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# Early and contemporary drivers of the HIV-1 group M pandemic

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## Abstract

***HIV emerged silently taking time to spread and become visible only through geographically isolated clusters of life-threatening immunodeficiency, known as AIDS since the early 80s. The clusters of infection expanded, overlapping to evolve into a pandemic that is ongoing and almost as silent. Phylogenetic analysis places the emergence of HIV-1 group M, the subtype responsible for the pandemic, in the human population more than 100 years ago. Once established, the rate and direction of spread of HIV-1 from local, to national, to contemporary pandemic proportions have varied over time and place. The literature presents many theories on the emergence and drivers of the spread of the virus over the past century. Here, historical evidence and phylogenetic models are reviewed to seek clarity on the emergence, geographic spread and key world events that mark the progression of the HIV-1 pandemic. This narrative review places particular focus on: war (both its direct and indirect affects), trade and economic expansion, changes in sexual behaviors, and public health policy. Investigating the impact of major world events and policy on the emergence and spread of HIV-1 may aid better understanding of what influences the viruses transmission dynamic. By identifying multilateral targets that influence transmission, up-scaled efforts to effectively control, if not remove, HIV-1 from the human population become a possibility. Suggestions for revisions in HIV-1 global public health policy are discussed. Refocused efforts to tackle HIV-1 transmission and replace the need to manage the pathology of this terrible disease are both ethically and economically just.***

## Keywords

***HIV-1. Molecular clocks. Phylogenetic analysis. Disease outbreaks. Pandemic drivers.***

## Introduction

### *HIV/AIDS pandemic today*

HIV, the virus that causes AIDS, was first isolated and described in 1983[1]. The discovery of the virus resulted from an investigation by the American Centres of Disease Control into unexplained clusters of Kaposi's sarcoma and pneumocystis pneumonia in New York and San Francisco[2]. The investigators found that the patients were immunocompromised, and linked to what is now known as the lesbian, gay, bisexual and transgender community. Initially called gay related immunodeficiency disease (GRID), the moniker GRID was replaced by AIDS as outbreaks among hemophiliacs and non-gay intravenous drug users were reported as early as 1982[3]. HIV was most likely prevalent in gay men in the USA before 1983, when the clusters of disease were apparent and the virus identified[4]. Indeed, Gilbert et al. estimated a 5% prevalence of HIV

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infection among men who have sex with men (MSM) in NYC by 1978[5]. Since then, HIV has developed into the ongoing pandemic, with 85.6 million people infected since the start of the pandemic, and 40.4 million cumulative deaths reported by January 2023[6]. Despite the global impact of HIV/AIDS, efforts to control and halt the spread of HIV worldwide are plagued by obstacles, including the lack of cure, civil unrest, and poverty in the countries and populations most affected by HIV/AIDS, as well as variable access to life-prolonging antivirals and the overlap with coexisting diseases[7]. It is worth noting that without access to modern anti-viral treatments, the overall untreated HIV mortality rate is over 90%[8].

The HIV virus is a genetically diverse lentivirus, a double-stranded RNA virus, with diversity driven by the poor precision, and lack of fidelity, of the reverse transcriptase enzyme responsible for viral genome replication[9]. Subsequent "daughter" viral particles, replications from the original, will have incorporated genetic errors that support rapid genetic divergence from the original, leading to the global diversity of HIV-1 types and subtypes that virologists have identified over the past 40 years. There are two main types of HIV virus: HIV-1 and HIV-2. HIV-1 accounts for most infections worldwide[10]. HIV-1 over time has diverged into four groups (M, N, O, and P), of which HIV-1 group M accounts for 90% of global infections[11]. Within HIV-1 group M, there are nine major subtypes, A-D, F-H, J and K, and other uncharacterized recombinant forms[12]. Different geographical regions tend to have specific subtypes that dominate their current HIV makeup, for example, HIV-1 group M subtype B dominates America and Western Europe, whereas subtype C, as well as a plethora of circulating recombinant forms, constitute just under half of all South African HIV-1 group M subtypes[13]. The genetic diversity of HIV-1 can be captured using nucleic acid sequencing and phylogenetics. Phylogenetic analyses, together with mathematical modeling, allow for detailed interrogation of HIV-1 group M divergence and spread over time[14].

Phylogenetic analyses have helped to explain the possible origin of HIV[15,16], identified drug resistance pathways[17], and been critical in identifying forces that drive the evolution of HIV[14]. This review will rely heavily on phylogenetics to support claims surrounding the epidemiological and anthropological factors that drive the HIV pandemic. Many models in the referenced literature utilize Bayesian phylogenetic methods and molecular clocks to calculate the "time to the most recent common ancestor" (tMRCA) for certain HIV-1 group M subtypes and epidemics. Bayesian phylogenetics are by nature probabilistic and an estimation based on what we already know[18]. The accuracy of these estimations, and hence molecular clocks, has been questioned due to the variable rate of evolution HIV exhibits (rate heterogeneity), the varying selection pressures within hosts and from anti-retroviral therapy (AVT), and until recently the lack of calibration samples[19]. However, growing evidence suggests that modern molecular clocks, particularly relaxed models, are flexible enough to account for these variables, and can provide accurate estimates regarding the history of HIV even in the absence of clock-like evolution[20]. Such claims are supported by the correct date estimation for new calibration samples extracted from archived tissues[21] and the clocks accuracy in more acute infection tracking[22]. Although advancements lie with newly discovered samples for revised calibration, an increase in the total number of complete HIV sequences accessible to researchers from a greater number of host populations and countries has also improved accuracy, and reduced sampling bias[23,24]. Therefore, there is sufficient evidence that molecular clocks are robust enough to accurately date sequences, and support the topics presented and discussed in this narrative review.

## Origins of HIV

Genetic evidence suggests that HIV-1 and HIV-2 both emerged, independently, from wild non-human primates and represent zoonotic diseases[25]. The zoonotic origin of HIV-1 groups M, N, O, and P is thought to be chimpanzees, specifically the subspecies *Pan troglodytes troglodytes* that inhabit the forests of modern-day Republic of Cameroon, Gabonese Republic, and Republic of the Congo[26]. Zoonotic transfer is hypothesized to have been facilitated by the use of, and trade in, primate bushmeat[27]. The HIV-1 virus is genetically similar to the primate simian immunodeficiency viruses (SIVcpz) that is prevalent in some, but not all, chimpanzee populations in West Africa[25]. Non-invasive fecal and urine sampling has demonstrated that two of the four subspecies of chimpanzee harbor SIVcpz. These chimp populations are roughly bounded by the rivers Congo in the south and Sanaga in the north, with infection rates of up to 30-50% within some troops[27]. This information is important, as it points to a potential geographic origin of HIV-1 suggesting that the SIV to HIV-1 crossover event took place in the forests of southern Cameroon[27].

Molecular genetics offer considerable insight into the early HIV pandemic and its origins. However, understanding the HIV pandemic relies not only on molecular genetics but also on the study of historical and anthropological events that uncover major non-viral influences in the emergence and spread of HIV-1. Human behavior, and at times politics, has helped drive the HIV-1 pandemic[28,29].

Here, literature discussing the emergence of HIV, and the sociohistorical/economic drivers that are purported to have fuelled the HIV pandemic, will be reviewed. These will be placed within the context of mathematical and phylogenetic models that describe HIV-1 group M diversity and evolutionary timelines. There remains much to be learned to halt this particular pandemic. This narrative review is designed to rekindle our awareness of the ongoing HIV-1 pandemic and highlight multilateral forces that influence the emergence and continued spread of HIV-1.

## Factors driving emergence and initial spread of HIV-1

The viral crossover event that resulted in the emergence of HIV-1 is estimated to have occurred around the beginning of the 20$^{th}$ century. Latterly, genome sequencing of archival samples suggests that HIV-1 group M was already well established in Kinshasa, Democratic Republic of the Congo (DRC) by 1960, and showing considerable diversity[30,31]. The question is what spurred HIV-1 group M transmission during this period?

Colonial trade was likely instrumental in the early spread of HIV-1 group Pepin et al.[32] suggest that during the period 1910-1940, steamers and powered boats on the Congo facilitated the rapid movement of people and consequently HIV-1 upstream from the forests of the modern-day Republic of Congo and Cameroon where HIV-1 "patient zero" is likely to have been[33]. The profitable Ivory and Rubber industries created trade between the German Cameroon (after 1920 a French colony) and the Belgian Congo, with state-owned companies dominating the market share during this time[34]. Gryseels et al.[21] further corroborate with Pepin et al., and hypothesize that commercial river traffic on the Congo brought HIV-1 to Kinshasa, a theory also supported by Keele et al.[35] and Hemelaar[11]. Gryseels et al.'s estimate of 1891-1918 as the tMRCA for HIV-1 group M overlaps with the height of colonial trade and enterprise. Indeed, the commercial interests of the colonial powers were so vehement; this period even saw French territory being ceded to the German Cameron in 1911, for improved logistics and ease of trade[36].

Similarly, Worobey et al.[31], Faria et al.[33], and Gryseels et al.[21] offer supporting evidence that Trade and Economic expansion were major factors in the spread of HIV-1 within the Congo basin beyond Kinshasa. Indeed, the period between 1920-1960 saw economic growth, increased transportation, and mass migration into and within the Congo basin that was related to trade[37]. This period saw the development of mining regions which led to the growth of the urban centers such as Lubumbashi, formerly Elizabethville, and Mbuji-Mayi. These cities became well connected with each other by the novel rail network that was carrying over one million passengers by 1948[37]. Urban connectivity infrastructure, miles of railway and extensive river docks, was dubbed the "voie national" (national track) on completion in 1928. Urbanization was often in the form of shanty towns, and migrant workers from across the colony moved to work in poor conditions and lived in the often squalor-ridden settlements constructed by the mining companies, such as Societé Minière de Bakwanga and Union Minière du Haut-Katanga[38]. Faria et al. suggest that it is likely that migrant workers from Kinshasa brought HIV-1 group M with them to these industrial centers along the newly created transport routes. HIV-1 group M, in these impoverished heavily populated hubs, diverged and evolved separately into subtypes[33]. Faria et al. estimate 1927 as the tMRCA for Lubumbashi's HIV-1 group M cluster, which aligns with the middle of Colonial Congolese economic expansion. This analysis supports the notion that economic growth and trade routes, paired with colonial disregard for African workers working conditions and health, facilitated HIV spread to other areas in the Congo basin.

Concomitantly, West Africa's population exploded exponentially by the mid-century through the development of large, densely populated, and interactive cities[39]. Population density, as well interactivity and trade would, unwittingly, have laid the foundation for infectious disease transmission. This is supported by recently discovered colonial medical records, documenting that sexually transmitted infections were rife among European colonists and Africans in major population centers during the colonial period, peaking around the estimated emergence time of HIV-1 group M in the 1910s[40].

The relative prosperity of Leopoldville and other cities in the Belgian Congo was the driver of sex work,

another important factor influencing viral diversity and spread. Sex work at this time in the Belgian Congo was dominated by *femme libres,* "free women" who engaged with only three to four regular clients[41]. Although HIV-1 spread into the larger cities via river transport, spread through *femme libres* sex workers in the late 40s/50s is thought to have been at replacement level. It was not until the rushed and mishandled decolonisation of the Congo by the Belgians, an event that culminated in the Congo Crisis (1960-65), that HIV-1 group M transmission exponentially increased[32,33]. Mass unemployment, economic crisis, and "pauperization", as coined by Pepin[32], due to the sudden economic downturn, forced a change in the sex work dynamic, from *femme libres* in favor of high-risk sex work involving hundreds of clients per year[32]. During this period, HIV-1 M spread exponentially and rapidly diversified. Phylogenetic analyses show that modern-day Kinshasa and Cameroon have the highest HIV-1 group M subtype diversity than anywhere else in the world[42].

It is clear that both colonial economic activity, as well as social deprivation as a consequence of colonialism, helped drive early HIV-1 group M transmission, which over time reached epidemic proportions and was the forerunner of the ongoing HIV-1 group M pandemic.

## Factors influencing epidemics and contemporary pandemic spread

### *Trade and economic growth*

The impact of trade and economic conditions on the HIV-1 pandemic persisted after the decolonization of Africa. In the wake of the various de-colonizing crises and economic decline the United Nations set up the "United Nations Organization in the Congo" (ONUC) to maintain peace, employ overseas professional aid, increase tradeand raise the standards of living as part of its commitment to decolonization[43]. The ONUC attracted, in particular, French speaking haitian professionals, who were recruited under the civilian operation program. Haitian expatriates could learn valuable skills working for both the UN and the Congolese civil service. Over the 4 year span of the initiative, thousands of Haitian professionals settled in the DRC, many with families[44]. Kevin Gaines discusses in his book "*African Americans in Ghana; Black Expatriates during the Civil Rights*"[45] that many young African Americans and Caribbeans saw decolonization as a nationalist project, but also an economic and investment opportunity – some decided to travel extensively, or emigrated, to Africa. The ONUC would have provided a conduit for this. However, owing to increased political violence during the lead-up to transition of the DRC from democracy to a Dictatorship (Zaire) in 1965, as well as the failed insurrection in Katanga, the UN withdrew its resources in 1964. Despite the initial success of ONUC, the subsequent failure resulted in Haitian repatriation (Fig. 1), and most likely the introduction of HIV-1 group M into the Caribbean through Haiti. Indeed, Worobey et al.[4] calculated the tMRCA for the Caribbean HIV-1 group M epidemic as 1964, and geographically placed it in Haiti.

Economics also seems to have played a part in the early introduction of HIV-1 group M into North America. In 1965 American immigration legislation was reviewed under President Lyndon B Johnson. Barriers to migrants from the Caribbean and South America were removed due to political pressure from the civil rights movement, but also to address labor shortages as the USA boomed in its post war economic expansion in the late 1960s[46]. Worobey et al.[4] calculated that HIV-1 group M subtype B, the most prevalent subtype in America, most likely came to the USA through New York City (NYC) around 1971, which aligns with historical economic events, and also a period of positive net and climbing immigration to the USA from the Caribbean[47]. This claim is given credibility due to advances in technology that have allowed HIV-1 group M genome sequences to be generated from serum samples archived in 1978. The sera were collected as part of a study conducted in 1978 on viral hepatitis C in MSM. Eight new complete HIV genomes were sequenced from the samples that added clarity to the history of the early American HIV-1 M epidemic. These sequences clustered heavily with Caribbean HIV-1 group M subtype B, particularly Haitian sequences, suggesting a Haitian introduction of HIV-1 group M into the USA during the early 1970's, and the foundation of the North American epidemic (Fig. 1). Such analysis provides robust phylogenetic corroboration with historical events, and supports the notion that drivers of economic prosperity are closely linked with the proliferation and spread of HIV-1.

### *War and colonialism*

War in general is socially and economically catastrophic. Communities immediately destabilized by war are vulnerable on many levels, especially vulnerable to infectious disease transmission. This is mainly through

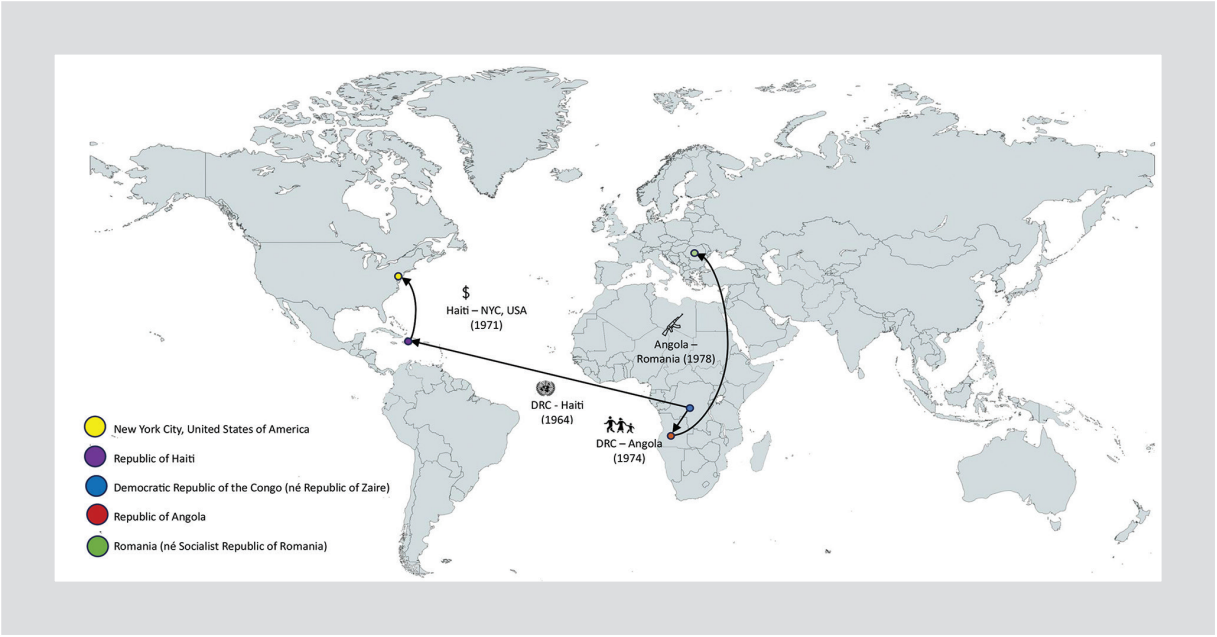


**Figure 1**. *Map detailing HIV transmission by theme and date.* **A:** *returning Haitian expatriates due to collapse of ONUC 1964.* **B:** *likely American introduction in 1971 via NYC with economic migration from Caribbean.* **C:** *angolan introductions relating to refugee migration in 1974.* **D:** *founding of Romanian epidemic in 1978, relating to communist Romanian political and military involvement during the Angolan civil war (1975-2002). Map courtesy of Mapchart.net (2024).*

the lack of basic healthcare, induced poverty, and poor sanitation that arise as infrastructures are destroyed, people displaced and mobilization begins. For example, it is estimated that more people died from disease during the Napoleonic wars than were killed by conflict, and more recently in the aftermath of the Rwandan genocide (1994), cholera and dysentery killed over 12000 refugees in camps in the DRC[48,49]. Furthermore, the political motivation behind war, specifically in post-colonial Africa, led to the involvement of multilateral coalitions, further destabilizing governments and leading to the exchange of military personnel. Within the context of HIV-1, a series of events in Angola, starting with the independence war and later the proxy civil war, links two separate HIV-1 group M epidemics thousands of miles apart.

After a decade long conflict, Angolan independence from Portugal was won in 1974. Although the most prevalent HIV-1 group M subtype in Angola is F1, a unique variant that accounts for 23% of infections, and despite virus prevalence being low at 3.7% relative to neighboring countries, Angola holds a high level of overall subtype diversity, mirroring that of the DRC[50,51]. High diversity indicates these locations are close to, or interactive with, the geographic origins of the HIV-1 group M pandemic, as suggested by Sharp[27], Pepin[52], and others[30,39]. Bello et al.[50] proposed that, when peace came in 1974, Angola suffered multiple HIV-1 group M introductions from various returning refugees from the DRC. This assumption is supported by Faria et al.[33] and Worobey et al.[53] whose data that captures a scenario of high HIV diversity across the DRC, in all major population centers and cities by 1974. Refugees returning to Angola from cities in the DRC increased the probability of introducing multiple variants of HIV-1 group M into Angola including the F1-subtype (Fig. 1). Furthermore this proposal is substantiated by UN records, which show the period 1975-1980 saw a positive and climbing rate of net migration back into Angola from the DRC[54]. Almost immediately following Angolan independence, the cold-war (1947-91) fuelled the Angolan civil war (1975-2002). Mehta et al.[55] estimated tMRCA for the Angolan F1 subtype in Angola was 1975, coinciding directly with the height of Angolan socio-political unrest.

Through the civil war and multilateral interference, Angola became a proxy for conflict between the United States of America (USA), the South African Backed "União Nacional para a Independência Total de Angola", and the Soviet communist "Movimento Popular de Libertação de Angola". Such a dynamic ultimately led to administrative and military support from

around the world[56], including the deployment of troops to Angola from many countries including communist Romania, a Soviet ally[57]. To date Romanian HIV-1 group M, infections are overwhelmingly of the F1 subtype (99%)[55]. Mehta et al.[55] suggest that it is highly likely that a single founder effect, an introduction of HIV-1 group M F1 from Angolan-based military personnel, was responsible for the contemporary Romanian HIV-1 epidemic[50] (Fig. 1). Mehta et al.[55] and Bello et al.[50] constructed maximum likelihood trees from just over 350 HIV-1 group M sequences from Romanian patients, and both demonstrate that Romanian HIV-1 group M F1 sequences clustered with Angolan F1 linages. Furthermore, Mehta et al.'s tMRCA estimate for the Romanian pandemic, 1978, coincides directly with Romanian military involvement in Angola.

Similarly there is evidence to suggest the establishment of HIV-1 group M subtype C in Zimbabwe, the former Rhodesia, was influenced by civil war. Following independence from the British in 1965, Rhodesia was ruled by a white minority until 1979. During this short-lived Rhodesian statehood, there was a series of guerrilla conflicts dubbed "the Bush Wars" where government forces engaged in skirmishes with the African liberation guerrilla army from 1964-79. Dalai et al.[58] estimated that the tMRCA for the Zimbabwean HIV-1 M subtype C epidemic was likely to have been 1973, which corresponds with the height of the bush-wars, and linked to low-level refugee migration and trade between Rhodesia and neighboring countries such as Zambia at the time. About 90% of subtype C sequences clustered with sequences originating from neighboring countries, which reaffirms this suggestion. Although freedom of movement was heavily restricted by security forces, this is the likely entry point for HIV-1 group M into the former Rhodesia[58]. Bayesian skyline plots show a significant increase in HIV introductions during 1979-81, coinciding with the fall of Rhodesia[58]. In 1978, the "Internal Settlement" was signed between the then Rhodesian Prime Minister Ian Smith and African nationalist leaders-the agreement allowed outlawed radicals and political opposition groups to return from exile in surrounding countries. The return of thousands of expatriates likely contributed to HIV-1 group M's expansion in Zimbabwe between 1979 and 81. In addition, the socio-political fallout following the fall of Rhodesia is suggested to have led to a rapid increase in HIV-1 group M infections into the mid and late 1980s, as demonstrated by Dalai et al.'s Bayesian skyline analysis and corroborated by NBTS (National Blood Service Zimbabwe) historical blood donor screening data[58]. Health-care infrastructure disruption with little public testing, and potential blood component contamination under the Mugabe regime post-1980, created prime conditions for HIV-1 spread[59]. Corroboration and alignment of data from various historical sources with the Dalai et al. skyline plots, and tMRCA estimates, support the hypothesis that war, and its consequences, played an important role in the Zimbabwean HIV-1 group M epidemic.

Wars of independence and subsequent civil wars strongly suggest that colonialism in Africa, and subsequent fights for freedom, were influential in the spread of HIV-1 group M. This ties to colonial economic trade, suggesting that the impact of colonial states created multifactorial pressures that spurred on the HIV-1 group M pandemic.

## *Public health*

Public health policy and health-care protocols have also contributed to the global spread of HIV-1 group M. By the early 1990s, it was evident that one of the largest HIV-1 group M pediatric epidemics was occurring in Romania as a result of earlier radical change in Romanian policy[60]. In the 1960s, the Romanian president Nicolae Ceausescu instituted pro-natalist policies that included a ban on abortion concomitant with the restricted use of contraception. The net result was a desired increase in population size but at the cost of thousands of abandoned children and abortion-related deaths[61]. By 1980, the government ran orphanages, Cighids, to provide basic shelter for abandoned and destitute children. The orphanages were impoverished, overpopulated, and inhumane, and until 1990 were hidden from international view[62]. Using tMRCA and Bayesian skyline plots, Mehta et al.[55] indicate that HIV-1 M subtype F was introduced to the orphanages in the mid-1980s. The introduction of HIV-1 into the orphanages was most likely due to vertical transmission from infected mothers, with the original introduction from Romanian military personnel returning from the Angolan Civil War in the late 1970s[50,55]. Poor hygiene and the use of unsterilized needles during mass vaccinations, blood-letting and transfusions is hypothesized as the main vehicle of high transmission rates of HIV-1 in the orphanages during the 1980s. Mehta et al.'s work corroborates that of Walker[62] who describe medical care in the orphanages as inadequate, with poorly educated medical professionals with little or no equipment or resources-prime conditions for needle contamination and the iatric spread of HIV-1.

Public health inadequacies, as in Romania, have also been hypothesized to have contributed to the early spread of HIV-1 group M in Central Africa. Faria et al.[33] reported the use of unsterilized needles in colonial sexual health clinics where new arrivals to Leopoldville were tested, treated, and or vaccinated in the clinics, strongly suggesting that many new migrant workers seeking fortune in the economically thriving Leopoldville of the late 1950s, typically younger men, may have inadvertently been infected with HIV-1 also through iatrogenesis[32,63]. Skyline plots constructed by both Faria et al. and Olabode et al.[64] show an increase in the rate of transmission and new infections between 1950 and 65 in the DRC. Along with increased urbanization and the consequences of decolonization in 1960s Kinshasa, an increased rate in HIV-1 group M transmission during this period, evident in phylogenetic analysis, may also be due to iatrogenic spread[52]. Belgian medical journals from this time describe hepatitis C outbreak following vaccine cohort effects based on the year of birth[65]. If there is evidence to suggest blood-born contamination from previous vaccination schemes in Colonial Africa, then it is possible that HIV-1 group M also spread via this insidious route. However, this theory is circumstantial, and in-depth investigations are hampered by the lack of colonial archives, it is likely that population growth and the change in sex work practices played a larger role in HIV-1 group M transmission from maintenance to exponential growth.

The argument that initial complacency and low public health interest around the early HIV/AIDS epidemic in the West that came from prejudice toward its main suffers, typically MSM and immigrants, deserves comment. The argument is supported by Worobey et al. in Bayesian skyline data that demonstrate huge increases in North American infections in the 1980s that leveled off in the 1990s when public awareness improved. The 1990s saw AVT become available, and public health measures to block transmission improved, for example, the promotion of needle exchange programs, point-of-care testing, and barrier contraceptives[66]. It could be argued that such measures occurred as infections became more pervasive in all groups in the 1990s, not just disempowered ones, leading to increased research funding and government action. Treichler[67] backs this claim and argues the stigma that evolved, and the lack of acknowledgement and research from governments of the 1980s, helped to propagate the HIV-1 pandemic among already marginalized groups.

## Conclusion and recommendations

Despite the prolonged HIV pandemic and lack of worldwide treatment equity, HIV-1 group M infections have decreased by 31% in the past 10 years-but 1-2 million people still become newly infected every year[6]. Greater understanding of the past may lead to better informed decision-making and strategic planning for the future. The events recounted here offer a tool to inform our current approach. When investigating HIV's past, the application of Bayesian phylogenetics and skyline plots can provide support for theories on viral proliferation, and provide evidence on important factors in the spread of HIV-1 group M. The HIV-1 group M pandemic is already diverse-without effective current global intervention, the virus will continue to mutate, resist treatment, and cause suffering in communities.

Based on the literature and events reviewed here, it would seem prudent to reassess the resources and attitudes toward the HIV/AIDS pandemic, and zoonotic disease emergence. Widespread distribution of prophylaxis, treatment, and education to those most at risk is a human right and scientifically optimal. HIV/AIDS follows poverty, and it follows vulnerable communities. Worldwide action surely must put aside profit and self-interest in favor of a collective effort to universally acknowledge the ongoing HIV pandemic through the introduction of robust and universal efforts to reduce the incidence of, and ultimately eradicate, HIV/AIDS. Furthermore, Neo-colonialism in Southern Africa along with the climate change crisis is both likely to induce and intensify the spread of HIV. This is especially relevant regarding wars of resources (such as water), and deteriorating public health infrastructure as a result of conflict and natural disasters. These factors highlight the need for swift and well-funded efforts towards HIV eradication. Evidence of HIV drug resistance further underpins the urgency of this recommendation[68].

The integrated approach of "One Global Health" is recommended to help detect and prevent zoonotic disease emergence[69]. On the backdrop of the COVID-19 pandemic and the ongoing HIV/AIDS pandemic, both emerging infections of zoonotic origin, well-funded and comprehensive strategies to detect and control emerging infections at local, national, and international levels are essential to prevent future pandemics. Such strategies are more relevant than ever and not something that humanity can sweep under the carpet; curbing emerging infectious diseases is one of the most important issues of our time. For now, breaking the silence of and halting the HIV pandemic is surely imperative.

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## Conflicts of interest

None.

## Ethical disclosures

**Protection of human and animal subjects.** The authors declare that no experiments were performed on humans or animals for this study.

**Confidentiality of data.** The authors declare that no patient data appear in this article. Furthermore, they have acknowledged and followed the recommendations as per the SAGER guidelines depending on the type and nature of the study.

**Right to privacy and informed consent.** The authors declare that no patient data appear in this article.

**Use of artificial intelligence for generating text.** The authors declare that they have not used any type of generative artificial intelligence for the writing of this manuscript nor for the creation of images, graphics, tables, or their corresponding captions.

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