Double-stranded RNA-specific adenosine deaminase

Catalog No: #AP78788

Package Size: #AP78788-1 50ug #AP78788-2 100ug #AP78788-3 1mg

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Description

Product Name	Double-stranded RNA-specific adenosine deaminase
Brief Description	Recombinant Protein
Host Species	E.coli
Purification	Greater than 90% by SDS-PAGE
Species Reactivity	Human
Immunogen Description	Recombinant protein
Other Names	ADAR1,DSRAD,G1P1,IFI4,136 kDa double-stranded RNA-binding protein,Interferon-inducible protein 4,K88DSRBP,p136,IFI-4
Accession No.	P55265Gene name:ADAR
Uniprot	P55265
GeneID	103;
Tag Info	His
Formulation	50mM NaH2PO4, 500mM NaCl Buffer with 500mM Imidazole,10%glycerol(PH8.0)
Storage	Store at -20C. (Avoid repeated freezing and thawing.)Repeated freezing and thawing is not recommended. Store working aliquots at 4°C for up to one week.

Background

Catalyzes the hydrolytic deamination of adenosine to inosine in double-stranded RNA (dsRNA) referred to as A-to-I RNA editing. This may affect gene expression and function in a number of ways that include mRNA translation by changing codons and hence the amino acid sequence of proteins; pre-mRNA splicing by altering splice site recognition sequences; RNA stability by changing sequences involved in nuclease recognition; genetic stability in the case of RNA virus genomes by changing sequences during viral RNA replication; and RNA structure-dependent activities such as microRNA production or targeting or protein-RNA interactions. Can edit both viral and cellular RNAs and can edit RNAs at multiple sites (hyper-editing) or at specific sites (site-specific editing). Its cellular RNA substrates include: bladder cancer-associated protein (BLCAP), neurotransmitter receptors for glutamate (GRIA2) and serotonin (HTR2C) and GABA receptor (GABRA3). Site-specific RNA editing of transcripts encoding these proteins results in amino acid substitutions which consequently alters their functional activities. Exhibits low-level editing at the GRIA2 Q/R site, but edits efficiently at the R/G site and HOTSPOT1. Its viral RNA substrates include: hepatitis C virus (HCV), vesicular stomatitis virus (VSV), measles virus (MV), hepatitis delta virus (HDV), and human immunodeficiency virus type 1 (HIV-1). Exhibits either a proviral (HDV, MV, VSV and HIV-1) or an antiviral effect (HCV) and this can be editing-dependent (HDV and HCV), editing-independent (VSV and MV) or both (HIV-1). Impairs HCV replication via RNA editing at multiple sites. Enhances the replication of MV, VSV and HIV-1 through an editing-independent mechanism via suppression of EIF2AK2/PKR activation and function. Stimulates both the release and infectivity of HIV-1 viral particles by an editing-dependent mechanism where it associates with viral RNAs and edits adenosines in the 5'UTR and the Rev and Tat coding sequence. Can enhance viral replication of HDV via A-to-I editing at a site designated as amber/W, thereby changing an UAG amber stop codon to an UIG tryptophan (W) codon that permits synthesis of the large delta antigen (L-HDAg) which has a key role in the assembly of viral particles. However, high levels of ADAR1 inhibit HDV replication.

References

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Note: This product is for in vitro research use only