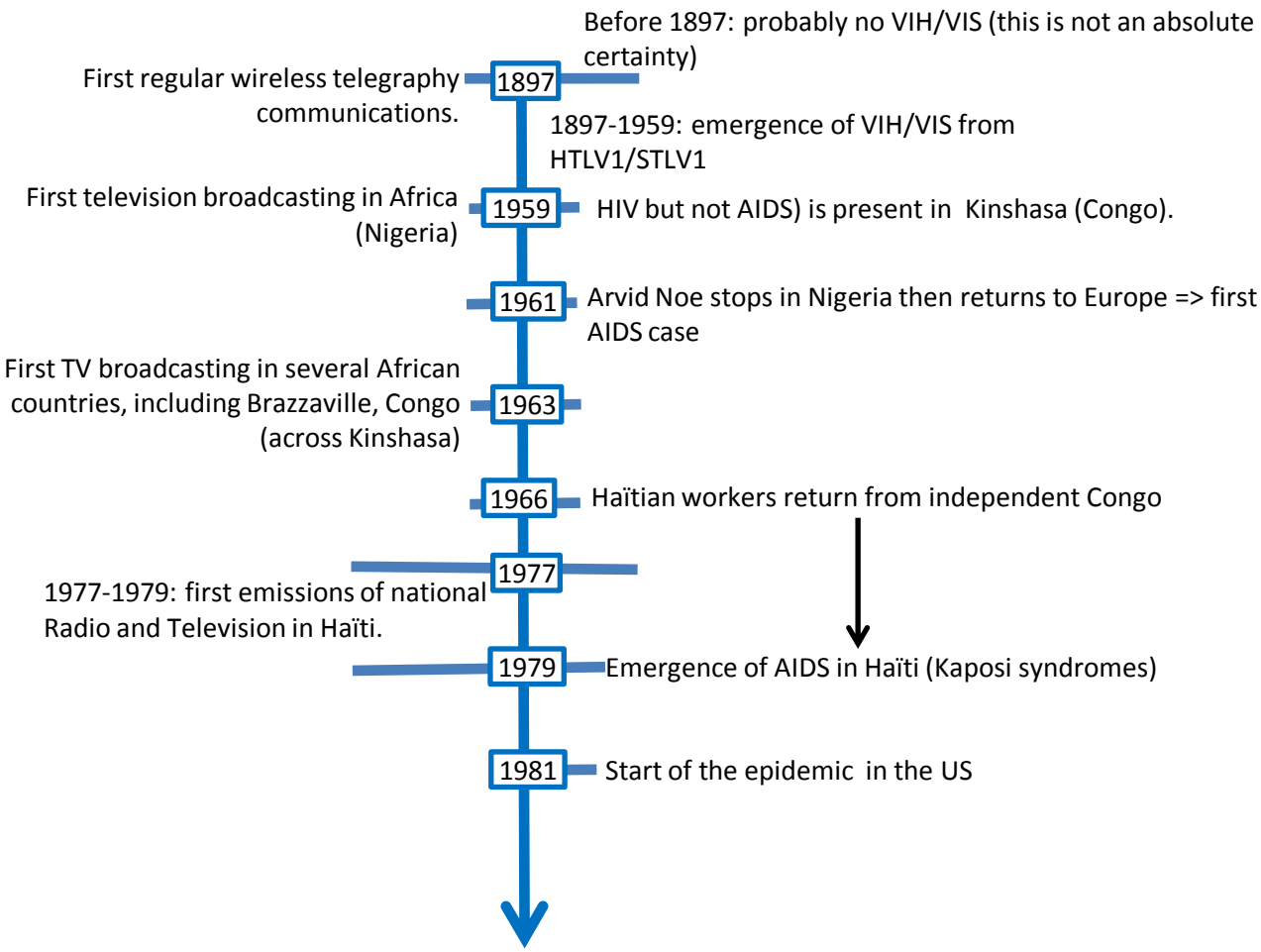
The emergence of HIV and AIDS

In short:

HIV is one in the family of immunodeficiency retroviruses, which affect several animal species. Immunodeficiency retroviruses descend from leukemia retroviruses, with which they co-evolved for some time. HIV is strongly connected to SIVs (Simian Immunodeficiency Viruses) which affect non-human primates. HIV/SIV probably evolved from HTLV-1/STLV-1 between 1897 and 1961. This evolution was accelerated by inter-species transmissions and by genetic diversity within the human species. It was rendered possible by the presence of protection gaps in the adaptive immunity, caused by artificial electromagnetic waves (see document "Interaction of electromagnetic waves with infectious diseases, including AIDS").

HIV/SIV emergence is connected with Television, which is a wideband emission thus having a strong pro-pathogen effect (based on inhibition INH of antigen recognition). HIV existed in Central Africa before the arrival of Television, but it did not cause AIDS. The arrival of Television accelerated its evolution and made it pathogenic. The first case of AIDS appeared in appeared in Europe (strong exposure) on a European who had traveled to Nigeria in 1961 shortly after the arrival of Television in that country. HIV was transported from Central Africa to Haïti in 1966 but the first case of AIDS in Haïti was in 1979 shortly after the onset of national radio and Television. From Haïti, the expansion of HIV to the United States and Europe was mainly limited by transmission of the virus.



The link with Leukemia Retroviruses

Group of species	Leukemia Retrovirus	Immunodeficiency Retrovirus
Old World Primates	Simian T-lymphotrophic virus (STLV-1)	Simian Immunodeficiency Virus (SIV) (Keele et al 2009)
	Human T-Lymphotropic Leukemia Virus (HTLV-1)	Human Immunodeficiency Virus (HIV)
Felidae	Feline Leukemia virus	Feline Immunodeficiency Virus (FIV) (Bendinelli et al 1995)
Bovinae	Bovine Leukemia Virus	Bovine Immunodeficiency Virus (BIV) (Zhang et al 1997)
Murinae	Murine Leukemia Virus	LP-BM5 (Cao et al 2012)
Koala	Koala Retrovirus (Denner and Young 2013)	

All species that have an Immunodeficiency Retrovirus have a Leukemia Retrovirus. Species that do not have Leukemia Retrovirus (canidae, Old World Monkeys, …) do not have Immunodeficiency Retrovirus.

The Immunodeficiency Retrovirus in Mice remained in a primitive form, associating a Leukemia Retrovirus and an Immunodeficiency Retrovirus which cannot replicate independently of the Leukemia Retrovirus.

In Humans, HIV is independent of HTLV-1 but remains favored by the presence of HTLV-1, with an abnormal number of co-infections and a faster evolution to AIDS in the presence of HTLV-1.

HIV proteins have conserved most functionalities of HTLV-1 proteins and are even interchangeable with HTLV-1 proteins in some cases.

The importance of inter-species transmissions and genetic diversity

One can define the viability of a virus as its capacity to replicate and survive on an individual host. The viability of HIV depends on its capability to evade recognition by the immune system and on the functionality of its proteins. The virus mutates progressively so as to increase its viability, and stabilizes when reaching a local maximum of its viability, that is when all simple mutations, typically single-nucleotide, yield a diminished viability. But this maximum is local and there may be other viral sequences which yield a better viability.

If after reaching this local viability maximum the virus is transferred to another species or even an other genetically different host, the viability maximum on the new host is not the same as on the initial host, so that the viral sequence of the transferred virus is no more stable. The viral sequence evolves again, reaching a new local viability maximum on the new host.

If the virus is transferred back to the initial host, it evolves again on the initial host, reaching a new local maximum which may correspond to a better viability than the initial local maximum.

Thus, transfer between different hosts participates in the evolution of the virus. The more different the hosts are (keeping a reasonable compatibility level so that the initially transferred virus can survive), the more the virus can pass stronger barriers separating different local maximums of viability in a particular host. Thus inter-species transfers can strongly accelerate virus evolution.

The presence of different hosts also allows recobinations of different parts of the virus, which accelerates evolution. It is thus commonly admitted that the SIV affecting chimpanzees (and therefore HIV, which is near to chimpanzee's SIV) would result from the recombination of two SIVs affecting different species.

The importance of transmissions between different species and between different hosts explains the emergence of HIV/SIV in Central Africa. Central Africa is where genetic diversity of both humans and non-human primates is highest. It is the sole place where humans co-exist with genetically near non-humans, i.e. chimpanzees and gorillas. It is a place where humans and non-human primates commonly interact (apes and monkeys are used as pets or for meat).

Can HIV/SIV descend from HTLV-1/STLV-1 ?

HIV/SIV is initially an HTLV-1/STLV-1 which has lost a functionality, i.e. the capability to stimulate replication of infected CD4+ T lymphocytes. Such losses of functionality are commonplace but often yield non-functional variants which later become eliminated. This primitive mutant will be noted HTLV-1-L/STLV-1-L).

But this particular loss of functionality allows HTLV-1-L/STLV-1-L to escape the humoral immune system. CD4+ T lymphocytes which are needed by B cells to produce antibodies against the mutant HTLV-1-L/STLV-1-L in the lymph nodes (hererafter ThL lymphocytes) are frequently infected by the mutant HTLV-1-L/STLV-1-L. Antibodies produced by B cells against HTLV-1-L/STLV-1-L bind ThL lymphocytes which are then eliminated by the immune system. Unlike the case of normal HTLV-1/STLV-1, the elimination of CD4+ helper T lymphocytes (ThL) which are needed for antibody production is not compensated by an accelerated mutation of these lymphocytes, so that antibody production ceases.

Absent electromagnetic waves and protection gaps, this modification cannot yield an autonomous viral variant, because it suppresses the main replication mechanism of HTLV-1, through lymphocytes which replication is stimulated. But in the presence of artificial electromagnetic waves, the modified HTLV-1-L/STLV-1-L can mutate to benefit protection gaps and largely escape the immune system. It then essentially escapes the immune system. It becomes capable of surviving independent of HTLV-1/STLV-1, yielding an independent HIV/SIV virus.

This evolution of HTLV-1-L/STLV-1-L towards HIV/SIV is a directed mutation, moving towards higher virus viability. Criterias are the conservation of basic functionalities of viral proteins, and the obtaining of proteins that escape the host's immune system. Evolution may thus be very fast. At the brute mutation speed of HIV/SIV (independent of the elimination of many variants by the immune system) 3 years are required to renew half the nucleotides, which is compatible with a formation of HIV/SIV from HTLV-1/STLV-1 in the early 20th century, even though other hypotheses exist.

There are two arguments against the HTLV-1/STLV-1 origin of HIV/SIV:
-The large genetic distance between HTLV-1/STLV-1 and HIV/SIV. As discussed above, material conditions are compatible with a complete renewal of nucleotides during the first half of the 20th century. Thus the genetic distance between the viruses does not justify excluding the STLV-1/HTLV-1 origin of HIV/SIV.
-The fact that HTLV-1/STLV-1 is a deltaretrovirus whilst HIV/SIV is a lentivirus. The distinction is based on morphological criteria which are unessential. The shape of the capsid depends on surface proteins, which in HIV/SIV are variable and thus do not yield the regular surface characteristic of deltaretroviruses. A number of simple mutations of HTLV-1/STLV-1 may modify the shape of the capsid.