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Rabbit Anti-HIV2 gp41 + gp160 antibody

SL10481R

Product Name	HIV2 gp41 + 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	Gp41; Gp160; HIV-2 gp41; HIV2 gp160; HIV2gp41; HIV2gp160; HIV 2; Human immunodefic Human Immunodeficiency Virus Type 2; ENV_HV2RO.
Research Area	Bacteria and viruses
Immunogen Species	Rabbit
Clonality	Polyclonal
React Species	(predicted:HIV2)
Applications	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） 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 gp41 + gp160: 621-700/858
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% Glyc
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 d applications.
PubMed	PubMed
Product Detail	Human immunodeficiency virus type 2 (HIV2), orginally isolated from patients in West Africa, form of HIV in West Africa capable of causing the acquired immunodeficiency syndrome (AIDS closely related to simian immunodeficiency viruses (SIV). HIV1 and HIV2 share similarity in th 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 HIV especially within their envelope genes and proteins. Additionally, HIV2 may have a longer incuba

may be less pathogenic than HIV1. HIV2 gp36 is a transmembrane protein located in the envelo
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 pri
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
maintain the highly conserved coreceptor-binding site in a cryptic conformation, protected from
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 an
disulfide bonds, causing major conformational changes in gp120. TXN, another PDI family mem
be involved in disulfide rearrangements in Env during fusion. These changes are transmitted to t
transmembrane protein gp41 and are thought to activate its fusogenic potential by unmasking its
The surface protein gp120 is a ligand for CD209/DC-SIGN and CLEC4M/DC-SIGNR, which ar
found on dendritic cells (DCs), and on endothelial cells of liver sinusoids and lymph node sinuse
interactions allow capture of viral particles at mucosal surfaces by these cells and subsequent tra
permissive cells. DCs are professional antigen presenting cells, critical for host immunity by ind
immune responses against a broad variety of pathogens. They act as sentinels in various tissues
up antigen, process it, and present it to T-cells following migration to lymphoid organs. HIV sub
migration properties of dendritic cells to gain access to CD4+ T-cells in lymph nodes. Virus tran
permissive T-cells occurs either in trans (without DCs infection, through viral capture and transmi
(following DCs productive infection, through the usual CD4-gp120 interaction), thereby inducin
infection. In trans infection, bound virions remain infectious over days and it is proposed that the
degraded, but protected in non-lysosomal acidic organelles within the DCs close to the cell mem
contributing to the viral infectious potential during DCs' migration from the periphery to the lym
On arrival at lymphoid tissues, intact virions recycle back to DCs' cell surface allowing virus tra
CD4+ T-cells. Virion capture also seems to lead to MHC-II-restricted viral antigen presentation,
the activation of HIV-specific CD4+ cells.
The transmembrane protein gp41 (TM) acts as a class I viral fusion protein. Under the current mo
has at least 3 conformational states: pre-fusion native state, pre-hairpin intermediate state, and po
hairpin state. During fusion of viral and target intracellular membranes, the coiled coil regions (h
assume a trimer-of-hairpins structure, positioning the fusion peptide in close proximity to the C-
of the ectodomain. The formation of this structure appears to drive apposition and subsequent fus
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 inter
before endosomal fusion, thus minimizing the surface exposure of conserved viral epitopes durin
reducing the efficacy of inhibitors targeting these epitopes. Membranes fusion leads to delivery
nucleocapsid into the cytoplasm.
The envelope glyprotein gp160 precursor down-modulates cell surface CD4 antigen by interacti
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. Th
glycoproteins expressed on the surface of infected cells induce apoptosis through an interaction
cells expressing the receptor (CD4) and the coreceptors CXCR4 or CCR5. This type of bystander
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 process, followed by rapid death. Third, it is possible that virus-infected cells, on the point of un apoptosis, fuse with CD4-expressing cells, in which case apoptosis is rapidly transmitted from o other and thus occurs in a sort of contagious fashion.
The gp120-gp41 heterodimer allows rapid transcytosis of the virus through CD4 negative cells s epithelial monolayers of the intestinal, rectal and endocervical epithelial barriers. Both gp120 an specifically recognize glycosphingolipids galactosyl-ceramide (GalCer) or 3' sulfo-galactosyl-ce present in the lipid rafts structures of epithelial cells. Binding to these alternative receptors allow transcytosis of the virus through the epithelial cells. This transcytotic vesicle-mediated transport 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 heterodimers. The resulting complex protrudes from the virus surface as a spike. There seems to spikes on the average virion. Surface protein gp120 interacts with human CD4, CCR5 and CXC P4HB/PDI-CD4-CXCR4-gp120 complex. Gp120 also interacts with the C-type lectins CD209/D 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 cel Single-pass type I membrane protein. Host endosome membrane; Single-pass type I membrane p (Potential). Note=It is probably concentrated at the site of budding and incorporated into the viri contacts between the cytoplasmic tail of Env and the N-terminus of Gag.
Surface protein gp120: Virion membrane; Peripheral membrane protein. Host cell membrane; Pe membrane protein. Host endosome membrane; Peripheral membrane protein (Potential). Note=T protein is not anchored to the viral envelope, but associates with the extravirion surface through TM. 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 synthesiz precursor that is heavily N-glycosylated and processed likely by host cell furin in the Golgi to yi 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 clea essential for their association with host cell membrane lipid rafts. Palmitoylation is therefore req envelope trafficking to classical lipid rafts, but not for viral replication.

SWISS:
N/A

Gene ID:
N/A

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Database links:
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