

Rabbit Anti-HIV2 gp120 + gp160 antibody

SL10482R

Product Name HIV2 gp120 + gp160

Chinese Name 人类免疫缺陷病毒 2 型/2 型艾滋病病毒 gp41+gp160 抗体

Alias Gp120; Gp160; HIV-2 gp120; HIV2 gp160; HIV2gp120; HIV2gp160; HIV 2; Human immunodeficiency virus type 2; Human Immunodeficiency Virus Type 2; ENV_HV2RO.

Research Area Bacteria and viruses

Immunogen Species Rabbit

Clonality Polyclonal

React Species (predicted:HIV2)

WB=1:500-2000,IHC-P=1:100-500,IHC-F=1:100-500,ICC/IF=1:100-500,IF=1:100-500,ELISA (Paraffin sections need antigen repair)

Applications not yet tested in other applications.
optimal dilutions/concentrations should be determined by the end user.

Form Liquid

Concentration 1mg/ml

immunogen KLH conjugated synthetic peptide derived from HIV2 gp120 + gp160: 311-380/858

Lsotype IgG

Purification affinity purified by Protein A

Buffer Solution (predicted:HIV2)1M TBS(pH7.4) with 1% BSA, (predicted:HIV2)3% Proclin300 and 50% Glycerol

Storage Shipped at 4°C. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles.

Attention This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

PubMed [PubMed](#)

Product Detail Human immunodeficiency virus type 2 (HIV2), originally isolated from patients in West Africa, is a form of HIV in West Africa capable of causing the acquired immunodeficiency syndrome (AIDS). It is closely related to simian immunodeficiency viruses (SIV). HIV1 and HIV2 share similarity in their mode of transmission, clinical features, immunological effects, and in their action of binding to the same receptor, but there are significant differences in the amino acid and nucleotide sequences of HIV2, especially within their envelope genes and proteins. Additionally, HIV2 may have a longer incubation period.

may be less pathogenic than HIV1. HIV2 gp36 is a transmembrane protein located in the envelope specific to HIV2 that binds to the putative cellular receptor proteins P45 and P62.

Function:

The surface protein gp120 (SU) attaches the virus to the host lymphoid cell by binding to the primary receptor CD4. This interaction induces a structural rearrangement creating a high affinity binding site for coreceptor like CXCR4 and/or CCR5. This peculiar 2 stage receptor-interaction strategy allows the virus to maintain the highly conserved coreceptor-binding site in a cryptic conformation, protected from neutralizing antibodies. Since CD4 also displays a binding site for the disulfide-isomerase P4HB/PDI, a P4HB/PDI-CD4-CXCR4-gp120 complex may form. In that complex, P4HB/PDI could reach and break the disulfide bonds, causing major conformational changes in gp120. TXN, another PDI family member, may be involved in disulfide rearrangements in Env during fusion. These changes are transmitted to the transmembrane protein gp41 and are thought to activate its fusogenic potential by unmasking its fusion peptide. The surface protein gp120 is a ligand for CD209/DC-SIGN and CLEC4M/DC-SIGNR, which are found on dendritic cells (DCs), and on endothelial cells of liver sinusoids and lymph node sinusoids. These interactions allow capture of viral particles at mucosal surfaces by these cells and subsequent transport to permissive cells. DCs are professional antigen presenting cells, critical for host immunity by inducing and regulating immune responses against a broad variety of pathogens. They act as sentinels in various tissues where they pick up antigen, process it, and present it to T-cells following migration to lymphoid organs. HIV subverts the migration properties of dendritic cells to gain access to CD4+ T-cells in lymph nodes. Virus transport to permissive T-cells occurs either in trans (without DCs infection, through viral capture and transport to T-cells) (following DCs productive infection, through the usual CD4-gp120 interaction), thereby inducing infection. In trans infection, bound virions remain infectious over days and it is proposed that they are not degraded, but protected in non-lysosomal acidic organelles within the DCs close to the cell membrane, contributing to the viral infectious potential during DCs' migration from the periphery to the lymphoid tissues. On arrival at lymphoid tissues, intact virions recycle back to DCs' cell surface allowing virus transport to CD4+ T-cells. Virion capture also seems to lead to MHC-II-restricted viral antigen presentation, resulting in the activation of HIV-specific CD4+ cells.

The transmembrane protein gp41 (TM) acts as a class I viral fusion protein. Under the current model, gp41 has at least 3 conformational states: pre-fusion native state, pre-hairpin intermediate state, and post-fusion hairpin state. During fusion of viral and target intracellular membranes, the coiled coil regions (gp41) assume a trimer-of-hairpins structure, positioning the fusion peptide in close proximity to the C-terminus of the ectodomain. The formation of this structure appears to drive apposition and subsequent fusion of target cell membranes. Complete fusion occurs in host cell endosomes and is dynamin-dependent. Lipid transfer might occur at the plasma membrane. The virus undergoes clathrin-dependent internalization before endosomal fusion, thus minimizing the surface exposure of conserved viral epitopes during transport, reducing the efficacy of inhibitors targeting these epitopes. Membranes fusion leads to delivery of the nucleocapsid into the cytoplasm.

The envelope glycoprotein gp160 precursor down-modulates cell surface CD4 antigen by interacting with the endoplasmic reticulum and blocking its transport to the cell surface.

The gp120-gp41 heterodimer seems to contribute to T-cell depletion during HIV-1 infection. The viral glycoproteins expressed on the surface of infected cells induce apoptosis through an interaction with bystander cells expressing the receptor (CD4) and the coreceptors CXCR4 or CCR5. This type of bystander effect can be obtained by at least three distinct mechanisms. First, the interaction between the 2 cells can induce

followed by nuclear fusion within the syncytium. Syncytia are condemned to die from apoptosis. Interacting cells may not fuse entirely and simply exchange plasma membrane lipids, after a sort of process, followed by rapid death. Third, it is possible that virus-infected cells, on the point of undergoing apoptosis, fuse with CD4-expressing cells, in which case apoptosis is rapidly transmitted from one to the other and thus occurs in a sort of contagious fashion.

The gp120-gp41 heterodimer allows rapid transcytosis of the virus through CD4 negative cells across epithelial monolayers of the intestinal, rectal and endocervical epithelial barriers. Both gp120 and gp41 specifically recognize glycosphingolipids galactosyl-ceramide (GalCer) or 3' sulfo-galactosyl-ceramide present in the lipid raft structures of epithelial cells. Binding to these alternative receptors allows transcytosis of the virus through the epithelial cells. This transcytotic vesicle-mediated transport from the apical side to the basolateral side of the epithelial cells does not involve infection of the cells.

Subunit:

The mature envelope protein (Env) consists of a homotrimer of non-covalently associated gp120-gp41 heterodimers. The resulting complex protrudes from the virus surface as a spike. There seems to be 3 spikes on the average virion. Surface protein gp120 interacts with human CD4, CCR5 and CXCR4. P4HB/PDI-CD4-CXCR4-gp120 complex. Gp120 also interacts with the C-type lectins CD209/DC-SIGN and CLEC4M/DC-SIGNR (collectively referred to as DC-SIGN(R)). Gp120 and gp41 interact with

Subcellular Location:

Transmembrane protein gp41: Virion membrane; Single-pass type I membrane protein. Host cell membrane; Single-pass type I membrane protein. Host endosome membrane; Single-pass type I membrane protein (Potential). Note=It is probably concentrated at the site of budding and incorporated into the virion. Contacts between the cytoplasmic tail of Env and the N-terminus of Gag.

Surface protein gp120: Virion membrane; Peripheral membrane protein. Host cell membrane; Peripheral membrane protein. Host endosome membrane; Peripheral membrane protein (Potential). Note=This protein is not anchored to the viral envelope, but associates with the extravirion surface through its N-terminus. It is probably concentrated at the site of budding and incorporated into the virions possibly between the cytoplasmic tail of Env and the N-terminus of Gag.

Post-translational modifications:

Specific enzymatic cleavages in vivo yield mature proteins. Envelope glycoproteins are synthesized as a precursor that is heavily N-glycosylated and processed likely by host cell furin in the Golgi to yield SU and TM proteins. The cleavage site between SU and TM requires the minimal sequence [KR...]. Palmitoylation of the transmembrane protein and of Env polyprotein (prior to its proteolytic cleavage) is essential for their association with host cell membrane lipid rafts. Palmitoylation is therefore required for envelope trafficking to classical lipid rafts, but not for viral replication.

SWISS:

N/A

Gene ID:

N/A



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Database links:

UniProtKB/Swiss-Prot: P04577