Product Name: ADAR1 (16R16) Rabbit Monoclonal

Antibody

Catalog #: AMRe06603



Summary

Production Name ADAR1 (16R16) Rabbit Monoclonal Antibody

Description Rabbit Monoclonal Antibody

Host Rabbit
Application WB,ELISA
Reactivity Human

Performance

Conjugation	Unconjugated
Modification	Unmodified
Isotype	IgG
Clonality	Monoclonal
Form	Liquid
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.
	Rabbit IgG in phosphate buffered saline , pH 7.4, 150mM NaCl, 0.02% New type
Buffer	preservative N and 50% glycerol. Store at +4°C short term. Store at -20°C long term.
	Avoid freeze / thaw cycle.
Purification	Affinity purification

Immunogen

Gene Name ADAR

Alternative Names ADAR; Adar1; AGS6; DRADA; Dsh; Dsrad; IFI4; P136;

 Gene ID
 103.0

 SwissProt ID
 P55265.

Application

Dilution Ratio WB 1:500-1:2000

Molecular Weight 136kDa

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Background

Converts multiple adenosines to inosines and creates I/U mismatched base pairs in double-helical RNA substrates without apparent sequence specificity. Catalyzes the hydrolytic deamination of adenosine to inosine in double-stranded RNA (dsRNA) referred to as A-to-I RNA editing (PubMed: 7972084, PubMed:7565688, PubMed:12618436). 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), editingindependent (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.

Research Area

Image Data

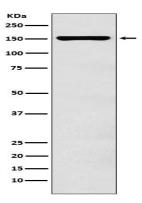
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Western blot analysis of ADAR1 expression in Ramos cell lysate.

Note

For research use only.

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