

# UNIVERSITY OF ILORIN



**THE ONE HUNDRED AND FORTY- NINETH (149<sup>TH</sup>)  
INAUGURAL LECTURE**

## **“REALITIES OF LIVING WITH HIV INFECTION”**

**By**

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Chairmanship**

of:

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## **Dedication**

This lecture is dedicated to all the people living with human immunodeficiency virus (PLHIV): the infected patients, their affected loved ones, the concerned health care givers and the supporting NGOs as well as other relevant stakeholders.

## **Courtesies**

The Vice Chancellor, Sir

Deputy Vice-Chancellors

The Registrar and the other Principal Officers of the University

Provost, College of Health Sciences

My Dean, Dean Faculty of Clinical Sciences

Deans of other Faculties

Professors and other Members of Senate

Chief Medical Director of the University of Ilorin Teaching  
Hospital (UITH)

Head of Department of Medicine and other Heads of  
Departments,

Academic and Non-Academic staff of the University

Men and women of the press

Students of the University of Ilorin

Students of the University of Ilorin Secondary School

Invited guests

Ladies and gentlemen

## **Preamble**

Mr Vice Chancellor Sir, permit me to lead you and the audience into the world of HIV care which is an evolving discipline in medicine and currently on the front burner of the whole world. I found myself in the field of HIV care courtesy of my teacher Prof. P.O. Oluboyo who in 2002 as the Chairman Medical Advisory Committee for UITH nominated me to a two-man committee on HIV care for the hospital. I had my training in internal medicine with a flare for the infectious aspects of pulmonary medicine especially tuberculosis (TB). However, when HIV made its debut and was found to be closely associated with TB. I included it in my portfolio of interest. That was how 15 years on I have developed a robust relationship with HIV/TB medicine. Today's inaugural lecture therefore, which is the 149<sup>th</sup> of its series will be on HIV/AIDS. It is titled REALITIES OF LIVING WITH HIV INFECTION. It will be delivered in three

parts i.e. what we know about the virus that causes AIDS, how we are managing HIV/AIDS as health care provider, and what need to be done to improve the lives of people living with the virus.

## **Introduction**

HIV is a retrovirus that lives in the blood, semen, and genital tract of the infected people. It is an emerging infection that is affecting humans in epidemic proportions across the world today. It infects human cells that have proteins called CD4 receptors on their surfaces. Helper and memory T-lymphocytes are the main targets of HIV. They are produced in the thymus and help to orchestrate both cellular and humoral immune response of the body to foreign invaders<sup>1</sup>. Normal count ranges from 500-1400cells/mm<sup>3</sup>. HIV uses CD4 cells as substrate to reproduce itself thereby depleting the body of it. At a certain low count, the body will lose its ability to mount adequate immune response to infections. Conventional pathogens will be causing severe or recurrent diseases and opportunistic ones that the body would ordinarily put in check would be having a field day. Knowing the level of CD4 counts in a PLHIV is therefore important for determining the severity of the infection and monitoring of responses to antiretroviral therapy (ART). In the national HIV treatment guidelines, a CD4 count of 350cells/mm<sup>3</sup> is an indication for initiating ART, and a decline in count of 25% is an indication for a change in therapy.

## **What is the origin of HIV?**

Scientists believed that HIV is a zoonotic disease that was transferred from animals to humans during the practice of bush meat hunting in some parts of Africa. It is believed that a cross-species transmission of a virus called simian

immunodeficiency virus (SIV) occurred from an Ape or monkey to a human when a bush meat hunter or vendor was bitten or cut while hunting or butchering an infected Ape<sup>2</sup>. Exposure to blood or body fluid of an infected chimpanzee or monkey was the suspected source of SIV infection<sup>3</sup>. Serological survey in support of this assumption showed that human infections by SIV are not rare in Central Africa, percentage of people showing evidence of current or past SIV infection was 2.3% among the general population of Cameroon, 7.8% in villages where bush meat was hunted and 17.1% in the most exposed people of these villages<sup>4</sup>.

How Ape's SIV transformed into HIV after infection of the bush meat hunter or handler is a matter of debate. However, theory of natural selection favours viruses that are capable of adjusting so that they could infect and reproduce in human cells. The concept of adaptation by serial passages proposed by Marx *et al.*,<sup>5</sup> that a foreign pathogen can increase its biological adaptation to a new host species if it is rapidly transmitted between hosts, while each host is still in the acute infection period lends credence to this hypothesis. Serial transmission of a virus encourages rapid accumulation of a better-adapted viral variant that could not be easily cleared by the host's immune system. Such a better-adapted variant could then survive in high numbers in the new host long after the short acute infection phase. Survival of the virus in high numbers increases its chance of an epidemic spread.

**Factors that facilitated the emergence of HIV:** There were interplay of many factors in the emergence of HIV from Apes to Humans and subsequent spread of the virus among humans. Perhaps it started overtime with natural process of evolution, with significant contribution from Human behaviour and practices. However, for establishment of HIV in humans at least five conditions must have been satisfied; 1) A vulnerable human population; 2) A nearby population of animal host (Chimpanzee, Gorrila or Mackak monkeys); 3) An infectious virus in the animal host that was capable of spreading from

animal to human; 4) Cross- species interaction between animal host and man to transmit enough of the virus to humans to establish a human foothold ; 5) Ability of the virus to sustain itself within human population i.e. spread from human to human and thus prevent the virus from dying out<sup>6</sup>.

Mr. Vice-Chancellor Sir, sub-Saharan Africa is rife for the above five conditions. African population is rising and human settlements are expanding, unequal distribution of resources is fuelling poverty, internal rebellion and civil strifes and sometimes wars across the land. People are therefore, forced to migrate into new geographical areas thereby encroaching on the habitats and coming in contact with animals that are potential hosts of infectious pathogens. It is strongly believed that HIV evolved from rural African regions, spread into cities aided by the city pull on the youths in search of better livelihood, and the attendant unrestricted social experimentations with drugs and sexual explorations. It then spread across national borders through land, air and sea travels. Combinations of these together with the use of blood and blood products in humans have facilitated the emergence, establishment and sustenance of HIV in human race long before it was realised.

**Types of HIV and their areas of distribution:** There are two **types of HIV**: HIV-1 and HIV-2. HIV-1 is the most virulent of the two, it is responsible for majority of the global pandemic. It is derived from SIV acquired from Chimpanzee (*Pan troglodytes troglodytes*), that are found in the forest of Central African nations of Cameroon, Gabon, and Republic of Congo<sup>7</sup>. The animal reservoir for HIV-2 is the sooty Mangabey monkeys (*Cercocebus atys*)<sup>8</sup> that are found in Senegal, Guinea, Sierra Leone, Liberia and Ivory Coast. By DNA sequencing, HIV-1 is subdivided into four groups; group M with a world-wide distribution, group O, N and P. The latter two are rare and are derived from Gorilla SIV found in West and Central Africa nations of Cameroon and Gabon<sup>9</sup>.



Group M is further sub-classified into 11 genetically distinct subtypes; A-K<sup>10,11</sup>, these are unevenly distributed the world over but Africa has a disproportionate higher share. Subtypes A and D are found in West, East and Central Africa as well as Eastern Europe, sub-type B is predominant among MSM<sup>12</sup> in Europe and North America. Subtype C is the major cause of all new HIV infections worldwide (South Africa, Brazil, and India). Subtype E in Southeast Asia. Subtype F: Brazil, Romania, D.R Congo. Subtype G: D.R Congo, Gabon, Russia. Subtype H: D.R Congo, Gabon, Russia, Central Africa. Subtype I: Cyprus, Subtype J: Central America, Subtype K: D. R Congo and Cameroon.

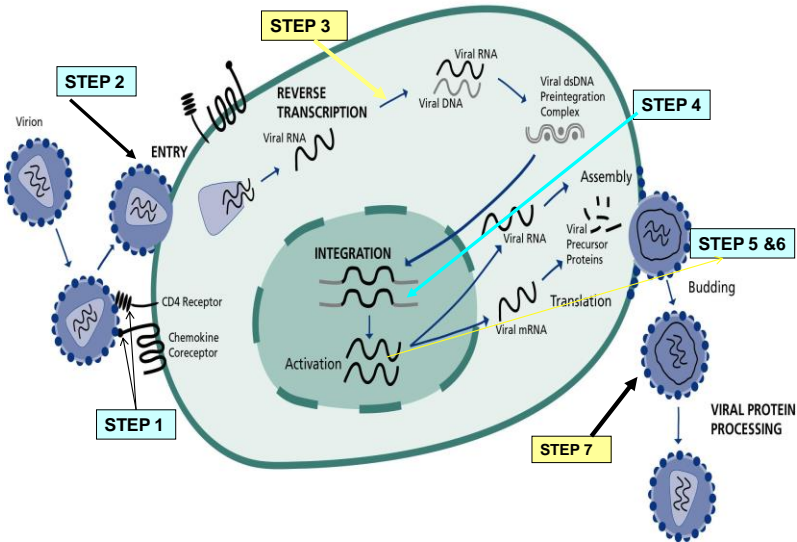
HIV-2 has eight subtypes (A-H), however, only subtypes A and B are in epidemic. It differs from HIV-1 in that patients have lower viral loads and slowly progress to AIDS.

**HIV super-infection:-** Occasionally, two viruses of different types or subtypes can co-infect a cell and mix their genetic material together to create a new hybrid virus<sup>13</sup>. The hybrid could be a weaker virus that is unable to replicate and dies out, or could be a strengthened virus that has become more virulent and less susceptible to ART. Importance of HIV super-infection is that it makes HIV diagnosis, its treatment and vaccine development very difficult to achieve.

**How HIV causes AIDS:-** HIV binds to CD4 receptors on the T-lymphocytes and other CD4 bearing cells via a tentacle called glycoprotein 120, figure 1. It then fuses and penetrates the host-cell membranes using chemokine-receptor; CCR5 or CXCR4. Other receptors such as: CCR3, CCR2, CCR8 and CX3CR1 could also facilitate HIV fusion and entry into the host cells<sup>14</sup>. Upon entry into the host cell, HIV will hide its genetic codes in that of its host, and when the host cell tries to make its own proteins, it will make new viral particles as well. These processes are facilitated by HIV reverse transcriptase, integrase and protease enzymes<sup>1</sup>

The newly produced daughter HIV will infect new CD4 cells which will in turn repeat the reproductive cycle. By estimates, more than  $10^{10}$  viruses are produced per day and about  $10^9$  CD4 cells are destroyed<sup>9</sup>. CD4 cells will be depleted to a point ( $<200\text{cells}/\text{mm}^3$ ) where it can no longer coordinate the patient's immune system. This is the stage of AIDS. At this point patients are susceptible to varieties of opportunistic infections (OIs) and some HIV related cancers.

Figure 1. HIV replication cycle



Courtesy- IHVN

**Post exposure prophylaxis:** During the first 24 hours of exposure to HIV, the virus is captured by nearby dendritic cells in mucous membranes and skin. The captured virus will be quarantined in the nearest regional lymph nodes for about five days before being released to commence uninhibited replication.

The knowledge of the time taken from exposure to the virus to its liberation to have unrestricted proliferation forms the basis of HIV Post- exposure prophylaxis (PEP). Early initiation of PEP could therefore prevent establishment of HIV infection in exposed individuals by the use of combination antiretrovirals (cARVs). This will prevent HIV from establishing footholds in the lymph nodes where it was earlier detained. Mr. Vice-Chancellor Sir, one hundred and twenty-nine health care providers, rape victims and others have been exposed to blood and body fluid of HIV positive patients in the last 10 years. They included; 22 medical students, (17%); 57 medical interns, (44.2%), 10 registrars, (7.8%), 3 consultants (2.3%), 13 nurses (10.1%), 21 rape victims, (16.3%) and 3 human bites (2.3%). All of them received a month course of PEP within 72 hours of exposure. None except one house officer who declined to take PEP as prescribed became seroconverted to the virus.

### **Why HIV cannot be eradicated from human body for now**

CD4 bearing lymphocytes and monocytes lineage are the principal targets of HIV. These cells have been shown to support HIV replication in infected patients. During replication however, a small percentage (< 0.01%) of CD4 cells enter into a post-integration latent phase and represent the main reservoir of HIV in human host. These resting cells are unaffected by the ART because they replicate at a very, very low-level. They serve as the refuelling points for HIV upon discontinuation of ART<sup>9</sup>. HIV infects a variety of other cells, which could not support its replication because they express small amount of CD4 molecule and its coreceptor on their surfaces<sup>15</sup>, table 1. Among these are; blood cells comprising circulating dendritic cells, epidermal langerhans; megakaryocytes; eosinophils; brain cells comprising astrocytes; oligodendrocytes; microglial cells; CD8- T cells; B cells; NK cells; renal epithelial cells; cells lining female genital tract; cells lining intestinal mucosal such as enterochromaffin, goblet, and columnar epithelial cells and cells from a variety of other organs, such as liver, lung, heart, eye, prostate, testes, and

adrenal gland. These cells represent additional HIV reservoirs for repopulating the body whenever, its stores of HIV are depleted<sup>16</sup>

**Table 1. Cells susceptible to HIV infection<sup>16</sup>**

System	Cells
Blood cells	CD8+ T-lymphocytes, Macrophages/Monocytes Dendritic cells, B-cells, NK cells, Stem cells and Megakaryocytes
Brain cells	Microglia, Astrocytes and Oligodendrocytes
Intestinal cells	Columnar epithelial cells
Others	Kupfer cells (liver), Synovial cells (joint) and renal epithelial cells

Clinical course of HIV infection without treatment :- After a few days of restriction in a regional node following exposure, the virus will undergo rapid replication for about 2-6 weeks before the onset of an immune response and clinical illness. This is seroconversion stage and lasts from 1-2 weeks. It could pass unnoticed or manifests non-specifically with fever, myalgia, arthralgia, and sore throat as well as, conjunctivitis, stuffy nose, and body rash. These manifestations resolve as antibodies to the virus become detectable in the serum. Thereafter, patients enter the first stage of chronic HIV infection, which is often asymptomatic but some patients may have a generalized lymphadenopathy (PGL). At this stage, viral load is low and CD4 count is above 500cells/mm<sup>3</sup> but will start to drop with course of time. This phase could last from months to years.

The second and third stages herald the onset of symptoms of HIV/AIDS. As the CD4 count drops between 350 and 500cells/mm<sup>3</sup> mild infections, particularly of the skin and mucosal surfaces such as rash, oral thrush, herpes zooster and stomatitis will start to appear, figure 2. Over time, these infections will persist or increase in severity. With further reduction in immunity; CD4 count between 200-350cells/mm<sup>3</sup>

patients may develop features of slim disease i.e. severe weight loss, diarrhoea and fever. In addition, patients are now more susceptible to severe bacterial pneumonia and tuberculosis<sup>17</sup>.

**Figure 2. Some Clinical Manifestations of HIV infection**



The fourth stage is that of AIDS, patient's CD4 count is less than  $200\text{cells}/\text{mm}^3$  and HIV related infections and cancers are quite common. In our experience, majority of our patients present at this stage. Of 167 newly recruited PLHIV in 2005, over 85% of them fell within stages 3 and 4 of the WHO clinical staging for HIV/AIDS<sup>18</sup>

### **Modes of presentation**

Because infectious diseases such as tuberculosis<sup>19</sup>, salmonellosis and bacterial pneumonia<sup>20</sup> as well as malaria, kala-azar<sup>21</sup> and syphilis<sup>22</sup> are endemic in this environment, they often complicate the course of HIV/AIDS in majority of our patients.

Upper respiratory tract and otorhinolaryngological features are equally common. In a prospective study of 100 newly diagnosed HIV positive patients in 2010<sup>23</sup>, 82.8% of the patients had clinical features of ear, nose and throat diseases that included chronic sinusitis, 45.6%, recurrent nasal discharge/obstruction, oro-pharyngeal candidiasis 44.9% and serous otitis media 21.5% in that order.

Skin lesions of various types and shades were many a times the first indicator of the presence of HIV infection in a number of our clients. A prevalence rate of 71.8% of dermatological lesions was found among 160 newly diagnosed adult HIV/AIDS patients in an hospital based study in 2013<sup>24</sup>. The commonest lesion was pruritic papular eruptions 33.9%, seborrheic dermatitis 17.4%, herpes zoster 10.4% and tinea versicolor 5.2%. Other skin lesions such as; psoriasis, scabies and warts as well as cutaneous Kaposi's sarcoma were found in small percentages.

Opportunistic infections (OIs) heralding or complicating the course of HIV infection varies widely. A study conducted in Ilorin to determine the spectrum of OIs amongst 293 PLHIV in 2006<sup>25</sup>, showed that 70% of the patients had infectious disease of different types and severity comprising in a descending order; bacterial infection; 41.2%, fungi infection; 33.8%, viral infection; 11.8% and parasitic infestations; 8.8% of the cases.

On a case by case basis, about one in four (22.6%) PLHIV were admitted for in-patient care in 2006 because of bacterial pneumonia<sup>26</sup>. *Streptococcus pneumoniae* and *Klebsiella pneumoniae* were the responsible bacteria in the early course of immunosuppression; CD4 350-510cells/mm<sup>3</sup>. However, fastidious bacteria like *Staphylococcus aureus*, *Pseudomonas aeruginosa* and *E.coli* were recovered from those with advanced immunosuppression<sup>27</sup>; CD4 30-160cells/mm<sup>3</sup>.

Intestinal parasitic infestation is a common complication of HIV/AIDS, with a prevalence rate of 87.8%<sup>28</sup>. It commonly manifests as chronic diarrhoea with severe malabsorption but sometimes presents as acute diarrhoea with a life threatening dehydration, uncommonly it could be asymptomatic. Implicated parasites from our study were protozoans such as *Cryptosporidium* spp, *Cyclospora* spp and *Isospora belli*. As well as helminthes such as *Strongyloides stercoralis*, *Giardia lamblia* and *Ascaris lumbricoides*. *Toxoplasma gondii*, an animal coccidian parasite that causes toxoplasmosis, is endemic in our community but uncommonly causes disease in man except in people with immunosuppression. It was found in 41.1% of

symptomatic PLHIV in a study that involved 180 HIV-seropositives and seronegative controls in 2009<sup>29</sup>.

Initially hard to isolate AIDS-defining cryptococcosis, cryptosporidiosis and microsporidiosis are now being isolated from PLHIV. We have established these special pathogens to be endemic in our environment if only we have the means to diagnose them. *Cryptococcus neoformans* was one of the common life threatening neurologic complications of AIDS in one of our series<sup>30</sup>. Similarly, a filamentous fungus, *scopulariopsis* species was isolated and documented as the first case of scopulariopsis associated meningitis in an HIV infected patient in Nigeria in 2003<sup>31</sup>. Screening for these fungi is now a routine for all HIV/AIDS patients presenting with nervous system symptoms.

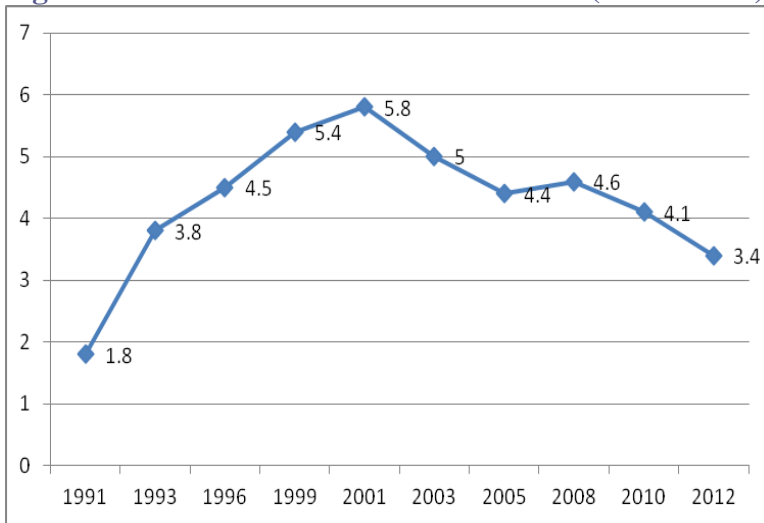
A comparative study on *Microsporidium* recently showed Microsporidiosis to be a cause of chronic diarrhoea amongst HIV/AIDS patients and a cause of systemic symptoms such as malaise, rashes and cough in advanced HIV illness<sup>32</sup>. Likewise, symptomatic Cytomegalovirus infection as indicated by elevated IgM antibody has been found to be relatively common; (11.1%) amongst PLHIV who have very low CD4 count<sup>33</sup>.

**Untimely hospital presentation:-** Most of our PLHIV presented to the hospital very late into their illnesses often with severely destroyed immunities. The average pre-treatment CD4 cells count in them was low; 194.9/mm<sup>3</sup> this was even lower; 117cells/mm<sup>3</sup> when they had a complicating OI<sup>18</sup>. Unfortunately, delayed hospital presentation was poorly correlated with survival. The documented average survival of PLHIV who had to be admitted in to the hospital in 2006 was 2 to 12 weeks from the time of diagnosis to death. Factors responsible for this were ignorance, fear of stigmatization and discrimination<sup>25</sup>.

### Status of the epidemic in Nigeria

The first case of AIDS in Nigeria was reported in 1986. Six years later in 1991, national spread of the disease was determined for the first time. Between 1991 and 2001, there was a rise in the seroprevalence rate from 1.8% in 1991 to 5.8% in 2001. It declined to 5.0% in 2003 and to 4.4% in 2005. There was a small rise in 2008 to 4.6% and then declined to 4.1% in 2010. It has even declined further to 3.4% in 2012<sup>34</sup>, figure 3.

**Figure 3 - National HIV Prevalence Trend (1991 - 2012)**



With an estimated population of 162,265,000, Nigeria is the most populated country in Africa, the continent that carries the globe’s heaviest burden of HIV/AIDS. About 3.5million Nigerians are currently living with the virus. This ranks Nigeria as the third most heavily burdened country after India and South Africa. Although the national median prevalence of HIV is reducing in recent years, the



reality is; the absolute number of people living with the virus has increased by almost half a million people in three years. This is because of the aggressive case detection drive of the programme executors. AIDS related death has also increased in the same period because the country is behind target in most success indicators for HIV treatment. For example only 1 in 3 (450000 of 1.5million) Nigerians living with HIV/AIDS and in need of ARV gets it, only 18% of pregnant women receive prophylaxis for PMTCT<sup>34</sup> and there are no group-specific HIV preventive measures in place for the most at risk members of the society such as the female sex workers (FSWs), injection drug users (IDUs) and men who have sex with men (MSM)<sup>35,36</sup>.

**New HIV infection :-** The incidence of new infection is rising and becoming worrisome in Nigeria. New infections are recorded largely amongst married sexual partners who are not ready to let go of each other even long after marriages to different spouses. Here the use of condom is low and the risk of transmission is high. Some high risk groups of the society also contribute significantly to the rising rate of new infection. They are hotel and road side based FSWs. They and their clients together with their clients partners contributed about 40% of the new infections in 2010. MSM and IDUs and their partners also contributed 10% and 9% of annual new infections during the same year<sup>37</sup>.

**Impact of HIV/AIDS:-** The impact of HIV/AIDS is felt at three interrelated levels: the families, communities, and the nation at large. The major impact is the loss of a loved one with its attendant emotional, social, and economic consequences. The grieving family will have to adjust to the change in the family structure. A young widow will now head the household for a

while until herself already infected becomes sick and less active. The responsibilities of the family will then fall on a grandmother who herself is a dependant. The children will become orphan and the household would dissolve from HIV induced poverty. The children will drop out of school and be applying for jobs as Juveniles. If lucky to be employed will be a cheap labour engaged in menial jobs.

Community impact of HIV is better appreciated in a closely knitted traditional Nigerian farming, animal rearing or fishing communities. Here the local economy of the community is dependent on the output of each household and when two or more members of such households are not healthy, the local economy will also not be healthy. Already, communities across some central parts of Nigeria where the state prevalence is quite high are facing a grim reality that may translate to declining standards of living. Here the state government has taken up the challenge and trying to reverse the ugly trend. Nationally, HIV/AIDS has taken its toll on the citizenry, it has deprived some Nigerian children of their parents, some regional economy of scarce skills and the nation of a generation in the prime of their useful working lives.

**What we are doing to care for PLHIV :-** Management of PLHIV started in 1999 under Dr. P.O. Olatunji of the Haematology department three years before UITH was named in 2001 as one of the 25 pilot centres for the FGN sponsored ARV trial programme. Dr. P.O. Olatunji (now Prof) was the Principal investigator and I was his deputy. A little above 50 patients were recruited for the exercise. By 2007 we had 943 adults and 35 children on active care. The figure increased to 1800 patients six months after we had support from the Institute of Human Virology, Nigeria (IHVN). The programme has however scaled up and today we have well over 5800 PLHIV accessing care from UITH. Our services include:

**HIV Counselling and Testing (HCT)** – which could be client or health care worker initiated. This is an essential component of HIV/AIDS care that helps to reduce risky behaviours and aids early detection and management of new infections. Individuals are counselled before and after the test. The courage of our clients is routinely commended for presenting for the test and their degree of vulnerability is explored to know areas of their social life to emphasise on during post-test counselling. Thereafter they are educated on measures of HIV prevention. Condoms are provided free. It use is demonstrated, and they are encouraged to use it during social sex. Clients are allowed to ask questions and encouraged to join our support groups if tested positive.

The level of public awareness of HIV/AIDS in the state is quite impressive, as many people (94%) know the routes of transmission of the virus, namely; unprotected sex, transfusion of an infected blood and accidental pricks from contaminated needles and sharp objects and from infected mother to child. They equally know that the virus is not contracted from touching, hugging, or sharing of household utensils or use of public toilets<sup>38</sup>. Despite this high level of AIDS-related awareness, majority of the populace have a very poor personal risk perception of acquiring the infection. Only 3.9% consider themselves at risk of contracting HIV and 60% are not ready to have HCT.

**HIV Status Disclosure:** Status disclosure is a strong prerequisite for a successful HIV/AIDS control programme. Clients are encouraged to disclose their status to spouse or a trusted family member. This may encourage them to seek testing or change from risky behaviour. In addition, disclosure encourages adherence to medications. However, when we assessed the disclosure rate amongst our patients 5 years after the services became available, the result was very poor, only 39.5% disclosure rate<sup>25</sup>. This

was far off the average mark of 79.0% projected for the developing world by WHO<sup>39</sup>.

Again, identified reasons for this low disclosure rate were fear of discrimination, stigmatisation and shame of being labelled sexually loose, fear of possible violence, outright rejection or divorce especially among the females in polygamous marriages and future difficulties of getting suitors for the singles amongst them.

The problem of disclosure was partly resolved by the clinic's firm request for each of our clients to have a treatment support partner who would be in the know of their status and give necessary supports and the employment of treatment support specialists (TSS) by the UITH's management who were role-playing positive leaving with HIV/AIDS at every clinic session. With these measures in place along with sustained community awareness campaign against discrimination, a far higher disclosure rate of 70% was recorded six years later<sup>40</sup>.

**HIV Care and Support:-** Psychological supports and reassurances are provided to our patients to cope with the news of being newly diagnosed HIV positive. They are taught how to positively live with the virus especially prompt report and treatment of HIV related illnesses and healthy life style. Water guard, insecticide treated mosquito nets, buckets and water jar are provided to the newly diagnosed patients and to those on follow up care; water guard and unlimited supply of condoms are provided at each clinic visit. PLHIV are encouraged to join and regularly attend one of our six support groups' meetings (Morning Stars, Save Lives, Anuluwa, Golden Women, Love and Care and Kiddies Club). They are encouraged to speak out on issues bordering them so that solution could

be proffered. By this we have dispelled some misconceptions and myths about HIV/AIDS. PLHIV now know that; their infection was not an affliction from their enemies, with ART their infection is no longer a death sentence and young ladies living with the virus are assured of having HIV seronegative babies if they apply themselves strictly to our clinic rules.

**Provision of adults ARV care.** For easy understanding; PLHIV could be grouped into three: asymptomatic group; (WHO stage 1), symptomatic group with early features of HIV infection; (WHO stages 2 & 3); and those with established AIDS defining illnesses; (WHO stage 4). What is common to all these groups is a persistent viral replication with varying degree of CD4 cells depletion. The aim of management in all the groups therefore is to reduce viral replication to as low a level as possible and maintain it there.

**Treatment of asymptomatic group** consists of advice on good nutrition, prevention of HIV transmission to others and follow-up counselling. PLHIV are enjoined to take locally available, cheap nutritious foods such as grains, legumes, lots of fruits and vegetables and poultry in adequate quantity. Courtesy of IHVN, patients are routinely given action meal supplements. This is a blend-in high energy meal from maize, soya beans and, groundnut that is fortified with minerals and essential vitamins.

Further transmission of the virus to others is discouraged by advising PLHIV to voluntarily abstain from sex or be faithful to their spouses or regularly use condoms if the earlier two options are difficult to adopt. To this end, free condoms are available at every clinic visit to all our clients and to people on courtesy and social visits to our clinic.

To prevent mothers-to-be from infecting their unborn babies, they are encouraged to get pregnant **only** when their CD4 cells count is well above 500cells/mm<sup>3</sup>, register early for antenatal care with our PMTCT team and report early to the hospital once they fall in to labour. By these measures, the mother is assured of a continuum of a comprehensive package of care from preconception stage with the adult ART team to having her pregnancy, labour and delivery supervised by the PMTCT team who will also offer her reproductive healthcare advice post-delivery. Her baby would also benefit from care and advice of the Paediatrics ART team on safer infant-feeding practices.

Mr. Vice- Chancellor Sir, to date and to our delight over **300** women who are positively living with HIV in our centre have been successfully delivered of HIV free babies.

**Treatment of symptomatic PLHIV** consists of prevention and treatment of OIs, provision of ARVs, and palliative care. OIs are common amongst PLHIV and they increase their morbidity and mortality. They are therefore better prevented or aggressively treated when diagnosed. The level of CD4 counts is an important guide for determining the time of initiation and discontinuation of prophylaxis against OIs<sup>41</sup>, table 2. We have observed a strong correlation between CD4 cells count and the level of immunologic competence of PLHIV as well as the OIs they are susceptible to<sup>42</sup>. Patients' with CD4 count less than 350cells/mm<sup>3</sup> are routinely commenced on co-trimoxazole prophylaxis.

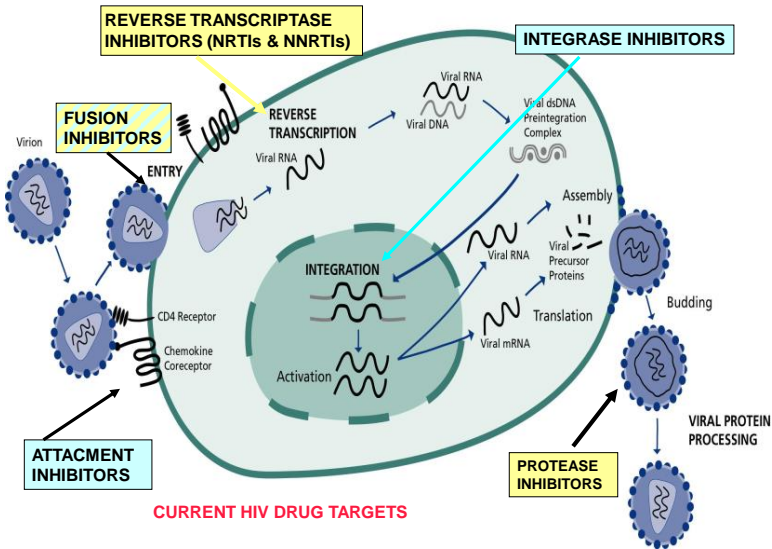
**Table 2. Correlation of infectious and non-infectious complications with CD4 cells counts<sup>41</sup>**

>500	Candida vaginitis
200-500	Pneumococcal disease, herpes zoster, Pulmonary TB, KS, thrush, cervical cancer
<200	<u>P</u> neumo <u>C</u> ystis jeroveci <u>P</u> neumoniae-PCP, miliary and extra-pulmonary TB, disseminated histoplasmosis, recurrent bacterial disease (e.g.pyomyositis), non-Hodgkin's lymphoma,
<100	Toxoplasmosis, Cryptococcosis, chronic cryptosporidiosis, microsporidiosis, Candida esophagitis/bronchitis
<50	Mycobacterium aviumintracellulare complex, Cytomegalovirus, CNS lymphoma, PML

**Provision of antiretroviral (ARV) drugs:-**

Knowledge of the life cycle of HIV has led to the development of drugs (ARVs) that target different stages of the viral reproductive process, figure 4. ARVs cannot cure HIV/AIDS, but can halt disease progression and allow the immune system to recover.

**Figure 4. Sites of action of different ARV drugs**



**Courtesy IHVN**

The drugs are taken daily for life. Its remedial effects are observed qualitatively as clinical improvement and could be measured quantitatively by estimates of both viral load and CD4 cells count. The results of the FGN pilot study to determine the effectiveness or otherwise of cARVs amongst Nigerian PLHIV in 2003<sup>18</sup> showed the drugs to be effective in ameliorating HIV/AIDS symptoms, improved their quality of life and survival. These drugs were well tolerated with minimal immediate side-effects. However, after over 12 years of clinical experience with these drugs, it is now evident that they could all cause albeit to varying extent long term physical and metabolic side effects<sup>43</sup>. That includes fat atrophy and hypertrophy in some areas of the body, glucose intolerance, hypercholesterolaemia and hypertriglyceridemia



**Indications to start ARV drugs.** National ART Guideline<sup>44</sup> recommends that therapy should be initiated in the following category of patients: 1. PLHIV with CD4 cells count less than 200cells/mm<sup>3</sup> with or without symptoms, 2. Symptomatic PLHIV with CD4 count less than 350cells/mm<sup>3</sup> and 3. Patient with an AIDS-defining illness. Such a patient will have clinical (OIs, psychiatric illness, pregnancy, alcohol) and laboratory [complete blood count, liver and renal function tests, serum glucose, lipid profiles and viral load] assessments to determine the appropriate ART that would suit him/her. Two Nucleoside reverse transcriptase inhibitors (NRTIs) are usually combined with a Non-nucleoside reverse transcriptase inhibitors (NNRTI) or a Protease inhibitor (PI), or an integrase inhibitor (II) to make up a triple drug regimen called Highly Active Antiretroviral therapy- HAART. See table 3 below

**Table 3. Classes of Antiretroviral Drugs<sup>44</sup>**

NRTIs	NNRTIs	PIs	IIs	FIs	EIs
Zidovudine	Nevirapine	Atazanavir	Raltegravir	Enfuvirtide	Maraviroc
Didanosine	Efavirenz	Saquinavir	Elvitegravir		
Lamivudine	Delavirdine	Indinavir	Dolutegravir		
Abacavir	Etravirine	Nelfinavir			
Emtricitabine	Rilpivirine	Lopinavir			
Tenofovir		Darunavir			
Zalcitabine		Tipranavir			

**Adherence counselling:** Adherence is to behave as agreed with health care provider i.e. come to 100% of appointments and to take 100% of ARV medications (in correct dose; correct frequency and at the correct time). This exercise involves active participation of the patient, their health care providers, and treatment support partners (family, friend or HIV<sup>+</sup> peers). Adherence is the key determinant of ART success. It leads to a long-term decrease in viral load with a reciprocal improvement in body immune system and a lower risk of OIs.

However, to achieve these, a minimum of 95% or greater adherence is needed from PLHIV i.e. a patients can only miss 1½ days of ARVs which is a maximum of 3 doses in a month. Adherence rate amongst adult patients is quite good, over 70.8% of 253 PLHIV evaluated in 2010 had excellent adherence<sup>45</sup>. Less than 10% (24 patients) of the studied population had poor adherence.

Adherence is however difficult to achieve in geriatric HIV infected patients because of the peculiarity of this age group. A few elderly patients, 73years and above that were treated in our facility in 2013 had initial perfect adherence until after sometimes, they started to deny their status and stopped taking ARVs<sup>46</sup>. To achieve near 100% adherence therefore, we anchor ARV intake to our patients’ daily routine such as morning and evening prayer time or breakfast and dinner time. We also encourage adherence by asking them to find a treatment partner who will be monitoring their drug intake. Pill boxes, calendars when available are provided to check off dosing and avoid dose confusion, figure 5.

**Figure 5. Three samples of pill boxes**



**Home based care:-** This is care given to PLHIV in their homes. It is about comfort measures such as: proper positioning, aiding mobility, bathing, skin care, guidance and support for adequate nutrition. Others include relieving pain and itching, treating diarrhoea and vomiting. In serious cases, patients are referred to the hospital for appropriate treatment. In addition, we educate family caregivers to protect themselves from contact with patients’ blood and body fluids by wearing gloves, using

disinfectants and detergents to clean up the patient and the linen and to regularly wash their hands with soap and water.

**Palliative care.** This is an essential component of care for PLHIV that aims to improve the quality of life of the patient and reduce the stress of care on both the health care worker and patient family care giver. It is provided as symptomatic care to relieve variety of discomforts PLHIV may experience during the course of their illness - such as pain, itching, diarrhoea, cough, nausea and seizures etc. Medications that **USED** to be available for Palliative care are as shown in the table 4 below.

Table 4. Medications for palliative Care

Medical condition	Available medication
Analgesics	Pethidine and amitryptiline
Antidiarrheals	Loperamide, codeine
Anti allergic	Calamine lotion chlorpheniramine
Anti-nausea products	Metoclopramide, promethazine
Antitussive- Non specific cough	Bromhexine, Codeine
Anticonvulsants	Diazepam, cabamazepine
Neuroleptics	Chlorpromazine, Haloperidol, Amitryptiline

Pain is a common problem amongst PLHIV, it was present in 27.8% of our client and its intensity was of moderate to severe in 1 out of every 4 cases. Commonly affected parts of the body were the lower limbs; 44.4%, head and neck, and the abdomen; 32% each. Its causes are diverse but respond satisfactorily to palliative measures<sup>47</sup>.

**Match making:** This is one of our success stories that is carried out in our support group meetings. We encourage marriages between seropositives men and

women and for this we often break the ice and facilitate interactions amongst our patients who are singles, widower or widow, and divorcee. We encourage commitment from both sides and once they understand each other's language we step aside. By discouraging marriages between serodiscordant men and women, we are reducing viral transmission, reducing the number of infected babies and reducing the number of orphans. Mr. Vice-Chancellor Sir, we have successfully arranged 27 marriages between seropositive couples amongst our clients.

**Stigma and discrimination:** Stigma is a set of attributes or characteristics used to qualify HIV infected persons in a way that devalues them. Stigmatization of PLHIV is commonplace in Nigeria. Majority see immoral behaviour as the cause of HIV infection and this affects societal attitudes towards PLHIV. They are denied social interactions, some are denied employment or lose their jobs and are sometimes barrier nursed in the hospitals. Stigma and discrimination are triggered by poor knowledge of how HIV infection is acquired and transmitted. This often leads to speculation and build-up of myths about the disease. The danger is; stigmatization of PLHIV will deter people from going for HCT and seropositives people will be infecting others ignorantly. It also delays PLHIV from having early diagnosis and initiation of treatment. This was my experience with a professional colleague who had HIV/Syphilis co-infection in 2007. Because of the fear of stigma in his place of work, diagnosis was delayed and he developed neurological extension of syphilis- neurosyphilis. When diagnosis was finally made and treatment instituted he got better and became stable but because of fear of negative reactions from his colleagues, he abandoned hospital care for a prayer house therapy, which unfortunately cost him his life<sup>48</sup>.

**Provision of DOTS services:** Tuberculosis (TB) is a chronic bacterial infection caused by *Mycobacterium*

*tuberculosis* (MTB) complex, a strict aerobic bacilli. In most infected individuals, the bacilli remain dormant for years because the host's cell-mediated immunity will prevent it from developing into active TB. HIV however, targets and depletes cells responsible for this protective immunity. In Nigeria now, HIV infection is the most potent risk factor for reactivation of latent TB focus and progression of new infection to active TB<sup>49</sup>. About 27% of all TB cases in Nigeria are HIV co-infected and it is the leading cause of death amongst them<sup>49</sup>. Early in the course of HIV, TB manifests as a localized pulmonary disease. However, as immunity declined clinical presentation becomes atypical, TB now presents as extrapulmonary, miliary or disseminated disease<sup>50</sup>.

Our local data here in Ilorin suggest a rising prevalence of TB in this environment. This could be a reflection of TB prevalence in the country at large. Salami et al<sup>51</sup> recorded a prevalence rate of 9.2% in 2002 which represented a 700% increase over a rate of 1.6% recorded 5 years earlier in a sentinel survey for HIV/TB co-infection in Kwara State.

The rate of HIV/TB co-infection has also been rising over the years largely because of the HIV induced immune dysregulation that enhances rapid progression of both diseases in the co-infected patients. In 2001, the prevalence rate of HIV/TB co-infection was 12.6%<sup>51</sup>. Sixty per cent of the affected adults were below 50 years of age; (15-44years) the same age group that is most vulnerable to HIV infection. Five years on in a study that reviewed the pattern and trend of HIV- associated TB in Ilorin<sup>52</sup>. The rate of HIV/TB co-infection had increased to 40% amongst 744 TB cases seen over that period; 79% of these were HIV/PTB and 21% were HIV/EPTB. The annual case detection rate had more than doubled to an average of 47 new cases diagnosed per year as against 21 new cases per year in the previous nine years (1991 to1999). This observation is a wake-up call if epidemic of HIV/TB co-infection is to be prevented in the country in tandem to the prevailing wave of HIV epidemic.

**Laboratory diagnosis-:** we usually make a presumptive diagnosis of TB from microscopic observation of AFB in the smear of specimens from infected tissue/fluid using Ziehl-Neelson (ZN) technique. Low sensitivity however, is the major drawback of this method. In a study to determine, the incidence and distribution of smear positive TB in UITH in 2006<sup>53</sup>, 17,535 pooled specimens of sputum; (97%); urine, (1.56%); and pleural aspirates, (1.0%) were reviewed. Only 10.6% of these were smear positive for AFB. However, our newly acquired fluorescent microscope (FM) - courtesy of IHVN that uses auramine-rhodamine stain is a much sensitive alternative. It picks AFB where ZN stain returns a smear negative result. Of the 110 ZN-stained smear negative reports in 2012, 67% became positive with FM (Salami, et al unpublished). It is important to acknowledge the role of x-ray imaging in the diagnosis of paucibacillary HIV/PTB co-infection and difficult to diagnose extrapulmonary TB<sup>54</sup>, figures 6A & 6B since high-tech diagnostic tools such as polymerase chain reaction and nucleic acid line probes are not yet available in our clinic.

Figure 6A. Bilateral soft nodular opacities worse in both lower lobes

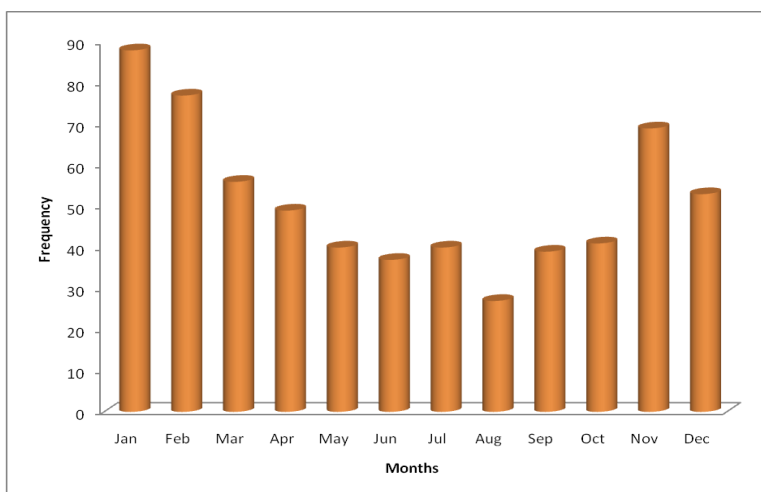


Figure 6B. right sided pleural effusion with nodular opacities



Smear isolation of tubercle bacilli is essential for epidemiological monitoring of TB. The proportion of sputum smear positive cases is calculated out of all new pulmonary cases

and has expected value of 55%-70%<sup>55</sup>. It is a measure of a country's national TB case detection rate (CDR). The lower it is the higher the prevalence of TB in that region. Nigerian national CDR is low, 13%-16%<sup>49</sup> perhaps because of the inadequate (45%) DOTS coverage. A slightly better local annual CDR of 33.2% has however been established for UITH<sup>53</sup> with a bimodal monthly distribution of cases, figure 7. The first peak was at the beginning of the year (January to March), due to increased bacilli transmission during the cold dusty weather of harmattan that commonly forces people indoors and in close contact for most of the time. The second peak was due to the annual end of the year (November to December) influx of people from different parts of the country for Xmas and New Year celebration as well as other festivities



**Figure 7: Monthly distribution of smear positive TB**

**Treatment of TB:** is implemented with a six-month regimen of four drugs comprising isoniazid, rifampicin, pyrazinamide, and ethambutol, all in mg/kg body weight of the patient. Anti-TB and ARV drugs are free and both are dispensed to co-infected patients during a single clinic visit. Anti-TB

chemotherapy is very effective amongst HIV negative TB patients. Sputum conversion is rapid and cure rate is good when diagnosis is made early and DOTS initiated on time at appropriate dosage and for correct length of time<sup>56</sup>.

However, treatment outcome was not always good amongst HIV/TB co-infected patients because of the increased pill burden and the overlapping toxicities of anti TB and ARV drugs on the patients. In a review of management outcome of 1,741 TB cases in 2003<sup>57</sup>. Only 43.7% cure rate was achieved, 44.2% of the cases defaulted treatment and 11.6% died. This cure rate was quite low when compared to the WHO expected target of 85% from a good and effective TB control programme. The national cure rate only fair a little better ranging from 66% in 2003 to 72% in 2010<sup>58</sup>.

In managing dual HIV/TB co-infection, rifampicin component of anti TB regimen significantly reduces the bioavailability of some ARV drugs (PIs and NNRTI) so much that these ARV drugs would be ineffective in suppressing HIV replication, table 5.

**Table 5. Rifampicin decreases blood levels of protease inhibitors and NNRTI**

Protease inhibitors	Effect of Rifampicin
Indinavir	↓ 90%
Nelfinavir	↓ 82%
Amprenavir	↓ 81%
Saquinavir	↓ 80%
Lopinavir/ritonavir	↓ 75%
Ritonavir	↓ 35%
NNRTI	
Nevirapine	↓ 37% - 58%
Efavirenz	↓ 13% - 26%

However, to limit mortality amongst HIV/TB co-infected patients, Anti TB and ARV drugs have to be co-administered in the best acceptable combinations with the aim of managing any resultant adverse effects. This is mild



in most cases, but could be severe in a few others. In our experience, hepatotoxicity, central nervous system toxicities such as paraesthesia, drug induced psychosis and seizure) and cutaneous hypersensitivities such as urticaria, Steven Johnson syndrome, toxic epidermal necrolysis and exfoliative dermatitis) were common adverse drug reactions among TB/HIV co-infected patients<sup>59</sup>.

**Occupational risk of HIV exposure** :- Health care workers (HCWs) are at risk of contracting HIV infection from occupational exposure, if necessary precautions are not observed. Such risks could result from contact with an infected blood, use of unsterilized instruments and accidental pricks from a contaminated needle. We evaluated this risk amongst 300 HCWs of UITH comprising 115 Doctors, 150 Nurses and 35 Laboratory scientists who were directly involved with patients care including PLHIV<sup>60</sup>. Results showed that 78% had been using hypodermic needles on PLHIV, 84.6% had been handling patients' tissues/body fluids and 90.7% had been taking deliveries of seropositive and seronegative mothers. Above all 57.7% of the respondents have had accidental needle stick injury and spillage of patients' body fluids. Despite this level of risk of exposure, just about half of them were willing to know their HIV status and only 41.4% had ever had voluntary HCT.

Health-care workers (HCWs) are also at a higher risk of acquiring work-related TB because of their repeated contacts with different category of patients including TB patients during the course of their hospital care. Hospital related TB was diagnosed amongst 1.5% of the 2173 staff strength of the UITH in 2007<sup>61</sup>. Pulmonary TB was the commonest (78.1%), followed by TB adenitis (12.4%), disseminated TB (6.3%) and TB spine (3.1%). HIV infection was the commonest risk factor.

## **Programme assessment**

It is now a common knowledge that cARV drugs have given PLHIV a favourable outlook the world over. HIV treatment programme has therefore, scaled up across the land. We now have about 5800 patients in care, and it will be immodest to say that there are no challenges of standards of care. A quality assurance committee was therefore set up in April of 2009 with the responsibility of periodically evaluating service deliveries in the clinic and making appropriate recommendation for improvement if inadequacies are observed. The quality of clinical and laboratory services rendered to a cohort of 440 patients who had been in care for more than a year was conducted in 2010<sup>62</sup>. The result was a wake-up call if quality is to be ensured. At recruitment into care; patients' weights, severity of their diseases and laboratory profiles were assessed in about 80% of them. However, during follow-up visits measurement of weights declined by 20%, CD4 cells count was done in about 52% of the patients. While liver and renal function tests and lipid profiles were only done in 31%, 32% and 36% respectively. Result of this poor self-auditing was not due to HCWs ineptitude but largely due to inadequacies of manpower to perform these tasks, energy to power the machines and, frequent brake down of equipment and repeated stock out of reagents/consumables for these tests. To ensure comprehensive quality of care therefore, needed supports and logistics should be provided to us in the HAART clinic.

**HIV cure:** The ultimate expectation of the world is to find a permanent cure for this disease. The reality for now is that this expectation may have to wait for a while, though a number of "functional cures" have been reported of late. None of these was a true cure but a preventive intervention by PEP in the case of the Mississippi baby born to an HIV positive mother who started cARV within 30hours of birth. The "Visconti cohort cure", was a case of rare genetic anomalies of high levels of

HLA-B\*07 and HLA-B\*35 alleles<sup>63</sup> that allow HIV infected patients to have a low viral load and host cells reservoir. The reality however, is that all the 14 French Visconti patients remained HIV seropositive till today but asymptomatic. The cure of the Berlin patient of his leukaemia and HIV infection after he received a marrow transplant from a CCR5 deficient donor was temporary. Other co-factors aside CCR5 have facilitated HIV's entry in to his cells and he is again symptomatic and back on ART.

Mr. Vice-Chancellor Sir, one of the realities of HIV infection is that once an individual is infected total eradication of the virus from his cells is not possible at least for now. To achieve a cure however, HIV in reservoir host cells will have to be activated and made susceptible to ART to ensure total viral clearance.

**Alternative therapy:** There have been several traditional claim of herbal cure for HIV/AIDS at local, national, and international levels. The reality is that there is no evidence that these herbs are effective in treating HIV infection. The immunopotency of alovera, moringa, ginsomin, beet root and many other herbs in restoring depleted CD4 cells in PLHIV have not been established. They should therefore not be taking at the expense of cARVs.

**Spiritual cure:** Nigerians are quite religious and they find great comfort from their Priests and Imams during chronic illnesses. These religious leaders provide good palliative care, but the ideas of abandoning ARVs for long weeks of prayer and fasting to eradicate HIV is nothing but outright deceit and suicidal engagement.

**HIV vaccine:** Because of the share number of HIV types, its subtypes and varieties of hybrids of the virus resulting from super-infection as well as its continuous mutations during replication, it has been difficult to get a fixed target on the virus

against which a neutralising antibody could be developed. Scientists have recently identified a constant non-changing spot on the viral structure tagged V1V2 site and are now developing antibodies against it. The preliminary observation is promising.

**In conclusion**, Mr. Vice Chancellor, as at today there is neither a cure for an established HIV infection nor a vaccine to prevent people from acquiring the infection. The reality on ground is that HIV/AIDS is no longer a killer disease but a chronic manageable one with the patient living a near normal life span, provided there is easy access to a good quality of care that include availability of ARVs, OIs drugs and palliative care and patient himself is living a healthy life style devoid of drug use and serious comorbidities.

**What need to be done (Recommendations)**

1. Nigeria has done well on the control of HIV infection; she has arrested and reversed the national seroprevalence rate from 5.8% to 3.4%. However, the country need to consolidate on this gain, reverse the rising rate of new HIV infection and ensure that PLHIV continue to live well and live long. To achieve these the country has to look inward and take full ownership of HIV care because aids from all the donor agencies will cease to flow by June of next year- 2015. The President has shown commitment in this regard by launching the comprehensive intervention package of 100 billion dollars
2. State government and local government councils should also set aside some HIV/AIDS intervention fund so that state and local government control agencies can get involved in clinical care of PLHIV.
3. HIV/AIDS treatment sites should be expanded to have a wider coverage of the population by involving private and corporate hospitals as treatment points for the

disease. This will ensure easy access of PLHIV to HAART services.

4. National orientation agency should advocate changes to some cultural practices that encourage transmission of the virus or serve as barrier to HIV prevention programme such as early marriages, taboo of sex education to children and non-acceptance of condom use in some quarters. Advocacies and involvement of the religious and traditional heads to help raise awareness about prevention programme of HIV/AIDS in religious gatherings; (churches, mosques, crusade and Eid-praying grounds), social gatherings; (marriages, thanks giving and reception parties) and in public places; (stadia, post offices and banks, ports)
5. Equally beneficial will be a one-on-one engagement of the high-risk groups with specific HIV prevention programmes rather than proclaiming punitive measures otherwise these people will withdraw to the background and will be practicing their trade with the attendant consequence of sabotaging the national preventive programme.
6. Government officials vested with responsibilities of coordinating HIV/AIDS care in the country should ensure prudent management of resources at their disposal.
7. Federal government should promulgate and enforce laws to destigmatise HIV/AIDS and encourage sex education at all levels of education in the country.
8. Well-meaning and public spirited Nigerians should be encouraged to set up foundations to fund HIV/AIDS treatment programme
9. Public and multinational corporations should be encouraged to assist FGN in financing HIV/AIDS care in the country from a small percentage of their annual profits.

**Achievement:** Mr. Vice- Chancellor Sir, I have supervised and co supervised Fellowship theses in respiratory and HIV related disciplines. I have contributed to hundreds of undergraduates and tens of postgraduate students realising their dreams. But my greatest achievement is that of restoring hopes and smiles to the faces of thousands of PLHIV in this environment.

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Mr. Vice-Chancellor Sir, I wouldn't have come this far in life if not for providence, I therefore give all the glory to GOD for writing my script of life this way and acting it out to my delight. I thank you Mr. Vice-Chancellor for the opportunity to present this inaugural lecture, I thank Prof. Y.A. Quadri for proofreading it and I thank the audience for their attention. Thank you all.



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