

APOBEC3G (CEM15) Antibody (Center E133)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP1351b**Specification**

APOBEC3G (CEM15) Antibody (Center E133) - Product Information

Application	WB, IHC-P,E
Primary Accession	O9HC16
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	46408
Antigen Region	118-148

APOBEC3G (CEM15) Antibody (Center E133) - 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

This APOBEC3G (CEM15) antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 118-148 amino acids from the Central region of human APOBEC3G (CEM15).

Dilution

WB~~1:1000

IHC-P~~1:50~100

E~~Use at an assay dependent concentration.

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

APOBEC3G (CEM15) Antibody (Center E133) is for research use only and not for use in diagnostic or therapeutic procedures.

APOBEC3G (CEM15) Antibody (Center E133) - Protein Information

Name APOBEC3G {ECO:0000303|PubMed:14557625, ECO:0000312|HGNC:HGNC:17357}

Function DNA deaminase (cytidine deaminase) which acts as an inhibitor of retrovirus replication and retrotransposon mobility via deaminase- dependent and -independent mechanisms (PubMed:[12808465](#), PubMed:[16527742](#), PubMed:[17121840](#), PubMed:[18288108](#), PubMed:[18849968](#), PubMed:[19153609](#), PubMed:[21123384](#), PubMed:[22791714](#), PubMed:[25542899](#)). Exhibits potent antiviral activity against Vif-deficient HIV-1 (PubMed:[12167863](#), PubMed:[12859895](#), PubMed:[14557625](#), PubMed:[20219927](#), PubMed:[21835787](#), PubMed:[22807680](#), PubMed:[22915799](#), PubMed:[23097438](#), PubMed:[23152537](#), PubMed:[31397674](#)). 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 (PubMed:[12808465](#), PubMed:[12808466](#), PubMed:[12809610](#), PubMed:[12970355](#), PubMed:[14528300](#), PubMed:[22807680](#)). 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 (PubMed:[12808465](#), PubMed:[12808466](#), PubMed:[12809610](#), PubMed:[12970355](#), PubMed:[14528300](#)). Selectively targets single-stranded DNA and does not deaminate double-stranded DNA or single- or double-stranded RNA (PubMed:[12808465](#), PubMed:[12809610](#), PubMed:[12970355](#), PubMed:[14528300](#)). 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) (PubMed:[15031497](#), PubMed:[16378963](#), PubMed:[18448976](#), PubMed:[19458006](#), PubMed:[20335265](#)). May inhibit the mobility of LTR and non-LTR retrotransposons (PubMed:[16527742](#)).

Cellular Location

Cytoplasm. Nucleus Cytoplasm, P-body. Note=Mainly cytoplasmic (PubMed:[16527742](#), PubMed:[16699599](#), PubMed:[21835787](#)). Small amount are found in the nucleus (PubMed:[18667511](#)). During HIV-1 infection, virion-encapsidated in absence of HIV-1 Vif (PubMed:[12859895](#))

Tissue Location

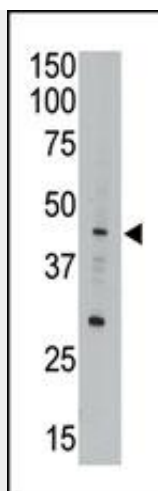
Expressed in spleen, testes, ovary and peripheral blood leukocytes and CD4+ lymphocytes. Also expressed in non-permissive peripheral blood mononuclear cells, and several tumor cell lines; no expression detected in permissive lymphoid and non-lymphoid cell lines Exists only in the LMM form in peripheral 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 (CEM15) Antibody (Center E133) - Protocols

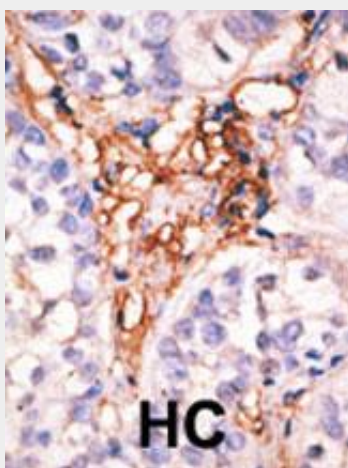
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

APOBEC3G (CEM15) Antibody (Center E133) - Images



The anti-CEM15 Pab (Cat. #AP1351b) is used in Western blot to detect CEM15 in A549 cell lysate.



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by AEC staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

APOBEC3G (CEM15) Antibody (Center E133) - 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.

APOBEC3G (CEM15) Antibody (Center E133) - 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).