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Rabbit Anti-HIV2 gp36/Biotin Conjugated antibody

SL6410R-Bio

Product Name	Anti-HIV2 gp36/Biotin
Chinese Name	生物素标记的人类免疫缺陷病毒 2 型/2 型艾滋病病毒 gp36 抗体
Alias	Gp36; HIV-2 gp36; HIV2 gp36; HIV2gp36; gp36; HIV 2; Human immunodeficiency virus 2; Human Immunodeficiency Virus Type 2; ENV_HV2RO.
Research Area	Cell biology immunology Bacteria and viruses
Immunogen Species	Rabbit
Clonality	Polyclonal
React Species	(predicted:HIV2)
Applications	WB=1000-10000,ELISA=1:500-5000 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight	43/92kDa
Form	Lyophilized or Liquid
Concentration	1mg/ml
immunogen	KLH conjugated synthetic peptide derived from HIV2 gp36
Lsotype	IgG
Purification	affinity purified by Protein A
Storage Buffer	1M TBS(pH7.4) with 1% BSA, 3% Proclin300 and 50% Glycerol.
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 1M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
Product Detail	**background:** Human immunodeficiency virus type 2 (HIV2), orginally isolated from patients in West Africa, is the dominant form of HIV in West Africa capable of causing the acquired immunodeficiency syndrome (AIDS). HIV2 is closely related to simian immunodeficiency viruses (SIV). HIV1 and HIV2 share

similarity in their genomes, transmission, clinical features, immunological effects, and in their action of binding to the same CD4 cellular receptor, but there are significant differences in the amino acid and nucleotide sequences of HIV1 and HIV2, especially within their envelope genes and proteins. Additionally, HIV2 may have a longer incubation period and may be less pathogenic than HIV1. HIV2 gp36 is a transmembrane protein located in the envelope of the virus 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 a chemokine coreceptor like CXCR4 and/or CCR5. This peculiar 2 stage receptor-interaction strategy allows gp120 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 reduce gp120 disulfide bonds, causing major conformational changes in gp120. TXN, another PDI family member could also 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 respectively found on dendritic cells (DCs), and on endothelial cells of liver sinusoids and lymph node sinuses. These interactions allow capture of viral particles at mucosal surfaces by these cells and subsequent transmission to permissive cells. DCs are professional antigen presenting cells, critical for host immunity by inducing specific immune responses against a broad variety of pathogens. They act as sentinels in various tissues where they take 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 transmission to permissive T-cells occurs either in trans (without DCs infection, through viral capture and transmission), or in cis (following DCs productive infection, through the usual CD4-gp120 interaction), thereby inducing a robust 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 thus 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 transmission to CD4+ T-cells. Virion capture also seems to lead to MHC-II-restricted viral antigen presentation, and probably to the activation of HIV-specific CD4+ cells.

The transmembrane protein gp41 (TM) acts as a class I viral fusion protein. Under the current model, the protein 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 (heptad repeats) assume a trimer-of-hairpins structure, positioning the fusion peptide in close proximity to the C-terminal region of the ectodomain. The formation of this structure appears to drive apposition and subsequent fusion of viral and target cell membranes. Complete fusion occurs in host cell endosomes and is dynamin-dependent, however some lipid transfer might occur at the plasma membrane. The virus undergoes clathrin-dependent internalization long before endosomal fusion, thus minimizing the surface exposure of conserved viral epitopes during fusion and reducing the efficacy of inhibitors targeting these epitopes. Membranes fusion leads to delivery of the nucleocapsid into the cytoplasm.
The envelope glyprotein gp160 precursor down-modulates cell surface CD4 antigen by interacting with it in 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 envelope glycoproteins expressed on the surface of infected cells induce apoptosis through an interaction with uninfected cells expressing the receptor (CD4) and the coreceptors CXCR4 or CCR5. This type of bystander killing may be obtained by at least three distinct mechanisms. First, the interaction between the 2 cells can induce cellular fusion followed by nuclear fusion within the syncytium. Syncytia are condemned to die from apoptosis. Second, the 2 interacting cells may not fuse entirely and simply exchange plasma membrane lipids, after a sort of hemifusion 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 cell 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 such as simple 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 (GalS) present in the lipid rafts structures of epithelial cells. Binding to these alternative receptors allows the rapid transcytosis of the virus through the epithelial cells. This transcytotic vesicle-mediated transport of virions from the apical side to the basolateral side of the epithelial cells does not involve infection of the cells themselves.

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 as few as 10

spikes on the average virion. Surface protein gp120 interacts with human CD4, CCR5 and CXCR4, to form a 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 GalCer.

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 virions possibly by 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=The surface protein is not anchored to the viral envelope, but associates with the extravirion surface through its binding to TM. It is probably concentrated at the site of budding and incorporated into the virions possibly by contacts 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 inactive precursor that is heavily N-glycosylated and processed likely by host cell furin in the Golgi to yield the mature SU and TM proteins. The cleavage site between SU and TM requires the minimal sequence [KR]-X-[KR]-R.
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.

Database links:
UniProtKB/Swiss-Prot: P04577

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