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Review Article

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HIV/AIDS in Africa: Trends, Missing Links and the Way Forward

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Abstract

Within the African regions, there are striking differences in human immunodeficiency virus (HIV) disease burden yet social and cultural differences are relatively small suggesting that sexual transmission alone may not explain HIV infections in Sub-Saharan Africa (SSA). Thus, sexual contact with an infected person represents only a necessary, but not sufficient, condition for HIV transmission through sex. Several factors may contribute to the differential spread of the HIV pandemic in the region, including behavioral factors, unsafe medical practices, ethnic variation in HIV restriction genes, host nutritional status, viral characteristics including other environmental factors such as co-infections with other pathogens in addition to HIV infection. Each of these factors alone or in combination may determine susceptibility to HIV infection and consequently dictate the rate of progression to AIDS. To this end, there is need to elucidate the precipitating factors which have contributed to relatively more efficient HIV-1 transmission in SSA resulting in the virus infecting more than a quarter of the population in some communities. This review seeks to discuss regional differences in HIV-1 diseases burden and potential cofactors that may be central in fueling the HIV epidemic in SSA.

Keywords

HIV; Africa; Trends; Co-infections; Missing links

Introduction

Historical background of HIV/AIDS

A syndrome associated with severe immunodeficiency was observed in the United States of America (USA) among previously healthy homosexual men and intravenous drug addicts in 1981 [1]. The aetiological agent was isolated from the lymph nodes of suspected patients two years later [2,3]. By then it was called human T-cell lymphotrophic virus type-3 (HTLV-III) or lymphadenopathy-associated virus (LAV) which was later re-named human immunodeficiency virus (HIV) [4]. Transmission can be through vaginal, anal or oral sex, blood transfusion, hypodermic needles or from a pregnant mother to her unborn child during pregnancy, childbirth or through breastfeeding [5-7]. HIV causes progressive immunodeficiency leading to Acquired Immunodeficiency Syndrome (AIDS). It is currently one of the most devastating infectious diseases in the history of mankind. The earliest anti-HIV-1sero-positive blood sample was from an individual in Kinshasa, Congo in 1959 [8]. By 2011 distressingly, 20 million people had since died from the infection whilst another 38.6 million are living with HIV/AIDS (Figure 1).

Origin of HIV and zoonosis

The origin of HIV can be traced back to a Simian Immunodeficiency Virus (SIV) isolated from a Chimpanzee (cpz) sub-species, *Pan troglodytes troglodytes*(SIVcpz) in Southern Cameroon [10]. It is hypothesized that cross species transmission of HIV occurred from its primary host, the SIVcpz to humans. This zoonotic transmission of the virus from the non-human primates (NHPs) to humans is thought to have occurred through practices of hunting and butchering of NHPs or during the process of caring for captive NHPs alongside with poor laboratory handling of their respective virally infected tissues and/or fluids [11,12]. However, there are other alternative but unsubstantiated propositions to this complex and controversial topic on the origin of HIV [13].

HIV prevalence and trends in Africa

During the 1980s, researchers in Africa observed a high HIV prevalence among female commercial sex workers and patients attending sexually transmitted infections (STIs) clinics [14-17]. Consequently, a consensus was reached among AIDS experts dealing with Africa that heterosexual and vertical transmissions were the primary modes of HIV acquisition in adults and children, respectively [18-20]. Thus, it is now widely accepted that the HIV-1 epidemic in SSA is driven mainly by heterosexual transmission [21]. Husbands have been shown to acquire HIV-1 infection initially from extra marital affairs and then proceed to infect their wives [22,23]. Cultural practices such as inheritance of widows and re-use of sharps by traditional healers have also been implicated in driving the pandemic to alarming levels in some regions [24,25]. The African continent HIV pandemic trend reflects many co-existing sub-epidemics as shown in figure 2.

Striking differences in HIV prevalence in SSA: Within Africa there are striking regional differences in HIV prevalence [27,28]. Among women attending antenatal care (ANC), HIV prevalence increased from 20–26% between 1997 and 2002 for Southern Africa, but actually declined from 14% to 1.4% and 5% to 4% for Eastern and Western Africa, respectively during the same period [29]. Interestingly, in Kinshasa, the purported region for the origin of

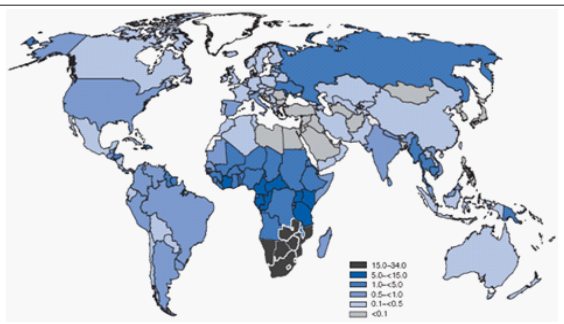

Figure 1: Global Heterogeneous HIV Burden [9].

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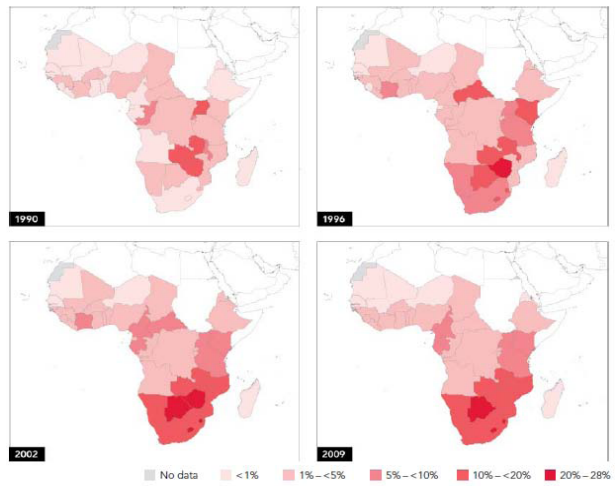

Figure 2: HIV Prevalence trends among the 15-49 year olds over the past 10 years in SSA [26].

HIV, sero-prevalence amongst pregnant women steadily rose from 0.25% in 1970, to 3.0% in 1980 and 5.7% in 1990s [30]. Remarkably, amongst some neighboring SSA countries huge differences in HIV prevalence have been observed being, 38%, 20%, 14% and 10% for Botswana, Zimbabwe, Mozambique and Tanzania, respectively yet social and cultural differences among these African countries are relatively small [31]. More than three decades into the HIV/AIDS scourge, the satisfactory explanation behind the observed regional differences in HIV transmission rates among different populations remains elusive.

A disturbing phenomenon in Africa is that unlike in Europe and the US where HIV is confined to intravenous drug users (IDUs) and homosexual men [32-35], the African epidemic is more widely distributed across the general population [36-38]. In view of the fact that many Africans even in stable sexual relationships are also infected, there has been growing interests to understand the dynamics and risk factors of HIV-1 transmission [39]. Thus, there is a research gap to elucidate the precipitating factors which could have contributed to a relatively much more efficient transmission of HIV-1 in SSA resulting in the virus infecting more than a quarter of the population in some communities [40-43].

Missing links and paradoxes

Studies have shown that differences in sexual behaviour across countries do not explain the observed differences in the epidemic trajectories [44]. African HIV incidence studies have reported more or less similar rates of transmission per coital act relative to the USA and Europe [45] yet SSA with just a mere tenth of the world's population, harbors about two thirds of all HIV infections globally and 94% of all pediatric HIV infections [46]. This observation is also inconsistent with the low probability of heterosexual HIV transmission per coital act [47,48]. Long-term concurrent relationships may be more common in parts of SSA, but studies have shown that national rates of concurrency do not correlate with rates of HIV [49-51]. Studies have also shown that existing SSA mathematical models grossly inflate per contact transmission efficiencies or rely on implausible assumptions regarding contact frequency with the average number of sexual partners being far much higher than what is supported by available data [48].

HIV infection has been confirmed in a number of pediatric cases where the source of infection has been not been adequately explained [52,53]. Interestingly, some early studies have observed all HIV cases in one age group [54]. Studies have reported adults who contracted HIV-1 infection without sexual experience suggestive of the possibility of other modes of transmission [55-63]. Critical analysis of the realities on the ground in an attempt to explain the observed heterogeneity in HIV prevalence is supportive of the hypothesis that HIV infections in SSA may not be explained by sexual or vertical transmissions alone [44,55,64-66]. Therefore, there is a research gap to satisfactorily explain the root causes of the disproportionate affliction of Africa by this pandemic especially when compared to other similarly sexually transmitted infections (STIs).

HIV and other STIS transmission rates dissonance

Studies have demonstrated that STIs may facilitate HIV transmission [67-69]. Thus these infections seem to fuel each other. Assuming this synergism, it defies common sense and logic to endorse the almost exclusive African heterosexual transmission hypothesis in the face of increasing HIV epidemic against the generally declining statistics of similarly transmitted STIs like syphilis [54,70]. Some studies have observed some anomalies in the epidemic trajectory between HIV and STIs where a linearly increasing HIV prevalence from 9% to 25% has been cited, against a remarkable decline in STI syndromic reports [71]. Ecological comparative studies from population based survey from high and relatively low HIV prevalence areas in Zimbabwe and Tanzania respectively, have reported more or less similar burdens of STIs but distinctive HIV prevalence [44,72]. Hepatitis B virus (HBV) which has similar modes of transmission to HIV and interestingly, even much more infectious has generally a much lower prevalence [73]. HBV infection is common in SSA with Mozambique having the highest incidence rate yet this country's HIV-1 prevalence is amongst the lowest in the region [74,75]. Several factors may contribute to the differential spread of the HIV pandemic within the regions including unsafe medical practices, viral characteristics, ethnic variation in host genetic factors as well as other co-infections. Each of these factors alone or in combination could determine susceptibility to infection and consequently the rate of progression towards AIDS.

Possibility of unsafe medical practices

Unacceptably high HIV-1 incidence among low risk married pregnant mothers within the region is also perturbing [20,76-79]. Studies in Africa showed an unexplained high HIV-1 incidence among pregnant women who were sero-negative at the first antenatal visit but sero-converted later during antenatal and post-partum periods [80,81]. This observation is suggestive that whatever happens during pregnancy and/or post-partum periods whether iatrogenic, sexual or otherwise accounts for the high HIV incidence rates observed among these generally low risky women. Injections have been found to be highly associated with pregnancy [62,82] alluding to the possibility of transmission facilitation from unsafe medical practices such as re-use of injections needles or unintentional administration of unsafe vaccines or drugs during pregnancy that could have resulted in the underlying HIV infection background effect observed some communities. There is a research gap to explain the underlying causes of high HIV-1 incidence associated with antenatal and postpartum periods.

Some researchers argue that the massive increase in the use of

medical injections for parenteral therapies to treat diseases could have been the possible source of background effect of HIV infection in some communities [59,60,83-86]. At the same time studies have shown HIV to stay infectious on a needle for more than two weeks [87]. This hypothesis of unsafe medical injections has been shown to be scientifically implausible as some countries like Egypt where despite the vigorous parenteral anti-schistosomal treatment campaigns have reported very low HIV prevalence but amusingly the highest hepatitis C virus (HCV) disease burden in the world. Some studies do not buy this hypothesis of unsafe medical injections as a possible source of background effect of HIV infection in some communities [83], yet it is impossible to ascertain the safety of the then administered injections and/or their contents in retrospect. HIV genetic diversity may also play significant roles in explaining the observed heterogeneity in HIV prevalence.

HIV genetic diversity

The general observation is that higher diversity of HIV-1 subtypes is associated with relatively slower epidemics while explosive epidemics have only one predominant subtype. In most southern African nations subtype C contributes to 93-100% of the HIV infection as shown in figure 3.

Differential subtype transmission efficiency may exist. Studies have shown HIV-1 subtype A viruses to have significantly higher rates of heterosexual transmission relative to subtype D viruses [88]. Subtype C has been shown to be more transmissible compared with other subtypes [89-92]. However, other studies have shown no apparent differences in the rate of MTCT of HIV-1 relative to other subtypes [93-95]. HIV sero-discordance has been found to be more common with particular HIV variants than others, again matching the HIV-1 disease burden distribution patterns [88]. Interestingly, HIV-1 subtype C common in SSA has been found to be the predominant subtype amongst sero-discordant couples trailed by subtypes B and A, respectively [96].

Discordant couples

An intriguing observation challenging the African exclusive heterosexual transmission hypothesis is the high prevalence of HIV discordant couples seen within the region [97-99]. Even more interestingly has been the observation that some of these HIV sero-discordant couples continue to bear children, implying unprotected sex [100]. Recent meta-analysis African studies have observed relatively large proportions of HIV-1 discordant couples with women as likely as men to be the index HIV-1 positive partner [101]. Regional differences in HIV-1 prevalence of discordant couples varies from 8-31%, and 16-31% for Eastern and Southern Africa respectively, coincidentally, reflecting the same trend with HIV-1 prevalence [65,97,102]. In Gugulethu district, South Africa, up to 31% of the HIV transmission among the discordant couples may not have been heterosexual [65]. This is suggestive that something other than simply heterosexual transmission may be involved taking cognizance that 96% and 77% of HIV-1 infected married women and men respectively, reported having sex exclusively on their matrimonial beds [103]. In Zimbabwe recent statistics amongst 2700 co-habiting couples has demonstrated that 11% of couples are HIV-1 sero-discordant [104]. Thus, sexual contact with an HIV infected person represents only a necessary but not sufficient condition for HIV transmission, raising a lot of questions. What accounts for high rates of HIV-1 discordance in SSA? Why do some individuals remain uninfected despite repeated sexual exposure to HIV-1 remains a mystery but offers a unique opportunity for studying host genetic factors conferring susceptibility or protection against HIV infection.

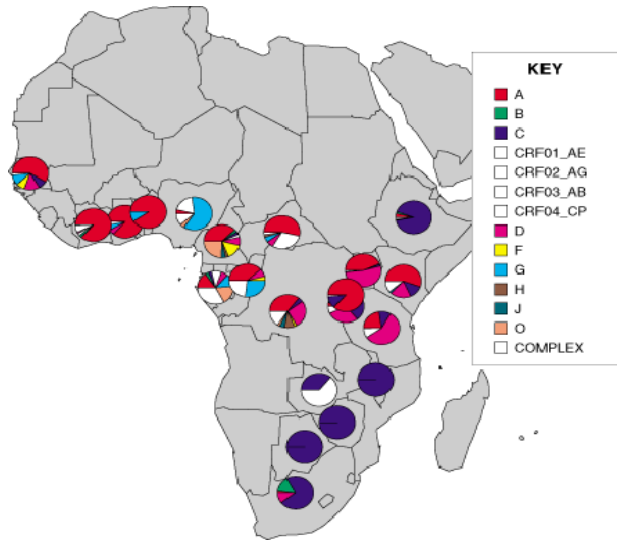

Figure 3: HIV-1 Diversity in Africa, Source: Los Alamos national laboratory.

Host genetic factors

There is substantial epidemiological evidence that host genetic factors such as closely linked genes of the Major Histocompatibility Complex (MHC) as well as non-MHC factors such as chemokine receptors are important determinants of susceptibility or resistance to infectious diseases. Among host genetic factors identified for their roles in HIV-1 transmission are polymorphisms in the genes encoding chemokine receptors CCR5, CCR2 and SDF-1, a natural ligand for CXCR4 receptor including killer cell immunoglobulin-like receptors (KIRs) [105,106]. Human Leukocyte Antigen (HLA) system is central in the recognition and presentation of pathogens to the immune system and therefore is a fundamental part of the human immune system. The distribution of these genetic polymorphisms has been shown to vary between different racial, ethnic including risk groups [107-109]. Several studies on HLA and disease association with HIV have been reported in different ethnic populations yet little information is available on HIV infected individuals in SSA. Considering the population specific differences in the frequencies of protective or susceptibility associated alleles and their influence on the disease acquisition; it is of utmost importance to strengthen ongoing efforts towards defining largely unknown genetic propensity in the SSA population. Thus, the study of host-virus genetics and epigenetics of drug naïve long term survivors of the AIDS pandemic may not be over emphasized if improvement on the control of possible future viral zoonotic epidemics is to be realised.

Other possible cofactors

Current thinking of scientific literature demonstrates that besides host genetic factors, ecological issues also play an important role in determining an individual's vulnerability to HIV infection and transmission. Studies have implicated some co-infections and other environmental factors as possible cofactors for the acquisition and transmission of HIV [110-113]. In the absence of the currently

unknown cofactors that may increase per-contact transmission rates, there is no evidence that African concurrency or networks are more risky than sexual networks elsewhere [114]. Data suggest that malnutrition and co-infections may be central in fueling the HIV epidemic in SSA.

Malnutrition: The geographic and pathophysiologic overlaps of the malnutrition and HIV infection epidemics in SSA has led to the realisation that nutrition is essentially a gene–environment interaction science that complicates relationship between the health of the individual, its genome, and the life-long dietary exposure [115]. Nutritional status is important in determining risk of acquiring diseases. There is a vicious cycle between HIV infection and nutritional status. HIV compromises the nutritional status of infected individuals and in turn, malnutrition worsens the effects of the infection itself by weakening the immune system consequently accelerating disease progression and death. Nutritional interventions can help people living with HIV and AIDS manage their symptoms, reduce susceptibility to opportunistic infections, promote response to medical treatment and improve overall quality of life. A 12 week Omega 3 polyunsaturated fatty acids intervention has been shown to be beneficial to HIV infected persons on antiretroviral therapy (ART) [116-118]. A significant proportion of patients in SSA who require ART are also malnourished because of a combination of HIV-associated wasting and inadequate nutrient intake [119] a situation further complicated by the general use of herbs and botanicals by HIV/AIDS patients in the region [120-123].

HIV and co-infections: Synergistic relationship between HIV and other co-infections has also been implicated as the biological cofactor for HIV acquisition and transmission [124]. Co-infecting pathogens exist in a dynamic homeostatic equilibrium with the host with pathogenesis attributable to the pathogen-triggered disturbance of this equilibrium and to the effectiveness of the host's immune responses [124]. Relative to single pathogen species, co-infection can alter transmission and clinical progression rates. On average 74% Africans are exposed to two or more bacterial, viral and parasitic infections whilst a good 26% grapple with six or more diseases including non-communicable diseases (NCDs) [125-127]. How these co-infections modulate HIV acquisition and disease progression requires further research especially for SSA where co-infections outnumber single infection a situation exacerbated by poverty and malnutrition. The scenario is further complicated by host genetic differences that may introduce more heterogeneity into the outcome of co-infection. Establishing the nature and consequences of co-infections requires integrated monitoring and research of different infectious diseases of which currently such data are rare.

Parasites: Reduced immune response caused by an HIV infection can lead to an increased susceptibility to parasitic infections. Recent studies have shown that parasitic infections such as helminthes infections and malaria could disturb the balance of anti-HIV immune responses and positively contributing to HIV replication, thereby accelerating progression to AIDS [128-130]. Dual helminthic and HIV-1 co-infections are quite common, particularly in SSA. Parasite-HIV co-infections are one of the neglected areas in HIV research yet HIV has become a major public health concern and research topic worldwide.

The convergent distribution of the HIV and helminthes infections is suggestive of a possible biologically plausible observation that persistent infection with helminthes may exacerbate the HIV epidemic in developing countries [131,132]. *Schistosoma hematobium* which affects almost 200 million people in SSA has been shown to act as a co-factor of HIV transmission. Lesions create open portals for HIV entry whilst inflammation in the genital area makes HIV transmission more efficient and consequently, risk of HIV acquisition is increased three-fold [133-137]. Chronic immune activation, altered immune cells distribution, immune suppression and altered cytokine profiles associated with a strong T-helper 2 (Th_2) bias increase susceptibility to the virus infection, including enhancing its replication and facilitating faster progression to AIDS [138]. Recent studies have shown that deworming HIV-infected persons results in a statistically significant increase in CD4 cell counts implying that a simple, inexpensive and effective deworming medication such as albendazole could allow HIV-infected people to postpone uptake of ART [139,140]. Areas of the world that are most affected by malaria also carry a heavy burden of HIV. Again in view of this overlap in their global distribution, it is presumed that malaria increases HIV RNA load as much as 10-fold thereby increasing transmission dynamics of the epidemic at the population level [141-144].

Bacterial co-infections: Emergence of HIV has exacerbated an already enormous number of cases of tuberculosis (TB) worldwide. T lymphocytes subset is important in the control of TB. Ironically HIV depletes CD4 T lymphocytes, which is likely to contribute to increased susceptibility of the co-infected persons to TB [145]. HIV is the potent factor in the progression of latent TB and is the most common serious infection associated with HIV infection. Co-infection with HIV and Tb is often described as 'a cursed duet" with up to 70-80% of HIV infected patients also having TB [146]. This situation reflects a large number of people receiving concurrent treatment in an era where drug-drug interactions and the role on treatment outcomes are not well documented.

Viral coinfections: In recent years data have accumulated on the interactions of co-infecting viruses with HIV. Co-infecting viruses generate negative and positive signals that suppress or up-regulate HIV-1 replication. Mounting scientific evidence points to a strong association between human papillomavirus (HPV), herpes simplex virus type 2 (HSV-2) and HIV acquisition [147-153]. HIV sero-positivity has also been found to be associated with the development of high-grade cervical squamous intraepithelial lesions among HIV-positive women in SSA. Due to the oncogenic activity of HPVs and the regional variations in the prevalent HPV types, it is clinically important to detect HPV infections and to accurately identify the particular HPV types associated with specific diseases and cancers locally. Thus an integrated and collaborative approach in dealing with the two conditions is warranted.

Conclusion

The reasons behind high HIV disease burden in SSA remain elusive. Co-infections could be facilitating efficient transmission in this region. A lot of studies have been done assessing single infections in isolation or independently yet in real life practical situation such solitary infections are rare. Most of currently published work often underplays co-infections and interactions consequently resulting in misleading conclusions. The prevalence of co-infections whether dual, triple, quads and so on in different population is not known and how these co-infections in isolation or combination(s) may modify or modulate disease acquisition/transmission, progression, mortality,

immune responses, neuro-or cognitive development and response to therapy are poorly described. Correlations of immunological profiles for different co-infections in immuno-competent including immune compromised in both ART naïve and experienced individuals and disease progression and or treatment outcome can be used to determine useful genetic markers in the hope of better predicting individuals at higher risk of developing AIDS faster or therapeutic failure. Dosage policy change will be the long term goal consequently, paving way for the much awaited "individualized medicine" tailor designed according to patient genotype and environmental factors.

A myriad of bottlenecks including high genetic variability, down regulation of MHC I molecules in infected cells, the shielding of the HIV by non-immunogenic glycan, which hinder binding of antibodies to the envelope protein and not to mention the latency of the virus complicate the development of an effective HIV vaccine. Correlates conferring protection against HIV also remain elusive and consequently prophylatic vaccine trials in human have failed to elicit protection. In view of these challenges, other prevention control strategies remain the cornerstone if HIV infection is to be curbed.

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