Research and Reviews: Journal of Medical and Health Sciences

HIV/AIDS: A Brief Overview

Prabhat Shukla*, Neeru Tyagi

Department of Pharmaceutical Technology, Meerut Institute of Engineering and Technology, Meerut, India

Short Communication

Received: 10/05/2015 Accepted: 29/05/2015 Published: 06/06/2015

*For Correspondence

Department of Pharmaceutical Technology, Meerut Institute of Engineering and Technology, Meerut, India.

Keywords: HIV/AIDS, Immune system, CD4 cell, Epidemiology

INTRODUCTION

AIDS (acquired immunodeficiency syndrome) is a disease caused by a virus called HIV (human immunodeficiency virus) [1]. It kills or damages the body's immune system cells. It is the most advanced stage of infection with HIV. AIDS was first reported in the US (United States) in 1981 and since then it became a major worldwide epidemic [2-6]. By killing or destroying cells of the body's immune system, HIV progressively destroys the body's ability to fight infections and certain type of cancers [7-10]. People diagnosed with AIDS may get life-threatening diseases called opportunistic infections. These infections are caused by microbes (such as viruses/bacteria) that usually do not make healthy people sick. HIV attacks the immune system by destroying CD4 positive (CD4+) T cells, a type of white blood cell that is essential for fighting off infections [3,11,12]. The destruction of these cells leave people infected with HIV vulnerable to other infections, diseases and other complications. A person infected with HIV is diagnosed with AIDS when he/she has one or more opportunistic infections, such as pneumonia or tuberculosis, and has a dangerously low number of CD4+ T cells (less than 200 cells per cubic millimeter of blood) [3.4,13]. Nearly 35 million people all over the world are infected with human immunodeficiency virus (HIV). Around 2 million people get infected with the virus each year and the pandemic continues to devastate despite three decades of our understanding of the pathogenesis [14,15]. The AIDS epidemic. in spite of considerable efforts continues to spread at formidable rate worldwide with an estimated 34 million people being infected with HIV at the end of 2010 [5]. The key step in the disease progression is viral binding to the host T lymphocytes and the entry [16]. Due to the constant mutation tendencies of the HIV virion it has become a big challenge to target this virus and eradication of the virus infection all together [12]. However, not everyone who has HIV progresses to AIDS.

EPIDEMIOLOGY of HIV/AIDS

HIV/AIDS is a global threat. All of the countries are affected by HIV/AIDS but the count varies from region to region [17-20]. Sub-Saharan Africa is the region that is most affected. In 2010, an estimated 68% of all HIV cases and 66% of all deaths occurred in this region. This implies that about 5% of the adult population in this area is infected. South Africa has the highest count of HIV infected individuals in the world. In South and South-East Asia HIV prevalence rate is less than 0.35 percent. The AIDS image in South Asia is dominated by the epidemic in Indian subcontinent. There is a rapidly growing epidemic of HIV in European Union, the rate of HIV infections began to grow rapidly from the mid-1990s [21-24].

HOW HIV is TRANSMITTED

Scientists believe that a virus similar to HIV first occurred in some populations of monkeys in Africa, where they were hunted for food. Contact with an infected monkey's blood during butchering or cooking may have allowed the virus to cross into humans and become HIV.

Person can be infected with HIV through contact with bodily fluids. HIV is found in the blood, semen, or vaginal fluid of someone who is infected with the virus. HIV cannot be transmitted through hugging, kissing, dancing or shaking hands-with someone who has HIV or AIDS. This is due to the fact that HIV cannot survive for very long outside of the body [25].

There are some of the common ways of HIV transmission [26-28]

Having unprotected sex: A person can become infected if he/she have vaginal, anal or oral sex with an infected partner whose blood, semen or vaginal secretions enter the body. The virus can enter your body through mouth sores or small tears that sometimes develop in the rectum or vagina during sexual activity. Also, engaging in activities like anal, vaginal, or oral sex with men who have sex with men, multiple partners, or anonymous partners without using a condom can lead to HIV transmission. Even though use of protective measures (mainly condoms), also don't guarantee 100% protection from HIV transmission [29-32].

Blood transfusions: Virus may be transmitted through blood transfusions. A study shows that individuals who received blood or clotting factor in United States from 1978 to 1985 have a greater risk of infection from HIV. Since then, American hospitals and blood banks now screen the blood supply for HIV antibodies. The rate of HIV transmission through blood transfusion is 100% [5,33].

Sharing needles: HIV can be transmitted through needles and syringes contaminated with infected blood. Local street sellers of syringes may repack used syringes and sell them as sterile syringes. For this reason, people who continue to inject drugs should get syringes from reliable sources, such as pharmacies or hospitals. It is important to know that sharing a needle or syringe for any use, including skin popping and injecting steroids, hormones, or silicone, can put you at risk for HIV infections. Tattooing or body piercing present a potential risk of HIV transmission. But, there is no significant evidence to prove the statement [34].

From mother to fetus: Infected mothers can infect their babies. According to Kourtis et al [35] approximately 4,00,000 children are infected with HIV in 2008. Mother-to-child transmission of HIV can occur during pregnancy, labour, delivery, or through breastfeeding. Breastfeeding is responsible for one third to one half of HIV infections in infants but, majority of infants are infected during delivery [36]. Various drugs have been developed to reduce the mother to child transmission (MTCT) of HIV. These drugs significantly reduce the risk of transmission of infection but do not completely eradicate it. Despite continuous researches the origin of HIV in the breast milk is not completely understood. One of the best approach to prevent HIV infection in infants, including transmission through breast milk, is to prevent HIV infection in young girls and women that have attained puberty. Also, educating them about safer sex, condoms, and diagnosis and treatment of sexually transmitted infections may reduce the risk of MTCT. Replacement feeding i.e. modifying feeding options of infants for HIV infected women may also reduce the risk of transmission. Commercial infant formula can be used for feeding infants with partial breast feeding [37-42].

COMMON MISCONCEPTIONS ABOUT HIV [43]

HIV cannot be transmitted through:

- Breathing the same air as someone who is HIV-positive
- Hugging, kissing, or shaking hands with someone who is HIV-positive (HIV may be found in saliva, but it is in too small amount to infect anyone)
- Sharing utensils with an HIV-positive person
- Using exercise equipment at a gym
- Mosquito bites

Antiretroviral therapy can decrease the amount of HIV in the body. But, it can still be transmitted to others.

FROM HIV INFECTION to AIDS

HIV is an enveloped RNA virus and is roughly spherical having a diameter of about 120 nm [5]. The viral core/capsid is usually bullet-shaped and is made from the protein p24 [44]. Inside of the core there are three enzymes which are required for HIV replication [34, 45,46]:

- reverse transcriptase
- integrase
- protease [47-51].

HIV's genetic material is enclosed within the core and it consists of two identical strands of RNA [52,53].

HIV can only replicate inside human cells. The process typically begins when a virus bumps into cells that have surface protein called CD4. The spikes on the surface of the HIV particle stick to the CD4 and allow them to fuse [54-58]. This results in releasing of the contents of HIV into the cell. HIV mainly infects helper T cells that are very important for the body's immune system. As HIV infects more cells, the immune system becomes weaker and thus results in AIDS. Once inside the cell reverse transcriptase converts the viral RNA into DNA. This is then transported to nucleus of human cell where it is inserted into the human DNA by the HIV enzyme integrase. At this stage the HIV DNA is known as provirus and the cell begins to die thereby, weakening the immune system.

Now, these proviruses remain dormant for a long time. But, after activation HIV genes works as human gene and secretes HIV proteins and enzymes which help in synthesizing new viral particles. After some time they are released from the cells by the process called budding. The enzyme protease then chops up the long strands of protein into smaller pieces, which are used to construct mature viral cores of new HIV particles. These newly formed HIV particles now infect new cells and same process repeats. In this way the virus quickly spreads through the body.

As a result of the above process the helper T cells die thus, weakening the immune system. At this stage, due to weak immune system various opportunistic infections attack the body and causes AIDS [59-62].

Common opportunistic infections that dominate in HIV infections are [63,64]:

- Tuberculosis
- Salmonellosis
- Candidiasis
- Meningitis
- Pneumonia [65]
- Lymphomas etc.

Symptoms of HIV infection

In the primary stages of HIV infection, most of the people have very few or no symptoms. But, after one or two months of infection, individuals may experience flu like symptoms which includes [66]:

- Fever
- Headache
- Tiredness
- Enlarged lymph nodes in the neck and groin area

These symptoms disappear within two weeks. People infected from HIV may have no symptoms for 12 yrs. or more.

During later stages of HIV infection individuals may have following symptoms:

- Rapid weight loss
- Recurring fever
- Profuse night sweats
- Extreme tiredness
- Prolonged swelling of the lymph glands
- Diarrhea
- Sores of the mouth, anus, or genitals

- Pneumonia
- Red, brown, pink, or purplish blotches on or under the skin or inside the mouth, nose, or eyelids
- Memory loss, depression, and other neurologic disorders [67]
- Kidney disease [68-70]

DIAGNOSIS and TREATMENT

HIV is commonly diagnosed by testing blood or saliva for antibodies to virus. However, these antibody tests may not detect HIV antibodies in someone who has been recently infected with HIV (i.e. up to 12 weeks). There are two methods by which HIV can be tested are ELISA (enzyme linked immunosorbent assay) and western blot [71-76].

Newer test that detects HIV antigen (a protein produced by the virus immediately after infection) confirms the diagnosis within the days of infection. This earlier diagnosis aids in taking extra precautions to prevent transmission of the virus to others and early treatment which may increase the patient's life time [77,78].

Various classes of drugs are used in the treatment of HIV these are [4,21,22,79-84]:

- 1. Entry Inhibitors: It interferes with the virus's ability to bind to receptors on the outer surface of the cell. When receptor binding fails, HIV cannot infect the cell. E.g. Fosfonet, Enfuvirtide etc.
- 2. Fusion Inhibitors: It interferes with the virus's ability to fuse with a cellular membrane, preventing HIV from entering a cell. E.g. Maraviroc, Enfuvirtide, Fostemsavir etc.
- 3. Reverse Transcriptase Inhibitors: It prevents the HIV enzyme reverse transcriptase from converting single-stranded HIV RNA into double-stranded HIV DNA.
- 4. Nucleoside/nucleotide RT inhibitors (NRTIs): They are faulty DNA building blocks. When one of these faulty building blocks is added to a growing HIV DNA chain, no further correct DNA building blocks can be added on thereby halting HIV DNA synthesis. E.g. zidovudine, lamivudine, emtricitabine, abacavir etc. [85-90]
- Non-nucleoside RT inhibitors (NNRTIs): It binds to reverse transcriptase, interfering with its ability to convert HIV RNA into HIV DNA. E.g. tenofovir, etravirine, efavirenz, nevirapine etc. [91,92]
- 6. Integrase Inhibitors: It blocks the HIV enzyme integrase, which the virus uses to integrate its genetic material into the DNA of the cell it has infected. E.g. raltegravir, dolutegravir etc.
- 7. Protease Inhibitors: It interferes with the HIV enzyme called protease, which normally cuts long chains of HIV proteins into smaller individual proteins. When protease does not work properly, new virus particles cannot be made. E.g. saquinavir, ritonavir etc.

CONCLUSION

Since its first clinical discovery in 1981, AIDS emerges as the global epidemic which has taken many lives of the people and continues to do so in the coming years [93]. It was believed that HIV was originated from primates in Africa, where they were hunted for food. Since then it continues to spread all over the world. The epidemiological distribution is not same for all the continents, yet it is one of the most dangerous and life threatening disease. Treatment options are available which can prolong the life of the patient but cannot cure the disease [94,95]. This is due to the complex structure of the HIV and rapid resistance to the drugs [96-99]. Now-a-days combination therapy [100] is given to combat resistance thereby, prolonging the life but, still cannot cure the disease [101,102]. In the end there is only one option left to be safe from HIV infection i.e. self-awareness and sex education. These also do not guarantee 100% safety but can limit the infection from spreading. As very well said that prevention is better than cure and so in the case of HIV same rule follows.

REFERENCES

- 1. Motilewa OO et al. A Comparative Study of Health Related-Quality of Life Among HIV Patients on Pre-HAART and HAART in Uyo South-South Nigeria. J Antivir Antiretrovir. 2015;7:060-068.
- 2. de Castro Castrighini C et al. Epidemiological Profile of HIV/Tuberculosis Co-infection in a City in the State of São Paulo, Brazil. J Antivir Antiretrovir. 2013;5:119-122.
- 3. Zerbato J and Sluis-Cremer N. HIV Infection of Naïve CD4+ T Cells: An Important Reservoir of Persistent HIV Infection? J Antivir Antiretrovir. 2013;S10- e001.
- 4. Ferir G, Palmer KE et al. Griffithsin, Alone and Combined with All Classes of Antiretroviral Drugs, Potently Inhibits HIV Cell-Cell Transmission and Destruction of CD4+ T cells. J Antivir Antiretrovir. 2012;4:103-112.
- 5. Wayengera M. Genomic-Regulation of Active Retroviral Elements as a Model for HIV Cure. J Antivir Antiretrovir. 2015;7:069-075.
- 6. Uddin SJ et al. In-vitro Antiviral Activity of a Novel Phthalic Acid Ester Derivative Isolated from the Bangladeshi Mangrove Fern Acrostichumaureum. J Antivir Antiretrovir. 2013;5:139-144.
- 7. Awasthi S. Immune Evasion by Persistent Viruses and Cancers: Blocking Evasion as a Rational Design to Treat Viral Infections and Cancers. J Antivir Antiretrovir. 2013;5:xxv-xxvi.
- 8. Li Y et al. Modulating Innate Immune Response to Combat Viral Infections-Use HCV as an Example. J Antivir Antiretrovir. 2013;5:xvii-xviii.
- Masekela R et al. (2013) Lack of Efficacy of an Immunomodulatory Macrolide in Childhood HIV-Related Bronchiectasis: A Randomised, Placebo-Controlled Trial. J Antivir Antiretrovir. 2013;5:044-049.
- 10. Lochmanova A and Lochman I. Immunity to Cytomegalovirus. J Antivir Antiretrovir. 2011;3:040-044.
- 11. Chen Z and Zhou J. Strengthening the Role of Dendritic Cells in AIDS Vaccine Development. J Antivir Antiretrovir. 2013;5:xxiii-xxiv.
- 12. Bastian AR. Microbicides that Can Pop HIV-1. J Antivir Antiretrovir. 2014;6:054-056.
- 13. Nampala H et al. Modelling Effective Antiretroviral Therapy that Inhibits HIV Production in the Liver. J Antivir Antiretrovir. 2015;7:043-051.
- 14. Bharaj P and Chahar HS. Immune Activation and HIV Pathogenesis: Implications for Therapy. J Antivir Antiretrovir. 2015;7:015-021.
- 15. Rao PSS et al. New Antiretroviral Therapies and Potential Drug Interactions in HIV-Infected Drug Abusers. J Antivir Antiretrovir. 2014;6:086-091.
- 16. Sindhura BR et al. Lectins: Magic Bullet towards HIV gp120. J Antivir Antiretrovir. 2012;4:101-102.
- 17. Asmare M et al. Level of ART Adherence and Associated Factors among HIV Sero- Positive Adult on Highly Active Antiretroviral Therapy in Debre Markos Referral Hospital, Northwest Ethiopia. J Antivir Antiretrovir. 2014;6:120-126.
- 18. Bismara BA et al. Antiretroviral Drug Resistance in Brazilian Children Infected by Human Immunodeficiency Virus Type 1. J Antivir Antiretrovir. 2012;4:066-074.
- 19. de Matos VTG et al. HIV Vertical Transmission: Why is it Still Happening in Brazil? J Antivir Antiretrovir. 2014;6:043-044.
- 20. Nomoto M et al. Socioeconomic Impact of HIV/AIDS on Households under Free Antiretroviral Therapy in Preah Sihanouk Province, Cambodia. J Antivir Antiretrovir. 2013;5:003-007.
- 21. Parruti G et al. Efficacy of 1998 Vs 2006 First-Line Antiretroviral Regimens for HIV Infection: An Ordinary Clinics Retrospective Investigation. J Antivir Antiretrovir. 2012;4:032-037.
- 22. Nomoto M et al. Socioeconomic Impact of HIV/AIDS on Households under Free Antiretroviral Therapy in Preah Sihanouk Province, Cambodia. J Antivir Antiretrovir. 2013;5:003-007.
- 23. Rosenberg NE et al. The Awareness Framework: A Novel Approach for Understanding HIV Testing and Disclosure in HIV-discordant Dyads. J Antivir Antiretrovir. 2013;5:008-011.

- 24. Ndou TV et al. Experiences of HIV Positive Patients on ARV Treatment at the Thulamela Municipality in the Vhembe District of Limpopo Province, South Africa. J Antivir Antiretrovir. 2013;5:123-131.
- 25. Wang X et al. Effect of Viral Load and Drug Resistance on Mortality among Chinese HIV-Infected Patients Receiving Antiretroviral Treatment. J Antivir Antiretrovir. 2012;4:060-065.
- 26. Panda S et al. Correlates of HIV Transmission from Husband to Wife among Heterosexual Married Couples in ART-era in West Bengal, India. J AIDS Clin Res. 2015;6:417.
- 27. Nachega JB et al. Adherence to Antiretroviral Therapy for the Success of Emerging Interventions to Prevent HIV Transmission: A Wake up Call. J AIDS Clinic Res. 2012;S4:007.
- 28. Hong SY et al. Knowledge of HIV Transmission and Associated Factors among HIV-Positive and HIV-Negative Patients in Rural Kenya. J AIDS Clinic Res. 2012;3:170.
- 29. Phillips DM et al. Mechanisms of sexual transmission of HIV: does HIV infect intact epithelia? Trends Microbiol. 1994;2:454-458.
- 30. Dai L et al. Quantitative Transmitted Drug Resistance (TDR) Variation in Acute/Recently Infected Men who have Sex with Men (MSM) Chinese HIV Patient Cohort. J Antivir Antiretrovir. 2013;6:013-021.
- 31. Mollaei HR et al. Antiviral Activity of Sirna UL42 against Herpes Simplex Virus Type 1 in HeLa Cell Culture. J Antivir Antiretrovir. 2014;6:114-119.
- 32. Pereira H. Condom Use and HIV-Related Behaviors in Portuguese Men who have Sex with Men: A Study of Sexual Behavior and Sexual Pleasure. J AIDS Clin Res. 2014;5:294.
- 33. Khalil MZ. Challenges in Management of Pericardial Effusion in Patients with HIV/AIDS. J Antivir Antiretrovir. 2013;5:001-002.
- 34. Rao PSS et al. New Antiretroviral Therapies and Potential Drug Interactions in HIV-Infected Drug Abusers. J Antivir Antiretrovir. 2014;6:086-091.
- 35. Kourtis AP and Bulterys M. Mother-to-child transmission of HIV: pathogenesis, mechanisms and pathways. Clin Perinatol. 2010;7:721-737.
- 36. McGowan JP and Shah SS Prevention of perinatal HIV transmission during pregnancy. J Antimicrob Chemother. 2000;46:657-668.
- 37. Nlend AEN et al. Birth Outcomes in HIV-1-Infected Women Receiving Highly Active Antiretroviral Therapy (HAART) Prior to Conception versus During Pregnancy in Yaoundé, Cameroon. J Antivir Antiretrovir. 2014;6:135-138.
- 38. Gidey B et al. Voluntary HIV Counseling and Testing Service Utilization among Pregnant Mothers in North West Ethiopia In 2014. J AIDS Clin Res. 2015;6:437.
- 39. Hamdela B et al. Knowledge on Mother to Child Transmission and Utilization of Services Designed to Prevent Mother to Child Transmission of HIV/AIDS among Pregnant Women in Hossana Town, Southern Ethiopia. J AIDS Clin Res. 2014;5:396.
- 40. Lilian O et al. The Role of a Special Prevention of Mother to Child Transmission Clinic in Improving Prevention, Care and Treatment of Infected and Exposed Infants; TASO Masaka Experience. J AIDS Clin Res. 2014;5:322.
- 41. Kalu SO et al. Infant Feeding Choices Practiced among HIV Positive Mothers Attending a Prevention of Mother to Child Transmission (PMTCT) of HIV Program in Nnewi, Nigeria. J AIDS Clin Res. 2014;5:300.
- 42. Anígilájé EA et al. The Prevalence and Predictors of HIV Infection among Children of Mothers who Missed Prevention of Mother to Child Transmission of HIV Interventions in Makurdi, Nigeria. J AIDS Clin Res. 2013;4:249.
- 43. Qian HZ et al. Association of misconceptions about HIV transmission and discriminatory attitudes in rural China. AIDS Care. 2007;19:1283-1287.

- 44. Dahake R et al. Potential Anti-HIV Activity of Jatropha curcas Linn. Leaf Extracts. J Antivir Antiretrovir. 20155:160-165.
- 45. Jones GS et al. Preclinical and Clinical Profile of HIV-1 Integrase Strand-transfer Inhibitor GS-9224 Compared to its Parent Compound GS-9160. J Antivir Antiretrovir. 2014;6:075-083.
- 46. Tchiakpe E et al. The Prediction of Integrase Inhibitors Efficacy in Third Line Regimen after First and Second Line Antiretroviral Therapy Failure in Senegal. J Antivir Antiretrovir. 2014;6:127-134.
- 47. Hitti J et al. Frequency of Antiretroviral Resistance Mutations among Infants Exposed to Single-Dose Nevirapine and Short Course Maternal Antiretroviral Regimens: ACTG A5207. J AIDS Clin Res. 2014;5:371.
- 48. Shao ER, Kifaro EG, Kimaro J, Mrema JG, Mwasamwaja AO, et al. (2014) HIV-1 Diversity in Tanzania and its Implication toward Development of Effective Vaccines: A Review Article. J Vaccines Vaccin. 2014;5:249.
- 49. Salah S et al. A Novel Approach to Inhibit HIV-1 Infection by Actively Neutralizing the Antibodies of Reverse Transcriptase System. J AIDS Clin Res. 2014;5:310.
- 50. Noorali S et al. Effect of Differentially Expressed MicroRNAs 602 and 323-5p on Hepatitis C Virus Genotype 1b Viral Load in Infected Liver Cells. J Infect Dis Ther. 2014;2:138.
- 51. Soni RK et al. Role of Reverse Transcriptase and APOBEC3G in Survival of Human Immune Deficiency Virus -1 Genome. Virol Mycol. 2013;3:125.
- 52. Srivastava S and Kanyalkar M. To Probe the Conformational Adaptability of Conserved G-P-G-R Segment in the V3 Loop of HIV-1. J Antivir Antiretrovir. 2012;4:088-093.
- 53. Babar MM et al. Virosomes-Hybrid Drug Delivery Systems. J Antivir Antiretrovir. 2013;5:166-172.
- 54. Kibirige C. The Use of Ultra-Sensitive Molecular Assays in HIV Cure- Related Research. J AIDS Clinic Res. 2013;S6:002.
- 55. Isaac A et al. Antiretroviral Therapy, CD4, Viral Load, and Disease Stage in Saudi HIV Patients: A Cross Sectional Study of Cases Diagnosed between 2001-2013. J AIDS Clin Res. 2015;6:447.
- 56. Szwarcwald CL et al. Estimation of the HIV Incidence and of the Number of People Living With HIV/AIDS in Brazil, 2012. J AIDS Clin Res. 2015;6:430.
- 57. Moodley A and Wood NH HIV-Associated Oral Lesions in HIVSeropositive Patients at an HIV-Treatment Clinic in South Africa. J AIDS Clin Res. 2015;6:422.
- 58. Ibrahim L et al. CD49d and CD26 are Independent Prognostic Markers for Disease Progression in Patients with Chronic Lymphocytic Leukemia. J Leuk (Los Angel). 2015;3:173.
- 59. Kumar A et al. Immunological and Virological Outcomes at 5 Years in HIV Infected Adults Who Start HAART at a CD4 Cell Count of Less Than 200 in Barbados. J AIDS Clin Res. 2015;6:406.
- 60. Mugairi AA et al. Thymic Immunophenotype, and Expression of CD4 and Myeloid Antigens is Associated with Outcome in Adult Patients with T–Cell Acute Lymphoblastic Leukemia. J Leuk (Los Angel). 2015;3:172.
- 61. Beck-Sague CM et al. Depression and Response to Antiretroviral Therapy in the Dominican Republic. J AIDS Clin Res. 2014;5:401.
- 62. Chakraborty A et al. Cytomegalovirus Retinitis with Multiple Co Infections in a HIV/AIDS Patient having Extreme Low CD4 Count: A Case Report and Review of Literature. J AIDS Clin Res. 2014;5:394.
- 63. Srirangaraj S and Venkatesha D. Short Term Survival after Anti- Retroviral Therapy Initiation among Aids Patients from South India. J Antivir Antiretrovir. 2012;S10.
- 64. Yanagisawa N et al. HIV-Infected Men with an Elevated Level of Serum Cystatin C have a High Likelihood of Developing Cancers. J Antivir Antiretrovir. 2012;4:038-042.
- 65. Green RJ et al. Severe Pneumonia in HIV-infected Infants–Clinical and Immunological Correlates. Trying to Improve Diagnosis and thereby Survival. J Antivir Antiretrovir. 2013;5:xxx-xxxi.

- 66. Keith Reeves. Mechanisms, Consequences, and Treatment of Chronic Inflammation in HIV Disease. J Antivir Antiretrovir. 2014;6:xxxvixxxvii.
- 67. de O' Leary JC et al. The Impact of HAART on Advanced Brain Aging: Implications for Mitochondrial Dysfunction and APP Processing. J Antivir Antiretrovir. 2012;S10.
- 68. Letto G et al. Acute Page Kidney Phenomenon Secondary to Lymphocele Compression in Renal Allograft Recipient: A Case Report. J Nephrol Ther. 2015;5:199.
- 69. Ahmadi F et al. Fetal kidney Measurement in 26-39 Weeks Gestation in Normal Fetuses of Iranian Pregnant Women. J Preg Child Health. 2015;2:139.
- 70. Nabatame M et al. Profound Hypotension during Kidney Transplantation for a Patient with a Depressive Disorder. J Anesth Clin Res. 2015;6:518.
- 71. Hainova K et al. Microflora of Intestinal and Respiratory Tract in AIDS Process. J Antivir Antiretrovir. 2013;S15:006.
- 72. Du P et al. Comparisons of VLP-Based ELISA, Neutralization Assays with Native HPV, and Neutralization Assays with PsV in Detecting HPV Antibody Responses in HIV-Infected Women. J AIDS Clin Res. 2015;6:433.
- 73. Jaen A et al. Impact of Fixed-Dose Combinations of Antiretrovirals on Prevalence Trends of HIV Resistance: A 7 Year Follow-Up Study. J AIDS Clin Res. 2015;6:416.
- 74. Farid AH and Segervall. A Comparison between ELISA and CIEP for Measuring Antibody Titres against Aleutian Mink Disease Virus. Virol-mycol. 2014;3:137.
- 75. Cecilia B et al. Menopause in HIV Infected Women: A Comprehensive Approach to Physical and Psychological Health. J Osteopor Phys Act. 2014;2:117.
- 76. de Oliveira Lanna ME et al. Diabetes Effects in Alzheimer Disease: The Interactive Role of Insulin and Aß Peptide. J Alzheimers Dis Parkinsonism. 2014;4:151.
- 77. von Hentig N. HIV treatment in 2013-Between Access and Cure or "Let's become Political•. J Antivir Antiretrovir. 2013;5:xxvii-xxviii.
- 78. Naga Anusha P. Antiretroviral Strategies for Treatment of HIV. J Antivir Antiretrovir. 2011;3:055-059.
- 79. Shittu RO et al. Adherence to Highly Active Antiretroviral Therapy, in Depressed Peoples Living with HIV/AIDS in Nigeria, West Africa. J Antivir Antiretrovir. 2013;6:006-012.
- 80. Ohrui H. A New Paradigm for Developing Antiviral Drugs Exemplified by the Development of Supremely High Anti-HIV Active EFdA. J Antivir Antiretrovir. 2014;6:032-039.
- 81. Babar M et al. Antiviral Drug Therapy- Exploiting Medicinal Plants. J Antivir Antiretrovir. 2013;5:028-036.
- 82. Kirchner JT. A Tolerability Review of Non-Nucleoside Reverse Transcriptase Inhibitors: Focus on Laboratory Measures of Clinical Relevance. J Antivir Antiretrovir. 2012;4:094-100.
- 83. Hentig NV. Personalizing HIV Therapy, Mission Impossible? J Antivir Antiretrovir. 2013;5:012-020.
- 84. Dong BJ et al. Safety and Effectiveness of Tenofovir/Emtricitabine or Lamivudine Plus Ritonavir Boosted Atazanavir in Treatment Experienced HIV Infected Adults at Two Urban Private Medical Practices. J Antivir Antiretrovir. 2012;4:001-005.
- 85. Jain SK et al. In-vitro and in-vivo Evaluation of Poly (Propyl Ether Imine) (PETIM) Dendrimer for Sustained Delivery of Zidovudine. J Antivir Antiretrovir. 2013;S10-004.
- 86. Alozie O et al. Switching from Abacavir/Lamivudine to Tenofovir DF/Emtricitabine Reduces Biomarkers of Inflammation: A Randomized Proof of Concept Study. J AIDS Clin Res. 2014;5:278.
- 87. Lazarus EM et al. Lamivudine Monotherapy as a Holding Strategy in HIV-Infected Children in South Africa. J AIDS Clin Res. 2013;4:246.
- 88. Ali MM et al. Mutation Patterns at Codons Rt204 And Rt180 of the HBV Polymerase Gene Associated with Lamivudine Resistance in Treated and Untreated Chronic HBV Patients in Kuwait: A Case Series. J Clin Case Rep. 2013;3:276.

- 89. Cuzin L et al. Tolerance and Durability of Abacavir/Lamivudine (ABC/3TC) Containing Regimens: Results from a large French Prospective Cohort. J AIDS Clinic Res. 2012;S1:019.
- 90. Feleder EC et al. Single-Dose Bioequivalence of a New Fixed-Dose Combination Tablet Containing Tenofovir Disoproxil Fumarate and Lamivudine. J Bioequiv Availab. 2011;3:236-243.
- 91. Umoren EB et al. Effect of Nevirapine Administration on Biliary Secretion/its Biochemical Composition in Albino Wistar Rats. J Antivir Antiretrovir. 2014;6:045-049.
- 92. Umoren EB and Obembe AO. Intestinal Fluid and Glucose Transport in Albino Wistar Rats Following Long Term Administration of Nevirapine. J Antivir Antiretrovir. 2014;6:057-063.
- 93. Behbahani M et al. Anti-HIV-1 Activities of Aerial Parts of Ocimum basilicum and its Parasite Cuscuta campestris. J Antivir Antiretrovir. 2013;5:057-061.
- 94. Herrero LJ et al. Antivirals: Bindarit-The Future in Alphavirus Treatment. J Antivir Antiretrovir. 2013;5:xxix-xxix.
- 95. Remor E. Release of an Online Self-Reporting Tool for Assessing Adherence to Antiretroviral Therapy (CEAT-VIH). J Antivir Antiretrovir. 2013;5:178-179.
- 96. Navarro-Mercadé J et al. Long-Term Effectiveness of First-Line Antiretroviral Theraphy in a Cohort of HIV-1 Infected Patients. J Antivir Antiretrovir. 2012;4:026-031.
- 97. Dai L et al. Prevalence of Transmitted HIV-1 Drug Resistance (TDR) Associated Mutations and Predicted Drug Sensitivity in Newly Diagnosed HIV-1 Patient Cohort in a Western New York, 2005-2011. J Antivir Antiretrovir. 2014;6:022-027.
- 98. Mwambete KD and Kamuhabwa AAR. Resistance of Commensal Intestinal Escherichia Coli and Other Enterics to Co-trimoxazole and Commonly Used Antibiotics in HIV/AIDS Patients. Clin Microbial. 2013;3:134.
- 99. Said J et al. HIV-1 Variants with Reduced Sensitivity to Sulfated Oligosaccharide Muparfostat Contain Mutations in the Envelope Glycoproteins gp120 and gp41. J Antivir Antiretrovir. 2013;5:050-056.
- 100. Hima Bindu A and Naga Anusha P. Adverse Effects of Highly Active Anti-Retroviral Therapy (HAART). J Antivir Antiretrovir. 2011;3:060-064.
- 101. Haidara A et al. Drug Resistance Pathways and Impact of Protease Mutation L10I/V in HIV-1 Non-B Subtypes. J Antivir Antiretrovir. 2012;4:043-050.
- 102. Macharia T et al. Antiretroviral Toxicity Leading to a Medication Change in Multiple HIV Clinics in Resource Limited Settings. J Antivir Antiretrovir. 2014;6:148-152.