

APOBEC3G Antibody (C-term) Blocking peptide
Synthetic peptide
Catalog # BP13299b**Specification**

APOBEC3G Antibody (C-term) Blocking peptide - Product InformationPrimary Accession [Q9HC16](#)**APOBEC3G Antibody (C-term) Blocking peptide - Additional Information****Gene ID** 60489**Other Names**

DNA dC->dU-editing enzyme APOBEC-3G, 354-, APOBEC-related cytidine deaminase, APOBEC-related protein, ARCD, APOBEC-related protein 9, ARP-9, CEM-15, CEM15, Deoxycytidine deaminase, A3G, APOBEC3G

Target/Specificity

The synthetic peptide sequence used to generate the antibody AP13299b was selected from the C-term region of APOBEC3G. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

APOBEC3G Antibody (C-term) Blocking peptide - Protein Information**Name** 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.

Cellular 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 Location

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 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.

APOBEC3G Antibody (C-term) Blocking peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

APOBEC3G Antibody (C-term) Blocking peptide - Images**APOBEC3G Antibody (C-term) Blocking peptide - Background**

This gene is a member of the cytidine deaminase gene family. It is one of seven related genes or pseudogenes found in a cluster, thought to result from gene duplication, on chromosome 22. Members of the cluster encode proteins that are structurally and functionally related to the C to U RNA-editing cytidine deaminase APOBEC1. It is thought that the proteins may be RNA editing enzymes and have roles in growth or cell cycle control. Alternatively spliced transcript variants encoding different isoforms have been identified.

APOBEC3G Antibody (C-term) Blocking peptide - References

Hu, C., et al. J. Virol. 84(22):11981-11993(2010) Miyagi, E., et al. J. Virol. 84(21):11067-11075(2010) Albin, J.S., et al. J. Virol. 84(19):10209-10219(2010) Lassen, K.G., et al. J. Biol. Chem. 285(38):29326-29335(2010) Dang, Y., et al. J. Virol. 84(17):8561-8570(2010)