

## **Product datasheet for TA388677**

## nef Rabbit Polyclonal Antibody

### **Product data:**

**Product Type:** Primary Antibodies

Applications: WE

Recommended Dilution: Recommended dilution: WB:1:500-1:2000

Reactivity: Human
Host: Rabbit
Isotype: IgG

Clonality: Polyclonal

Immunogen: Recombinant Human immunodeficiency virus 1 Protein Nef protein (1-209AA)

**Formulation:** Preservative: 0.03% Proclin 300

Constituents: 50% Glycerol, 0.01M PBS, pH 7.4

**Concentration:** lot specific

**Purification:** Antigen Affinity Purified

**Conjugation:** Unconjugated

**Storage:** Upon receipt, store at -20°C or -80°C. Avoid repeated freeze.

**Stability:** 1 year from dispatch.

Database Link: <u>A0A1L4CSI1</u>

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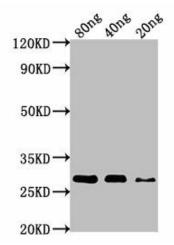


#### Background:

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. Extracellular Nef protein targets CD4+ T-lymphocytes for apoptosis by interacting with CXCR4 surface receptors. 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. In infected CD4+ Tlymphocytes, 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 networkassociated 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). Down-regulates host SERINC3 and SERINC5 thereby excluding these proteins from the viral particles. Virion infectivity is drastically higher when SERINC3 or SERINC5 are excluded from the viral envelope, because these host antiviral proteins impare the membrane fusion event necessary for subsequent virion penetration. 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/ASK1. Decreases the half-life of TP53, 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 host BAD.



# **Product images:**



Western Blot
Positive WB detected in Recombinant protein
All lanes: nef antibody at 1:1000
Secondary
Goat polyclonal to rabbit IgG at 1/50000 dilution
Predicted band size: 28 kDa

Observed band size: 28 kDa