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[Primary Antibody]

Rabbit Anti-APOBEC3G antibody

Catalog Number: SL15407R

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	APOBEC3G
Immunogen:	KLH conjugated synthetic peptide derived from human APOBEC3G: 201-300/384
Purification:	affinity purified by Protein A
Concentration:	1mg/ml
Storage:	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol. Shipped at 4℃. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles.
Applications:	WB=1:500-2000 （Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	Human
Predicted MW.:	46kDa
Subcellular Location:	The nucleus cytoplasmic
Background:	APOBEC3G is a member of a family of enzymes that have potent DNA mutator activity. APOBEC3G deaminates deoxycytosine to deoxyuracil in the minus strand of HIV-1 DNA, resulting in G to A hypermutation in the plus strand of DNA. Thus, APOBEC3G provides a mechanism for innate immunity to retroviruses and also likely contributes to sequence variation observed in many viruses. Viral infectivity factor (Vif) imparts APOBEC3G resistance to HIV through impaired translation of APOBEC3G mRNA and accel-erated posttranslational degradation of APOBEC3G by the 26S proteasome. Inter-estingly, HIV-1 Vif cannot form a complex with APOBEC3G of mouse origin as it does with the human protein, and thus mouse APOBEC3G functions as a potent inhibitor of wild type HIV-1 replication, where human APOBEC3G is only able to inhibit Vif-deficient HIV-1 replication. This implies that induction of APOBEC3G activity or a method of blocking its interaction with Vif may provide a method

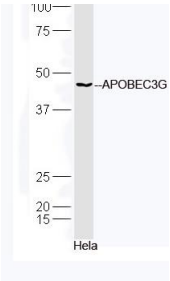
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	for therapeutic intervention. CEM15 is a 429 amino acid mouse protein that is thought to function as an ortholog of human APOBEC3G. **Function:** DNA deaminase (cytidine deaminase) which acts as an inhibitor of retrovirus replication and retrotransposon mobility via deaminase-dependent and -independent mechanisms. Exhibits potent antiviral activity against vif-deficient HIV-1. After the penetration of retroviral nucleocapsids into target cells of infection and the initiation of reverse transcription, it can induce the conversion of cytosine to uracil in the minus-sense single-strand viral DNA, leading to G-to-A hypermutations in the subsequent plus-strand viral DNA. The resultant detrimental levels of mutations in the proviral genome, along with a deamination-independent mechanism that works prior to the proviral integration, together exert efficient antiretroviral effects in infected target cells. Selectively targets single-stranded DNA and does not deaminate double-stranded DNA or single-or double-stranded RNA. Exhibits antiviral activity also against simian immunodeficiency viruses (SIVs), hepatitis B virus (HBV), equine infectious anemia virus (EIAV), xenotropic MuLV-related virus (XMRV) and simian foamy virus (SFV). May inhibit the mobility of LTR and non-LTR retrotransposons. **Subunit:** Homodimer. Homooligomer. Can bind RNA to form ribonucleoprotein complexes of high-molecular-mass (HMM) or low-molecular-mass (LMM). HMM is inactive and heterogeneous in protein composition because of binding nonselectively to cellular RNAs, which in turn are associated with variety of cellular proteins. The LMM form which is enzymatically active has few or no RNAs associated. Its ability to form homooligomer is distinct from its ability to assemble into HMM. Interacts with APOBEC3B, APOBEC3F, MOV10, EIF2C2/AGO2, EIF4E, EIF4ENIF1, DCP2 and DDX6 in an RNA-dependent manner. Interacts with EIF2C1/AGO1, EIF2C3/AGO3 and PKA/PRKACA. Interacts with HIV-1 VIF and reverse transcriptase/ribonuclease H. Interacts with hepatitis B virus capsid protein. **Subcellular Location:** Cytoplasm. Nucleus. Cytoplasm, P-body. Note=Mainly cytoplasmic. Small amount are found in the nucleus. During HIV-1 infection, virion-encapsidated in absence of HIV-1 VIF. **Tissue Specificity:** Expressed in spleen, testes, ovary and peripheral blood leukocytes and CD4+ lymphocytes. Also expressed in non-permissive peripheral blood mononuclear cells, and several tumor cell lines; no expression detected in permissive lymphoid and non-lymphoid cell lines. Exists only in the LMM form in peripheral blood-derived resting CD4 T-cells and monocytes, both of which are refractory to HIV-1 infection. LMM is

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	converted to a HMM complex when resting CD4 T cells are activated or when monocytes are induced to differentiate into macrophages. This change correlates with increased susceptibility of these cells to HIV-1 infection. **Post-translational modifications:** Ubiquitinated in the presence of HIV-1 VIF. Association with VIF targets the protein for proteolysis by the ubiquitin-dependent proteasome pathway. Phosphorylation at Thr-32 reduces its binding to HIV-1 VIF and subsequent ubiquitination and degradation thus promoting its antiviral activity. **Similarity:** Belongs to the cytidine and deoxycytidylate deaminase family. Contains 2 CMP/dCMP deaminase zinc-binding domains. **Database links:** **Entrez Gene:** 200316 Human **Entrez Gene:** 60489 Human Omim: 607113 Human **SwissProt:** Q9HC16 Human **Unigene:** 659809 Human **Unigene:** 660143 Human

[VALIDATION IMAGES]



Protein: Hela(human)cell lysate at 40ug;
Primary: rabbit Anti-APOBEC3G (SL15407R) at 1:300;
Secondary: HRP conjugated Goat-Anti-rabbit IgG(SL0295G-HRP) at 1: 5000;
Predicted band size: 46 kD
Observed band size: 46 kD

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