艾滋病病毒 Nef 抗体

产品货号： mlR17454

英文名称： HIV1 Nef

中文名称： 艾滋病病毒 Nef 抗体

别　　名： C terminal core protein; F protein; NEF_HV1A2; Nef; Negative factor; p27.

研究领域： 细胞生物　细菌及病毒

抗体来源： Rabbit

克隆类型： Polyclonal

交叉反应： HIV1

产品应用： WB=1:500-2000 ELISA=1:500-1000 IHC-P=1:400-800 IHC-F=1:400-800 ICC=1:100-500 IF=1:100-500
（石蜡切片需做抗原修复）

not yet tested in other applications.

optimal dilutions/concentrations should be determined by the end user.

分 子 量： 24kDa

性　　状： Lyophilized or Liquid

浓　　度： 1mg/ml

免 疫 原： KLH conjugated synthetic peptide derived from human HIV1 Nef:121-210/210

亚　　型： IgG

纯化方法： affinity purified by Protein A

储 存 液： 0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.

保存条件： 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 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 ° C.

PubMed： PubMed

产品介绍： Nef is a early protein that appears to play a role in optimizing the host cell environment for viral replication without causing cell death by apoptosis. Nef enhances virus infectivity and pathogenicity. It down

modulates surface MHC I molecules and internalized molecules are sequested to the trans-Golgi network. The number of cell surface CD4 antigen are decreased by interacting with the Src family kinase LCK thereby inducing LCK CD4 dissociation and by increasing clathrin-dependent endocytosis of this antigen to target it to lysosomal degradation.

Function:

Factor of infectivity and pathogenicity, required for optimal virus replication. Alters numerous pathways of T-lymphocytes function and down-regulates immunity surface molecules in order to evade host defense and increase viral infectivity. Alters the functionality of other immunity cells, like dendritic cells, monocytes/macrophages and NK cells. One of the earliest and most abundantly expressed viral proteins (By similarity).

In infected CD4(+) T-lymphocytes, down-regulates the surface MHC-I, mature MHC-II, CD4, CD28, CCR5 and CXCR4 molecules. Mediates internalization and degradation of host CD4 through the interaction of with the cytoplasmic tail of CD4, the recruitment of AP-2 (clathrin adapter protein complex 2), internalization through clathrin coated pits, and subsequent transport to endosomes and lysosomes for degradation. Diverts host MHC-I molecules to the trans-Golgi network-associated endosomal compartments by an endocytic pathway to finally target them for degradation. MHC-I down-regulation may involve AP-1 (clathrin adapter protein complex 1) or possibly Src family kinase-ZAP70/Syk-PI3K cascade recruited by PACS2. In consequence infected cells are masked for immune recognition by cytotoxic T-lymphocytes. Decreasing the number of immune receptors also prevents reinfection by more HIV particles (superinfection) (By similarity).

Bypasses host T-cell signaling by inducing a transcriptional program nearly identical to that of anti-CD3 cell activation. Interaction with TCR-zeta chain up-regulates the Fas ligand (FasL). Increasing surface FasL molecules and decreasing surface MHC-I molecules on infected CD4(+) cells send attacking cytotoxic CD8+ T-lymphocytes into apoptosis (By similarity).

Plays a role in optimizing the host cell environment for viral replication without causing cell death by apoptosis. Protects the infected cells from apoptosis in order to keep them alive until the next virus generation is ready to strike. Inhibits the Fas and TNFR-mediated death signals by blocking MAP3K5. Interacts and decreases the half-life of p53, protecting the infected cell against p53-mediated apoptosis. Inhibits the apoptotic signals regulated by the Bcl-2 family proteins through the formation of a Nef/PI3-kinase/PAK2 complex that leads to activation of PAK2 and induces phosphorylation of Bad (By similarity).

Extracellular Nef protein targets CD4(+) T-lymphocytes for apoptosis by interacting with CXCR4 surface receptors (By similarity).

Subunit:

Homodimer (By similarity). Interacts with Nef associated p21-activated kinase (PAK2); this interaction activates PAK2. Associates with the Nef-MHC-I-AP1 complex; this complex is required for MHC-I internalization. Interacts (via C-terminus) with host PI3-kinase (via C-terminus). Interacts with host PACS1; this interaction seems to be weak. Interacts with host PACS2. Interacts with host LCK and MAPK3; these interactions inhibit the kinase activity of the latters. Interacts with host ATP6V1H; this interaction may play a role in CD4 endocytosis. Associates with the CD4-Nef-AP2 complex; this complex is required for CD4 internalization. Interacts with TCR-zeta chain; this interaction up-regulates the Fas ligand (FasL) surface expression. Interacts with various cellular proteins including MAP3K5, beta-COP, HCK, and PTE1. Interacts with human GNB2L1/RACK1; this increases Nef phosphorylation by PKC (By similarity).

Subcellular Location:

Host cell membrane; Lipid-anchor; Cytoplasmic side (By similarity). Host cytoplasm, host perinuclear region (By similarity). Virion (By similarity). Secreted (By similarity). Note=Predominantly found in the paranuclear area, probably in the TGN. Correct localization requires PACS1. Also associates with the inner plasma membrane through its N-terminal domain. Nef stimulates its own export via the release of exosomes. Also incorporated in virions at a rate of about 10 molecules per virion, where it is cleaved (By similarity).

Post-translational modifications:

The virion-associated Nef proteins are cleaved by the viral protease to release the soluble C-terminal core protein. Nef is probably cleaved concomitantly with viral structural proteins on maturation of virus particles (By similarity).

Phosphorylated on serine residues, probably by host PKC.

Similarity:

Belongs to the lentivirus primate group Nef protein family.

Important Note:

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