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CEM15 Antibody (N-term) Blocking Peptide
Synthetic peptide
Catalog # BP1351a

Specification

CEM15 Antibody (N-term) Blocking Peptide - Product Information

Primary Accession Q9HC16

CEM15 Antibody (N-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 AP1351a was selected from the N-term region of human CEM15. 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.

CEM15 Antibody (N-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.

CEM15 Antibody (N-term) Blocking Peptide - Protocols

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

- Blocking Peptides

CEM15 Antibody (N-term) Blocking Peptide - Images

CEM15 Antibody (N-term) Blocking Peptide - Background

CEM15 is a member of the cytidine deaminase family. It is the product of 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. CEM15 has been found to be a specific inhibitor of human immunodeficiency virus-1 (HIV-1) infectivity.

CEM15 Antibody (N-term) Blocking Peptide - References

Kao, S., et al., J. Virol. 77(21):11398-11407 (2003).Stopak, K., et al., Mol. Cell 12(3):591-601 (2003).Mangeat, B., et al., Nature 424(6944):99-103 (2003).Zhang, H., et al., Nature 424(6944):94-98 (2003).Wedekind, J.E., et al., Trends Genet. 19(4):207-216 (2003).