INTRODUCTION

Human immune deficiency virus (HIV) is closely related to the simian immunodeficiency virus which is found in monkeys and apes. HIV-I is closely related to the strains of SIV found in chimpanzees, and HIV-II is closely related to the strains of SIV found in the sooty mangabeys [1, 2]. In 1999 some scientists found a strain of SIV (called SIVcpz) in chimpanzees that was closely related to HIV-I in humans. It was then concluded that this proved that chimpanzees were the source of HIV-I and had crossed species to humans at some point in time [3]. Furthermore it was thought that these chimpanzees got the SIV virus by eating two smaller monkeys (red-capped mangabeys and greater spot nosed monkeys) and became infected with two strains of SIV. These two strains joined together to form the SIVcpz strain which has the ability to infect humans [4]. This was followed by the concept of the hunter who ate these chimpanzees and became infected or the viruses were able to access the hunter through wounds and cuts. Occasionally the hunter would be able to ‘fight’ off this SIV, but sometimes they were able to survive in humans as HIV [2].

HIV-II comes from SIVsmm in sooty mangabey monkeys rather than chimpanzees [5]. The crossover to humans is believed to have happened in a similar way (through the killing and consumption of monkey meat).

It is far rarer, and less infectious than HIV-1. As a result, it infects far fewer people, and is mainly found in a few countries in West Africa like Mali, Mauritania, Nigeria and Sierra Leone [2]. There are four groups of HIV strains M,N,O,P with M being the most common with nine subtypes (A-D,F-H,J,K). There are so many strains because in the attempt to enter and adapt to survive within the human system the SIV had to evolve into several strains the most successful being able to survive the new environment [6].

Researchers have come to the conclusion that this “zoonotic” infection of humans with this virus started around 1920 in the Democratic Republic of Congo [6]. The same area is known for having the most genetic diversity in HIV strains than anywhere else, reflecting the number of different times SIV was passed to humans. Many of the first cases of AIDS were recorded there too. The extensive transport routes in that part including the railways, rivers and road networks together with the high migrant population and growing sex trade all supported the spread of the HIV virus. The lack of transport routes in the north and east of the country accounted for the lower reports of infections from these regions at the time [7]. By 1980, half of all infections in DR Congo were in locations outside of the Kinshasa area, reflecting the growing epidemic [6].

Molecular epidemiological studies have indicated that most, if not all, of the early diversification of HIV-1 group M likely occurred in the area around Kinshasa, then called Leopoldville. All of the known HIV-1 group M subtypes were identified there, as well as additional lineages that have remained restricted to this area. Leopoldville was also the place where the earliest strains of HIV-1 group M were discovered. Genetic analysis of infected blood and tissue samples collected from residents of Kinshasa in 1959 and 1960, respectively, revealed that HIV-1 had already diversified into different subtypes by that time. Finally, demographic data indicate that pandemic HIV-1 emerged at a time when urban populations in west central Africa were expanding. Leopoldville was the largest city in the region at that time and thus a likely destination for a newly emerging infection. Moreover, rivers, which served as major routes of travel and commerce at the time, would have provided a link between the chimpanzee reservoir of HIV-1 group M in southeastern Cameroon and Leopoldville on the banks of the Congo. Thus, all current evidence points to Leopoldville/Kinshasa as the cradle of the AIDS pandemic [8-10].

The spread to the western hemisphere was thought to have occurred when HIV-1 strains arrived in New York City from Haiti around 1971. It then spread from New York City to San Francisco around 1976.
HIV-1 is believed to have arrived in Haiti from central Africa, possibly from the Democratic Republic of the Congo around 1967. The current consensus is that HIV was introduced to Haiti by an unknown individual or individuals who contracted it while working in the Democratic Republic of the Congo circa 1966, or from another person who worked there during that time. A mini-epidemic followed, and, circa 1969, yet another unknown individual brought HIV from Haiti to the United States. The vast majority of cases of AIDS outside sub-Saharan Africa can be traced back to that single patient (although numerous unrelated incidents of AIDS among Haitian immigrants to the U.S. were recorded in the early 1980s, and, as evidenced by the case of Robert Rayford, isolated incidents of this infection may have been occurring as early as 1966).

The virus eventually entered the male gay communities in large United States cities, where a combination of casual, multi-partner sexual activity with individuals reportedly averaging over 11 unprotected sexual partners per year and relatively high transmission rates associated with anal intercourse allowed it to spread explosively enough to finally be noticed. Because of the long incubation period of HIV (up to a decade or longer) before symptoms of AIDS appear, and, because of the initially low incidence, HIV was not noticed at first. By the time the first reported cases of AIDS were found in large United States cities, the prevalence of HIV infection in some communities had passed 5%. Worldwide, HIV infection had spread from urban to rural areas, and had appeared in regions such as China and India [11-13].

The present AIDS epidemic officially began on June 5, 1981, when the U.S. Centers for Disease Control and Prevention in its Morbidity and Mortality Weekly Report newsletter reported unusual clusters of Pneumocystis pneumonia (PCP) caused by a form of Pneumocystis carinii (now recognized as a distinct species Pneumocystis jirovecii) in five homosexual men in Los Angeles [14]. Health authorities soon realized that nearly half of the people identified with the syndrome were not homosexual men. The same opportunistic infections were also reported among hemophiliacs, users of intravenous drugs such as heroin, and Haitian immigrants—leading some researchers to call it the “4H” disease [15-17].

By August 1982, the disease was being referred to by its new CDC-coined name: Acquired Immune Deficiency Syndrome (AIDS). The next important step was isolation and identification of the virus as the cause of HIV. In May 1983, doctors from Dr. Luc Montagnier’s team at the Pasteur Institute in France reported that they had isolated a new retrovirus from lymphoid ganglions that they believed was the cause of AIDS. [75] The virus was later named lymphadenopathy-associated virus (LAV) and a sample was sent to the U.S. Centers for Disease Control, which was later passed to the National Cancer Institute (NCI) [18, 19].

In May 1984 a team led by Robert Gallo of the United States confirmed the discovery of the virus, but they renamed it human T lymphotrophic virus type III (HTLV-III) [20]. In January 1985, a number of more-detailed reports were published concerning LAV and HTLV-III, and by March it was clear that the viruses were the same, were from the same source, and were the etiological agent of AIDS [21, 22].

In May 1986, the International Committee on Taxonomy of Viruses ruled that both names should be dropped and a new name, HIV (Human Immunodeficiency Virus), be used [19, 23]. From then on till 1996 when the highly active antiretroviral therapy (HAART) was introduced the diagnosis of infection with this virus was a death sentence as it was only a matter of time before the patient’s immune system succumbed to the invasion of the virus and death followed. There has been a dramatic decline in the number of AIDS cases and AIDS related deaths with the introduction of HAART. HAART is the use of a combination of multiple drugs (often three) to retard the multiplication of the HIV virus and subsequently reduce the viral load, protecting the immune system. However it is not curative. These drugs include the following:

- Nucleoside/nucleotide reverse transcriptase inhibitors, also called nucleoside analogs, such as abacavir, emtricitabine, and tenofovir. These medicines are often combined for best results.
- Nonnucleoside reverse transcriptase inhibitors (NNRTIs), such as efavirenz, etravirine, and nevirapine.
- Protease inhibitors (PIs), such as atazanavir, darunavir, and ritonavir.
- Entry inhibitors, such as enfuvirtide and maraviroc.
- Integrase inhibitors, such as dolutegravir and raltegravir.

The introduction of HAART lead to the evolution of a deadly disease into what is now more like a chronic ailment. However not without attendant complications of HIV patients on the drugs. These include anaemia caused by Zidovudine. Other well recognized complications include metabolic syndrome (MetS), abnormal fat distribution, cardiovascular diseases (CVD) and abnormal glucose metabolism in some cases leading to diabetes mellitus (DM). Others include elevated blood pressure and increased levels of triglycerides (TY) and other dyslipidaemias [24]. However it must be stated that the
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In June 2015 and 7.5 million in 2010. This means that 46% of receiving antiretroviral treatment (ART) - up from 15.8 million in 2010. As of December 2015, 17 million people living with HIV were in efforts to slow the spread of new infections. Some countries have achieved a decline of 50% or more in new HIV infections among adults (15+). The vast majority of them (an estimated 19 million) live in sub-Saharan Africa. The major of them (an estimated 19 million) live in east and southern Africa which saw 46% of new HIV infections globally in 2015. Around 40% of all people living with HIV do not know that they have the virus [35, 36].

In 2015, there were roughly 2.1 million new HIV infections, 150,000 of which were among children. Most of these children live in sub-Saharan Africa and were infected via their HIV-positive mothers during pregnancy, childbirth or breastfeeding 37. Progress in decreasing new HIV infections among adults has slowed in recent years. Since 2010, the annual number of new infections among adults (15+) has remained static at 1.9 million. A comparison of country data shows huge discrepancies in efforts to slow the spread of new infections. Some countries have achieved a decline of 50% or more in new HIV infections among adults over the last 10 years, while many have made no measurable progress. Yet others are experiencing worrying increases in new HIV infections 36.

As of December 2015, 17 million people living with HIV were receiving antiretroviral treatment (ART) - up from 15.8 million in June 2015 and 7.5 million in 2010. This means that 46% of all adults and 49% of all children living with HIV are now accessing ART 36. Significant progress has also been made in the prevention of mother-to-child transmission of HIV (PMTCT). In 2015, 77% of all pregnant women living with HIV accessed treatment to prevent HIV transmission to their babies 36.

In 2015, US$ 19 billion was invested in the HIV and AIDS response in low- and middle- income countries with 57% of the total HIV resources in these countries coming from domestic budgets 36.

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Advances are being made in efforts to create a vaccine that would effectively neutralize the HIV virus. South Africa is currently participating in an experimental vaccine programme that could prevent HIV infection. With 5 400 adults taking part, the study, called HVTN 702, is the biggest and most advanced HIV vaccine trial to take place in the country. The vaccine being used is based on one that was tried in Thailand previously. Those results, released in 2009, showed that at 31.2% effectiveness, that vaccine could only modestly prevent HIV infection. It is believed the updated vaccine could provide greater protection. The results of these trials are expected in 2020. (NIH and SouthAfrica.info reporter).

To take the AIDS response forward, UNAIDS has developed a Fast-Track approach to reach a set of time-bound targets by 2020. The targets include 90% of all people living with HIV knowing their HIV status, 90% of people who know their HIV-positive status having access to treatment and 90% of people on treatment having suppressed viral loads. They also include reducing new HIV infections by 75% and achieving zero discrimination.

The Fast-Track approach combined with a social justice agenda that puts people first and ensures that their sexual and reproductive health and rights needs are fully respected and met will be unstoppable. All this with a view to end the epidemic by 2030, making the worlds AIDS day (1st of December every year) redundant.

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