# The human immunodeficiency virus hiv biology essay

Design

The human immunodeficiency virus ( HIV ) is one of the members of the genus Lentivirus in the Retroviridae household. Lentiviruses by and large affect different groups of carnal viruses. Lentiviruses were premier campaigners when AIDS research workers looked for a causative agent of human immune upsets and subsequently neurologic syndromes. In 1983, HIV isolates were recovered from the blood of many patients with AIDS every bit good as neurologic syndromes. From so, the function of HIV as an aetiologic agent on the disease was strongly supported. However, the name HIV was non until 1986, when the International Committee on Taxonomy of Viruses decided to give the AIDS virus a separate name. HIV was found in two distinguishable subtypes, HIV-1 and HIV-2 ; where HIV-2 was recovered from AIDS patients in West Africa shortly after the find of HIV-1.

Both subtypes can do AIDS, although HIV-2 has less infective nature.

## HIV-1 versus HIV-2

The genome of HIV-1 is really similar to that of HIV-2 except for the presence of vpx cistron and the absence of vpr cistron in HIV-2. The major serologic difference between the two HIV subtypes is found in the glycoproteins of the viral envelope. The HIV-2 antibodies can by and large cross-react with HIV-1 proteins, but can non even observe its envelope proteins and frailty versa. In footings of pathogenesis, patients who develop AIDS due to entirely HIV-2 infection survive longer without the disease than those infected with HIV-1. Although it is still non clear why subtype HIV-2 differs from HIV-1 in footings of transmissibility and pathology, several characteristics can offer an account. Low plasma viral burden is observed in HIV-2 septic persons, which

could be 100 fold less than in HIV-1 septic persons. Besides, lower degrees of HIV-2 compared to HIV-1 are found in seeds.

Furthermore, the big figure of go arounding septic cells in instance of HIV-2 impairs the ability of such cells to bring forth new HIV-2 atoms compared to HIV-1. Another account indicates that there is reduced immune activation and T-cell programmed cell death in instance of HIV-2 infection compared to HIV-1 infection. In add-on, HIV-2 envelope induces the production of IA?-chemokine that could hold antiviral activity. Fig. 1 A typical HIV virion with the structural and other virion proteins, where the location of Vif and Nef proteins still undefinedAnother determination that is potentially related to some HIV-2 isolates is the decreased cytopathic belongingss in cell civilization and the absence of the CD4 antigen transition on the cell surface.

These observations could propose the presence of comparatively non-cytopathic strains of HIV-2 in some instances. The hold in pathogenesis might besides be due to strong immune response in the host, therefore restricting HIV-2 reproduction. The elaboration of several viral genome parts and the subsequent DNA sequence aid in comparing of different sequences derived from HIV-1 and HIV-2 isolates, particularly in the envelope part. The full length viral genome sequencing revealed the presence of three HIV-1 subgroups named M ( chief ) , O ( outlier ) , and N ( non M or O ) .

Eight HIV-2 subgroups are presently known.

## The HIV virion construction

As revealed under negatron microscopy, HIV-1 and HIV-2, like all Lentiviruses, have a conic nucleus formed of the viral protein p24 Gag mirid bug ( CA ) . The virion is measured to be about 100 to 120 nanometers in diameter, with different morphological forms.

By convention, the viral protein is designated as P followed by a figure stand foring the protein size. Typically, HIV virion consists of envelope and three structural Gag proteins ( see Fig. 1 ) . The envelope is formed of proteins derived from a 160 kDa precursor glycoprotein ( gp160 ) . Cellular enzymes found in Golgi setup cleave gp160 into a gp120 that forms the external surface envelope, and a gp41 transmembrane protein. The three structural Gag proteins are: ( 1 ) matrix ( MA, p17 ) ; it forms the inner shell of the virion merely below the viral membrane, ( 2 ) CA ( p24 ) ; it forms the conic nucleus aa,¬ ” as determined above – that encloses the genomic RNA of the virus, and ( 3 ) nucleocapsid ( NC, p7 ) ; that interacts with the viral RNA inside the mirid bug.

These viral proteins are generated from the processing of the Gag precursor propolyprotein HVI-1 p55 by the viral peptidase. Inside the Gag mirid bug, two frequently indistinguishable RNA strands are found, associated with the NC proteins, the viral RNA-dependent Deoxyribonucleic acid polymerase ( pol ) – besides known as contrary RNA polymerase – and integrase ( IN ) enzyme which helps in the integrating of the viral complementary DNA. Therefore, the assembly of HIV needs protein: nucleic acid interactions in the viral

nucleus. Other proteins are found in HIV, such as Nef and Vif proteins ( from 7 to 20 Vif molecules per virion ) which are associated with the viral nucleus.

Besides, the viral accessary cistron merchandise Vpr ( and Vpx for HVI-2 ) is found within the virion outside the nucleus. It is suggested that the presence of these proteins play function in the early events of HIV infection: Nef protein enhances viral infectivity, and it interacts with cellular proteins to assist in cell activation and signal transduction. Normally, viruses missing nef have no ability to retroflex either in vivo or in vitro. Vif protein increases the infectivity of virus and cell to cell transmittal ; and helps in proviral synthesis and virion assembly. Vpr protein helps in virus reproductionVpx protein helps in entry and infectivityThe virion besides contains certain cytoskeletal proteins ( e. g. actin, ezrin, and emerin ) . Although their functions are still unknown, emerin is found to function as a span between the interior atomic envelope of virus and the chromatin so as to ease the interaction of the viral complementary DNA with chromatin and subsequent integrating.

Electron microscopy besides reveals the presence of spikes on the surface of HIV virion, arranged as tripod-like constructions. Finally, like other retroviruses, HIV isolates show the inclination to integrate specific lipid membranes from the host cell membrane during viral budding.

## HIV genome

The size of HIV genome is about 10 kilobits, affecting unfastened reading frames coding for many viral proteins. The full length messenger RNA transcript of the virus is translated into the Pol and Gag proteins. Splicing

procedures occur to bring forth many subgenomic messenger RNA responsible for the synthesis of other viral proteins.

In this context, Pol precursor polyprotein is autoclaved into the viral enzmes RT, PR and integrase ( IN ) by its ain PR part. Besides, the Gag precursor p55 is cleaved bring forthing little viral proteins that include p24, p17, p9 and p6 mentioned above, in add-on to p1 and p2. The ratio between Gag and Gag-pol synthesized merchandises is about 20: 1. Gene merchandises of other spliced messenger RNAs involve assorted viral regulative and accessary proteins that take portion in HIV reproduction in different cell types. By and large, the ratio between the unspliced, and singly and multiply spliced messenger RNA is determined by the rpm cistron, which itself produced from multiply spliced messenger RNA.

Rev protein aa,¬ ” the rev cistron merchandise aa,¬ ” regulates viral protein look. It interacts with an RNA cringle known as the Rev-responsive component, located in the messenger RNA of the viral envelope. Such interaction occurs between the cellular proteins and the Rev multimers leting the unspcliced messenger RNA to come in the cytol from the karyon and bring forth the full length viral proteins needed for progeny production.

Several surveies on molecular characteristics of HIV cistron merchandises found that the virus look ordinance involves the interaction of many viral proteins and cellular factors, taking to either high or low look, or even a latent province. For illustration, the Rev produced in late stages of viral replicative rhythm can down-regulate its ain look, and restricting the HIV reproduction in bend. It is noticeable that the HIV genome encodes three

major enzymes holding cardinal functions at different times during the viral replicative rhythm.

The RNA-dependent DNA polymerase maps early in the viral replicative rhythm to bring forth dual stranded complementary DNA transcript of the viral RNA. The IN integrates the viral complementary DNAFig. 2 illustrates the mechanism of HIV infectioninto the chromosomal Deoxyribonucleic acid of the host inside the cell nucleus. The PR processes the Gag and Gag-pol polyproteins in the budding virus, therefore assisting in the ripening of the viral atoms into an infective HIV. Therefore, these three enzymes are premier marks for antiretroviral attacks.

## HIV entry and reproduction

HIV is a cytopathic virus formed of a cardinal conic nucleus of RNA surrounded by a lipid envelope with glycoprotein ( general practitioner ) surface markers.

Many HIV-specific antigens are now identified such as gp120/160, p24 and p41 that are mentioned above. In order to successfully infect a cell, HIV must adhere at two separate sites, the CD4+ receptor and a 7-transdomain chemokine receptor. The most of import chemokines able to function this map are CCR5 and CXCR4. CCR5 is the chief co-receptor encountered by macrophage-tropical viral strains largely found in early HIV infection, whilst CXCR4 is the chief co-receptor used by T-tropic viral strains that predominate subsequently in HIV infection. Equally shortly as the virus binds at the receptor/co-receptor composite, the virus fuses with the cell membrane and addition entree to the host cell ( see Fig. 2 ) . In acute

infection, the HIV first encounters the CCR5 receptors harbored on dendritic cells ( DC ) surface. The function of GI piece of land in early HIV infection is still under probe.

It was observed that the CD4+ T-cells found in the intestine are quickly depleted during early HIV infection with limited recovery. However, early intervention can forestall gut CD4+ T-cell loss. The HIV contrary RNA polymerase generates frequent mistakes in viral reproduction, giving the chance for rapid viral development and variegation.

Therefore, HIV can easy mutate and free itself of immunologic and pharmacologic control. Once the HIV enters the cell, contrary written text occurs to bring forth a complementary DNA transcript from the viral RNA. At this point, the HIV would hold two possible tracts ; either undergoes active viral reproduction, or go incorporated into host genome as proviral DNA and enters a latent province which can last indefinitely. The proviral DNA is transcribed into messenger RNA when activation occurs. After the viral proteins are formed utilizing the host cell machinery, new virions are assembled and bud from the septic cells.

The budding virions become infective merely when processed by viral peptidase. Such infective virions circulate until they identify new mark cells. Most phases of HIV cellular infection have been pharmacologically targeted. Untreated HIV infection leads to gradual lessening in immune map boulder clay reaches AIDS, which is characterized by deteriorated unsusceptibility and susceptibleness to timeserving infections and malegnancies.