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ADAR1 Rabbit mAb

Catalog # AP77910

Product Information

Application	WB, IHC-P, IF, FC, ICC
Primary Accession	P55265
Reactivity	Human
Host	Rabbit
Clonality	Monoclonal Antibody
Isotype	IgG
Conjugate	Unconjugated
Immunogen	A synthesized peptide derived from human ADAR1
Purification	Affinity Chromatography
Calculated MW	136066

Additional Information

Gene ID	103
Other Names	ADAR
Dilution	WB~~1/500-1/1000 IHC-P~~N/A IF~~1:50~200 FC~~1:10~50 ICC~~N/A
Format	Liquid in 10mM PBS, pH 7.4, 150mM sodium chloride, 0.05% BSA, 0.02% sodium azide and 50% glycerol.
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.

Protein Information

Name	ADAR
Synonyms	ADAR1, DSRAD, G1P1, IFI4
Function	Catalyzes the hydrolytic deamination of adenosine to inosine in double-stranded RNA (dsRNA) referred to as A-to-I RNA editing (PubMed:12618436, PubMed:7565688, PubMed:7972084). 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 since the translational machinery read the inosine as a guanosine; 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.

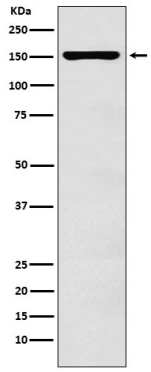
Cellular Location

[Isoform 1]: Cytoplasm. Nucleus. Note=Shuttles between the cytoplasm and nucleus (PubMed:24753571, PubMed:7565688). Nuclear import is mediated by TNPO1 (PubMed:24753571).

Tissue Location

Ubiquitously expressed, highest levels were found in brain and lung (PubMed:7972084). Isoform 5 is expressed at higher levels in astrocytomas as compared to normal brain tissue and expression increases strikingly with the severity of the tumor, being higher in the most aggressive tumors.

Images



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