea]). This most probably occurred in the course of hunting and butchering apes for food. Because such hunting is traditional, an obvious question is, “Why now?”

The earliest direct evidence of HIV-1 comes from a blood sample collected in Kinshasa in 1959,<sup>2</sup> and this has led some to concentrate attention on post-World War II (WWII) social changes connected with the end of European colonial power in the region (basically, urbanization combined with “breakdown of colonial control” leading to increased opportunities for transmission of sexually transmitted diseases [STDs]). However, while the post-WWII period did bring many changes to Central Africa that have probably played a role in the origin of the current epidemic, the origin of the disease must lie some years earlier. We suggest that the origin of the disease may have occurred prior to WWII, during the period of French colonial administration of French Equatorial Africa (see Table 1 for chronology). What factors might have contributed to the initial zoonosis and to its subsequent spread during this period, in the absence of evidence of endemic HIV-1?

It seems likely that transmission of SIVcpz to humans has taken place regularly at some low frequency for thousands of years; previously either strains were nonpathogenic in humans (and lost owing to random factors), nontransmissible between humans, or infected individuals died without spreading the virus (owing either to low population density or to [hypothetical] traditional practices limiting sexual promiscuity). We are thus looking for factors associated with the colonial period that would do the following:

1. Increase exposure risk (i.e., increased reliance on chimpanzee bushmeat and/or changes in hunting or butchery practices resulting in increased risk of blood–blood contact)
2. Increase the probability of virus transmission (via sexual or blood–blood routes)
3. Increase the probability of adaptation of the virus to humans, increasing the probability that it would be transmitted

*1. Exposure risk.* Colonial authorities conscripted people for work as porters and as forced labor on railroads and other infrastructure projects; during WWI, they also required villagers to harvest large quantities of rubber.<sup>4–6</sup> All these led to (a) some people fleeing villages to live in the forest, and (b) those conscripted having little time, energy, or opportunity to devote to agriculture. It may be conjectured that both would have led to

TABLE 1. CHRONOLOGY<sup>a</sup>

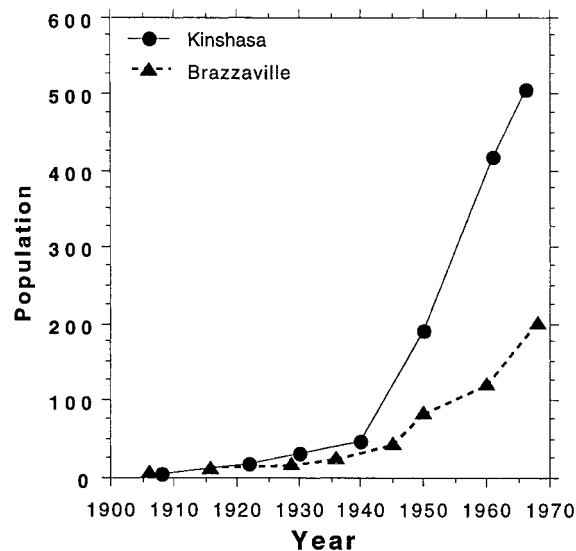
	<i>Event</i>
1892	Start of steamboat service upriver from Kinshasa/Brazzaville to Kisangani. Riverboat traffic regular and significant from this point onward, connecting areas and people who had previously had little contact
1893–1912	Smallpox vaccination campaigns prior to development of dry vaccine in 1914. The cowpox virus used in vaccinations was difficult to transport into the interior, and so arm–arm vaccinations were used. More than 78,000 people in present-day Gabon, Republique Populaire du Congo (Brazzaville), Central African Republic, and Chad were vaccinated during this period, with at least 35,000 of these being in interior regions where arm–arm methods were most likely to be used. <sup>b</sup>
Late 1890s–1908	Period of intense rubber demand. In the Belgian Congo (now Democratic Republic of the Congo), this demand was associated with draconian quota system resulting in social disruption and dislocation. From 1908 to the 1920s, less severe methods were used to force collection. <sup>c</sup>
1920–1935	French campaign against sleeping sickness—mobile clinics. Procedure involved (1) examination of blood and lymph to identify carriers, followed by (2) treatment with intravenous drugs. This procedure was based on the work of Dr. Edward Jamot, who treated >89,000 people in Ubangi Shari (now Central African Republic) in 1916 with six syringes and two hand centrifuges (number of needles and methods of sterilization not known). From 60,000 to 600,000 people treated per year.
1921–1930+	Construction of railroad connecting Brazzaville with the coast (estimated 20,000 deaths), and other infrastructure projects requiring large labor pools. Labor was generally forced and involved concentration of workers from wide (and nonlocal) areas into camps. The sex ratio in the camps was highly skewed (498 women and 6173 men in one camp, 1929) <sup>b</sup>
1960	Independence

<sup>a</sup>Source: Ref. 3.<sup>b</sup>See Ref. 5.<sup>c</sup>See Ref. 6.

significant increases in reliance on bushmeat. In addition, increasing access to guns would have played a role in obtaining large animals such as chimpanzees.

**2. Viral transmission.** There was a massive influx of people into the major cities after WWII, constituting "... movement of previously isolated people into the newly expanding cities"<sup>7</sup> (e.g., the population of Kinshasa increased almost 10-fold, from 49,000 in 1940 to 420,000 in 1961<sup>3</sup>). Whatever role this influx may have had in the development of the epidemic, it should be kept in mind that the cities were not "newly" expanding; the population of Kinshasa (and Brazzaville) also increased about 10-fold between 1905 and 1940 (Fig. 1). This earlier period of urbanization would have created conditions favorable to the initial establishment of the disease.

In addition, the social turmoil associated with forced resettlements and labor undoubtedly disrupted traditional sexual practices and networks. More directly, some of the labor camps (of thousands of men) encouraged the presence of women for "recreational" purposes.<sup>5</sup> Finally, massive vaccination campaigns were carried out with limited resources (e.g., six syringes used to screen and treat nearly 90,000 people for sleeping sickness in 1917–1919).<sup>8</sup> An unknown proportion of nearly 100,000 smallpox vaccinations prior to 1914 employed arm–arm direct inoculation with material from pox vesicles.<sup>5,9</sup> (con-



**FIG. 1.** Estimated population of Kinshasa and Brazzaville, 1906–1968, in thousands.

taining a high concentration of lymphocytes, the primary target of HIV-1).

3. *Viral adaptation*. Arm–arm inoculation may have resulted in passing of virus through a series of hosts over a relatively short period of time, a process that can select for more virulent pathogen strains.<sup>10</sup> Without knowing more about the details of the arm–arm campaigns (how many successive “donors” were used in the course of a campaign? how many people vaccinated?) the importance of this possible serial passing cannot be evaluated.

It is not clear whether understanding the origins of HIV-1 will make a difference in treating AIDS, and the preceding scenario presents only plausible risk factors—it is not clear how or if any of them can be confirmed as playing a role in the origin of AIDS. However, to the extent that it is useful/interesting to understand the origin of HIV-1, if this scenario is correct it suggests that investigation should be concentrated in French archives pertaining to French Equatorial Africa between 1890 and 1930, with special reference to the following:

1. The details of techniques used during medical campaigns against smallpox and sleeping sickness, particularly those involving Moyen Congo and Gabon, heart of *P. t. troglodytes* range)
2. The prevalence of bushmeat as an alternative food source for porters and laborers (analogous to modern reliance on bushmeat by logging companies<sup>11</sup>)
3. Changes in the availability of guns (including locally made muzzle-loaders) and the role such changes may have played in making apes easier to hunt. While there is some literature on the impact of firearms on West African wars and slaving,<sup>12</sup> we have found nothing on their early history in bushmeat hunting

We have not dealt in detail with conditions in West Africa that might have been associated with the origins of HIV-2 from SIVsm carried by sooty mangabeys (reviewed in Ref. 13). Unlike the comparatively sudden impact of colonialism on small-scale societies in French Equatorial Africa, in the west European contact began in the fifteenth century and interacted in a complex way with existing kingdoms through some 400 years of trade and exploitation—prominently including the slave trade.<sup>14</sup> In addition, in West Africa (but apparently not in French Equatorial Africa) smallpox inoculation (variolation) was either indigenous or introduced early.<sup>15</sup> These differences make it more difficult to point to a specific time and place as the likely site of the origin of the disease.

It is, however, worth noting that while the highest reported seroprevalence of HIV-2 is in Guinea-Bissau, the greatest diversity of subtypes is in Sierra Leone.<sup>13,16–18</sup> These countries straddle Guinea, a country for which there seems to be relatively little information published about HIV seroprevalence. Northwestern Guinea includes the heart of the nineteenth century state of Fouta Djallon, which played a major role in the slave trade and did not fall to the French until 1895, not long after the rubber boom began in the area.<sup>14</sup> Entire districts were depopulated, and then repopulated, as Fouta fell and antislavery laws were slowly enforced over the next decade. Because

it took time to reclear fields and rebuild abandoned villages, many of these returning people were forced to live by hunting bushmeat. More than 50,000 porters were used per year to headload rubber from the interior to the coast by 1906; a railroad was under construction from 1899 through 1913, mostly with slave labor. The “[d]iversion of labor was so great that food was in short supply and had to be imported” and if laborers sickened they received no rations, having to scavenge in the forests.<sup>14</sup> Finally, the French army of the Sudan operating in the late nineteenth century (mostly in what is now northeastern Guinea and western Mali) made extensive use of slaves for both labor and sex; officers regularly got their choice of slave women and promiscuity was high.<sup>14</sup>

In short, similar risk factors were present. They peaked slightly earlier (the turmoil was greatest roughly in 1880–1910) and less sharply (e.g., indigenous variolation was practiced for hundreds of years), but they were present. While spread throughout the region, the combination of clashing states and intensive forced labor was slightly greater in the region of Fouta Djallon and neighboring Wasulu, both located primarily within the modern nation of Guinea.

The widespread occurrence of these epidemiologically relevant factors prior to WWII suggests that HIV-1, and possibly HIV-2, emerged as infectious human pathogens earlier than generally believed, a conclusion consistent with recent and ongoing genetic analyses.<sup>19</sup>

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## REFERENCES

1. Gao F, Bailes E, Robertson DL, Chen Y, Rodenburg CM, Michael SF, Cummins LB, Arthur LO, Peeters M, Shaw GM, Sharp PM, and Hahn BH: Origin of HIV-1 in the chimpanzee *Pan troglodytes troglodytes*. *Nature* (London) 1999;397:436–441.
2. Zhu TF, Korber BT, Nahmias AJ, Hooper E, Sharp PM, and Ho DD: An African HIV-1 sequence from 1959 and implications for the origin of the epidemic. *Nature* (London) 1998;391:594–597.
3. Hance WA: *Population, Migration, and Urbanization in Africa*. Columbia University Press, New York, 1970.
4. Hartwig GW and Patterson KD (eds.): *Disease in African History*. Duke University Press, Durham, North Carolina, 1978.
5. Headrick R: *Colonialism, Health and Illness in French Equatorial Africa, 1885–1935*. African Studies Association Press, Atlanta, Georgia, 1994.
6. Hochschild A: *King Leopold's Ghost: A Story of Greed, Terror, and Heroism in Colonial Africa*. Houghton Mifflin, Boston, 1998.
7. Essex ME: Origin of acquired immunodeficiency syndrome. In: *AIDS: Etiology, Diagnosis, Treatment and Prevention*, 4th Ed. (Devita VT Jr, Hellman S, and Rosenberg SA, eds.). Lippincott-Raven, Philadelphia, 1997, pp. 3–14.
8. McKelvey JJ: *Man against Tsetse: Struggle for Africa*. Cornell University Press, Ithaca, New York, 1973.
9. Copeman SM: *Vaccination: Its Natural History and Pathology*. Macmillan, London, 1899.
10. Ebert D: Experimental evolution of parasites. *Science* 1998;282:1432–1435.

11. Robinson JG, Redford KH, and Bennett EL: Wildlife harvest in logged tropical forests. *Science* 1999;284:595–596.
12. Inikori JE: The import of firearms into West Africa 1750–1807: A quantitative analysis. *J Afr Hist* 1977;18:339–368.
13. Schim van der Loeff MF and Aaby P: Towards a better understanding of the epidemiology of HIV-2. *AIDS* 1999;13(Suppl. A):S69–S84.
14. Klein MA: *Slavery and Colonial Rule in French West Africa*. Cambridge University Press, Cambridge, 1998.
15. Herbert EW: Smallpox inoculation in Africa. *J Afr Hist* 1975; 16:539–559.
16. De Cock KM, Brun-Vézinet F, and Soro B: HIV-1 and HIV-2 infections and AIDS in West Africa. *AIDS* 1991;5(Suppl. 1):S21–S28.
17. Chen Z, Luckay A, Sodora DL, Telfer P, Reed P, Gettie A, Kanu JM, Sadek RF, Yee J, Ho DD, Zhang L, and Marx PA: Human immunodeficiency virus type 2 (HIV-2) seroprevalence and characterization of a distinct HIV-2 genetic subtype from the natural range of simian immunodeficiency virus-infected sooty mangabeys. *J Virol* 1997;71:3953–3960.
18. Gao F, Yue L, Robertson DL, Hill SC, Hui H, Biggar RJ, Neequaye AE, Whelan TM, Ho DD, Shaw GM, Sharp PM, and Hahn BH: Genetic diversity of human immunodeficiency virus type 2: Evidence for distinct sequence subtypes with differences in virus biology. *J Virol* 1994;68:7433–7447.
19. Korber B, Theiler J, and Wolinsky S: Limitations of a molecular clock applied to considerations of the origin of HIV-1. *Science* 1998;280:1868–1871.

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