

Colonialism and the First HIV Epidemic

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Abstract

Since the HIV-1 pandemic began, 79 million people have been infected, and 36 million have died (UNAIDS, 2021). While the origins of the HIV-1 pandemic are not widely known, research suggests that HIV originated in Kinshasa—the capital city of the former Belgian-Congo colony (Faria et al., 2014; Pépin, 2013). HIV circulated within Africa for decades before its global spread (Faria et al., 2014). The question then rises, how did a virus originating from Africa spread globally? Evidence points to colonialism (Faria et al., 2014; Giles-Vernick et al., 2013; Pépin, 2013). Colonial influences on urbanization, social dynamics, and healthcare created conditions that favoured the early spread of HIV (Giles-Vernick et al., 2013; Pépin, 2013). Given the rise of infectious diseases such as COVID-19, addressing the impacts of colonialism and its influence on the spread of HIV is critical. Understanding how infectious diseases emerge can aid in future prevention efforts.

Keywords: HIV epidemic, colonialism

In 1981, Los Angeles reported five cases of pneumocystis carinii pneumonia, a rare lung infection among five healthy gay men (Altman, 1981). Later, a report documented Kaposi Sarcoma, a rare, aggressive cancer among gay men in New York and Los Angeles (Altman, 1981). Healthy men were developing rare diseases, later known as acquired immunodeficiency syndrome (AIDS). Researchers later discovered that the human immunodeficiency virus (HIV) was the cause of AIDS (Faria et al., 2014). These reports marked the start of the HIV/AIDS epidemic. It also marked decades of research and funding into the prevention, treatment, and cure of HIV/AIDS. Since 1981, HIV has infected seventy-nine million people and killed thirty-six million (UNAIDS, 2021). Many refer to the initial cases in New York and Los Angeles as the origins of HIV, but cases were reported decades before HIV arrived in North America (Faria et al., 2014; Sharp & Hahn, 2011). Researchers have traced the origins of HIV to Kinshasa—the capital city of the former Belgian-Congo colony (Faria et al., 2014). Although the epidemiological triangle can help us identify factors that influenced the HIV/AIDS epidemic, what led to these factors? Recent research suggests colonialism in Africa enhanced the spread of HIV infection (Faria et al., 2014; Pépin, 2013; Rupp et al., 2016).

Literature Review

The Epidemiological Triangle

CDC (2021) suggests that the epidemiological triangle model can explain disease emergence. The triangle consists of a host, an agent, and the environment. Environments promote the interaction between the host and agent, resulting in disease. An agent refers to an infectious pathogen that can cause diseases. The host is the organism that can get the disease. The CDC (2021) also mention that biological and individual factors influence how the host responds to an agent. Environmental factors can affect host exposure risk to an agent, including

the physical, biological, and socioeconomic environment. Using this model, we can explain how colonialism contributed to the spread of HIV.

Agent

HIV spreads through contact with bodily fluids such as blood, semen, pre-seminal fluid, vaginal and rectal fluids, and breast milk (HIV.gov, 2019). It is a lentivirus, meaning it has a long incubation period, persists lifelong, and mutates at high rates (Stanford, n.d.). HIV has one of the highest rates of mutations, resulting in large viral diversity, allowing it to evade and infect human immune systems (Andrews & Rowland-Jones, 2017; Stanford, n.d.). Individuals develop AIDS when their immune system weakens and the risk of fatal opportunistic infections increases (Stanford, n.d.). There is no cure for HIV or AIDS, but modern treatment can lead to untransmissible viral loads, enabling individuals to live long and healthy lives (HIV.gov, 2019).

Host

HIV-1 group-M is the dominant epidemic strain found in 95% of infections (Sharp & Hahn, 2011). Using the evolution tree of HIV, researchers discovered that HIV-1 group-M and simian immunodeficiency viruses (SIV) in monkeys are similar (Faria et al., 2014; Sharp & Hahn, 2011). Studies using serum and bodily fluids of Central Africans found that SIV infections are common among bushmeat handlers (Sousa et al., 2017). SIV strains from Pan troglodytes troglodytes chimpanzees (SIVcpzPtt) were the closest to HIV-1 group M (Sharp & Hahn, 2011). SIVcpzPtt originated in southeast Cameroon, where cross-species transmission likely occurred (Sharp & Hahn, 2011). Researchers believe the first HIV epidemic happened in the Democratic Republic of the Congo (DRC)—formerly the Belgian-Congo colony (Faria et al., 2014). The genetic diversity of HIV-1 group-M was many and complex in the Belgian-Congo colony (Sharp & Hahn, 2011). Researchers also sourced the two oldest strains of HIV-1 group-M from

Kinshasa, the current capital city of the DRC (Sharp & Hahn, 2011). For simplicity, HIV-1 group-M will be referred to as HIV throughout this paper.

Humans became the host of HIV when multiple cross-species transmissions from chimpanzees in southeast Cameroon adapted to humans in the late 1800s (Faria et al., 2014; Rupp et al., 2016). Identifying factors influencing human susceptibility to HIV is challenging. Research proposes that genetics, sexually transmitted diseases (STDs), sexual behaviours, and injections increase the risk of HIV in Central Africa (Pépin, 2013; Sousa et al., 2017). These factors, along with economic and socio-political factors, changed the susceptibility to HIV (Rupp et al., 2016).

Environment

Popular theories regarding the origins of Kinshasa's HIV epidemic and environmental factors include the cut-hunter hypothesis, colonial urbanization, and colonial healthcare (Giles-Vernick et al., 2013; Pépin, 2012, 2013; Rupp et al., 2016). The cut-hunter theory explains how humans acquired HIV from chimpanzees infected with SIV (Rupp et al., 2016). Colonial urbanization and healthcare systems highlight how shifts in mobility, sociality, and medical practices contributed to the spread of HIV (Rupp et al., 2016).

The Cut-Hunter Hypothesis. Rupp and colleagues (2016) mention that the cut-hunter hypothesis is the most popular theory of cross-species transmission. It states that the introduction of firearms by European colonizers made hunting easier for Africans. During hunting, an SIV-infected chimpanzee cut or injured a hunter. The hunter then travelled from Cameroon to Kinshasa, and introduced HIV into the population. Researchers conclude that the hunter initiated the HIV spread in Kinshasa. However, Rupp et al. (2016) criticized the cut-hunter hypothesis for its oversimplification of HIV transmission. It does not recognize the ecological, economic, and

sociopolitical factors, nor the existence of SIV pre-colonialization which are critical to understanding the HIV epidemic.

Urbanization and Social Factors. Giles-Vernick and colleagues (2013) state that in the late 1800s and early 1900s, Central Africa experienced urbanization and demographic changes. Kinshasa was a hub for goods exchanges, ideas, and people, leading to its expansion and population growth. During colonization, Belgian-Congo shifted from a resource state to an urban region. Giles-Vernick et al. (2013) mention that in Kinshasa, the development of schools, hospitals, and churches attracted rural migrants; mainly male workers who lived in poor settlements. As it was illegal for single women to live in the city until the mid-1930s, they were smuggled in, resulting in a biased sex ratio. Researchers theorized that prostitution—a lucrative profession for women—emerged due to the gender imbalance and subsequently contributed to the early spread of HIV in Kinshasa (Giles-Vernick et al., 2013; Pépin, 2013). Giles-Vernick et al. (2013) state that some criticized this theory because of colonial ignorance towards African practices. Unlike European prostitution, which involved a high turnover of male partners, early African ‘prostitution’ involved various sexual and domestic services. Termed *free women*, African women had few but regular male customers, exchanging services for money, shelter, and food. Although this practice may have allowed HIV to persist and adapt in this region, researchers do not believe it resulted in an HIV epidemic (Giles-Vernick et al., 2013; Pépin, 2013).

The Massive Parental Transmission Hypothesis. The Massive Parental Transmission Hypothesis is another theory that can explain the spread of HIV (Pépin, 2013; Sousa et al., 2017). Pépin (2013) notes that during World War I (1914-1918), French and Belgian-Congo colonizers established compulsory injection programs to control tropical diseases in Africans.

These programs targeted sleeping sickness, syphilis, yaws, and leprosy, and were implemented to protect colonizers from infections. Treatment consisted of drug injections with improperly sterilized syringes. After the implementation of these health interventions, outbreaks of Hepatitis C Virus (HCV) occurred within southern Cameroon communities. The presence of HCV after mass injection programs suggests iatrogenic spread—an illness caused by the medical treatment of a blood-borne virus. Similar scenarios of blood-borne viruses occurred in other African regions, such as Ebolowa, Nola, and Guinea-Bissau (Pépin, 2013).

Following the decline in tropical diseases, Pépin (2012) also discusses the colony implemented STD clinics in the 1950s. Clinics presumed *free women* and migrants had STDs and forced treatment upon them. Injectable treatments often lasted months, using unsterilized syringes which often had blood residue from previous patients and were large enough to transmit diseases. Cases of hepatitis B, a blood-borne virus, increased from 1951 to 1952. Researchers theorized that HIV could have been transmitted the same way. Previous transmissions of HCV from tropical disease treatments support this theory. Since STD treatment was compulsory for *free women*, they would have iatrogenically contracted HIV and passed it to their clients. These clients would transmit it to other women and the virus, as a result, would pass through the general population. Each transmission allowed HIV to adapt to its new host, building resilience and sustainability within the population (Sousa et al., 2017). Health treatments for tropical diseases and STDs allowed HIV to persist in these sub-populations (Giles-Vernick et al., 2013; Pépin, 2012). However, colonial health treatments do not fully explain the exponential global spread of HIV.

Gender Shifts. It was the gender revolution of the 1950s and post-colonialism that drove the exponential spread of HIV in Kinshasa (Giles-Vernick et al., 2013; Pépin, 2012). Giles-

Vernick et al. (2013) describe how young *free women* revolted against male-centred gender norms and patterns. With their new wealth and social status, they became known as fashionable women who frequented beer halls and nightclubs. These younger *free women* often changed male partners, seeing up to 1000 men per year (Giles-Vernick et al., 2013; Pépin, 2012). Similar behavioural patterns emerged 20 years later in Nairobi where the number of HIV infections increased after shifts in the social culture (Pépin, 2012).

Discussion

The epidemiological triangle explains the environmental factors that enhanced the global spread of HIV. From the evidence above, colonialism is likely a contributing factor to the local spread of HIV. Colonialism and its influence on urbanization, societal changes, and healthcare systems provided an environment for the rapid spread of HIV.

Other studies have analyzed novel enabling factors in Kinshasa that facilitated the spread of HIV (Sousa et al., 2017). Sousa et al. (2017) suggest that injections and sexual behaviour alone do not explain the exponential spread of HIV. They found that the peak of HIV cases coincided with genital ulcer disease (GUD). GUD is a sexually transmitted disease that increases one's chance of acquiring HIV when a patient participates in sexual intercourse (Sousa et al., 2017). Arguing the novelty of GUD and its coincidence with HIV cases, Sousa et al. (2017) contend that HIV could not have exponentially spread without GUD-infected patients engaging in enhanced sexual intercourse. Early injection campaigns and increasing promiscuity may have contributed to HIV's early adaptation to humans, but not its exponential spread across countries. Regardless of the exact origins of the HIV epidemic, Sousa et al. (2017) and other researchers acknowledge the role of colonialism in altering social and cultural dynamics through economic and health mechanisms (Giles-Vernick et al., 2013; Pépin, 2013). Colonialism created sustained

impacts that allowed the *environment* to converge with the *agent* and the *host*, resulting in the exponential spread of HIV.

The connection between colonial-induced changes in Central Africa and the HIV epidemic illustrates the significance of understanding how and why diseases emerge and spread. In the context of the recent COVID-19 pandemic, the history of HIV can help prevent future outbreaks by bringing attention to factors beyond the biological, such as the effects of urbanization and changes in social and cultural aspects of life (Morens et al., 2020). The interconnectedness of the world through international travel and anthropogenic changes such as deforestation and environmental degradation, bring hosts and agents closer together, resulting in conditions that favour outbreaks (Morens et al., 2020). Preventing future pandemics requires a multidisciplinary approach, addressing a broader range of factors that enhance the spread of diseases (Giles-Vernick et al., 2013).

Conclusion

Research suggests that colonialism likely enhanced the spread of HIV infection. Colonialism in Central Africa altered the social and physical environment, creating conditions influencing human and chimpanzee interactions (Giles-Vernick et al., 2013). Colonial urbanization, healthcare, and subsequent shifts in social dynamics allowed HIV to adapt, persist and spread, leading to an epidemic (Giles-Vernick et al., 2013; Pépin, 2013). Identifying the origins of infectious diseases requires approaches that recognize social, political, economic, and ecological factors (Giles-Vernick et al., 2013; Rupp et al., 2016). We currently live in an anthropogenic era in which human activities change the environment (Morens et al., 2020). These changes are especially relevant as the world recovers from the COVID-19 pandemic.

Addressing the impacts of colonialism is important to understand how human exploitation and shifts in society and culture can impact the spread of infectious diseases, like HIV. Future infectious diseases can be prevented by understanding how they emerge and applying multidisciplinary and holistic approaches to public health responses (Giles-Vernick et al., 2013).

References

- Altman, L. K. (1981, July 3). Rare cancer seen in 41 homosexuals. *The New York Times*.
<https://archive.nytimes.com/www.nytimes.com/library/national/science/aids/070381sci-aids.html>
- Andrews, S. M., & Rowland-Jones, S. (2017). Recent advances in understanding HIV evolution. *F1000Research*, 6, 597. <https://doi.org/10.12688/f1000research.10876.1>
- CDC. (2021, December 20). *Lesson 1: Introduction to epidemiology*.
<https://www.cdc.gov/csels/dsepd/ss1978/lesson1/section8.html>
- Faria, N. R., Rambaut, A., Suchard, M. A., Baele, G., Bedford, T., Ward, M. J., Tatem, A. J., Sousa, J. D., Arinaminpathy, N., Pépin, J., Posada, D., Peeters, M., Pybus, O. G., & Lemey, P. (2014). The early spread and epidemic ignition of HIV-1 in human populations. *Science*, 346(6205), 56–61. <https://doi.org/10.1126/science.1256739>
- Giles-Vernick, T., Gondola, C. D., Lachenal, G., & Schneider, W. H. (2013). Social history, biology, and the emergence of HIV in colonial Africa. *The Journal of African History*, 54(1), 11–30.
<https://doi.org/10.1017/S0021853713000029>
- HIV.gov. (2019, June 24). *How is HIV transmitted?* HIV.Gov.
<https://www.hiv.gov/hiv-basics/overview/about-hiv-and-aids/how-is-hiv-transmitted/>
- Morens, D. M., Breman, J. G., Calisher, C. H., Doherty, P. C., Hahn, B. H., Keusch, G. T., Kramer, L. D., LeDuc, J. W., Monath, T. P., & Taubenberger, J. K. (2020). The origin of COVID-19 and why it matters. *The American Journal of Tropical Medicine and Hygiene*, 103(3), 955–959. <https://doi.org/10.4269/ajtmh.20-0849>
- Pépin, J. (2012). The expansion of HIV-1 in colonial Léopoldville, 1950s: Driven by STDs or

STD control? *Sexually Transmitted Infections*, 88(4), 307–312.

<https://doi.org/10.1136/sextrans-2011-050277>

Pépin, J. (2013). The origins of AIDS: From patient zero to ground zero. *J Epidemiol Community Health*, 67(6), 473–475. <https://doi.org/10.1136/jech-2012-201423>

Rupp, S., Ambata, P., Narat, V., & Giles-Vernick, T. (2016). Beyond the cut hunter: A historical epidemiology of HIV beginnings in central Africa. *EcoHealth*, 13(4), 661–671. <https://doi.org/10.1007/s10393-016-1189-6>

Sharp, P. M., & Hahn, B. H. (2011). Origins of HIV and the AIDS pandemic. *Cold Spring Harbor Perspectives in Medicine*:1(1), a006841. <https://doi.org/10.1101/cshperspect.a006841>

Sousa, J. D., Müller, V., & Vandamme, A.-M. (2017). The epidemic emergence of HIV: What novel enabling factors were involved? *Future Virology*, 12(11), 685–707. <https://doi.org/10.2217/fvl-2017-0042>

Stanford. (n.d.). *Lentivirus fact sheet – Stanford environmental health & safety*. Retrieved April 1, 2022, from <https://ehs.stanford.edu/reference/lentivirus-fact-sheet>

UNAIDS. (2021). *Global HIV & AIDS statistics—Fact sheet*. <https://www.unaids.org/en/resources/fact-sheet>



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