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ADAR2 Polyclonal Antibody

Catalog # AP68302

Product Information

Application	WB, IHC-P, IF, ICC, E
Primary Accession	P78563
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	80763

Additional Information

Gene ID	104
Other Names	ADARB1; ADAR2; DRADA2; RED1; Double-stranded RNA-specific editase 1; RNA-editing deaminase 1; RNA-editing enzyme 1; dsRNA adenosine deaminase
Dilution	WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. ELISA: 1/20000. Not yet tested in other applications. IHC-P~~N/A IF~~1:50~200 ICC~~N/A E~~N/A
Format	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.
Storage Conditions	-20℃

Protein Information

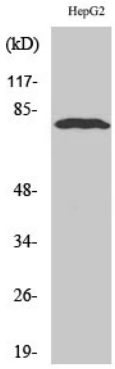
Name	ADARB1 (HGNC:226)
Function	Catalyzes the hydrolytic deamination of adenosine to inosine in double-stranded RNA (dsRNA) referred to as A-to-I RNA editing. This may affect gene expression and function in a number of ways that include mRNA translation by changing codons and hence the amino acid sequence 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 GRIK2) and serotonin (HTR2C), GABA receptor (GABRA3) and potassium voltage-gated channel (KCNA1). Site-specific RNA editing of transcripts encoding these proteins results in amino acid substitutions which

	consequently alter their functional activities. Edits GRIA2 at both the Q/R and R/G sites efficiently but converts the adenosine in hotspot1 much less efficiently. Can exert a proviral effect towards human immunodeficiency virus type 1 (HIV-1) and enhances its replication via both an editing-dependent and editing-independent mechanism. The former involves editing of adenosines in the 5'UTR while the latter occurs via suppression of EIF2AK2/PKR activation and function. Can inhibit cell proliferation and migration and can stimulate exocytosis.
Cellular Location	Nucleus. Nucleus, nucleolus. Note=Shuttles between nucleoli and the nucleoplasm. [Isoform 2]: Nucleus. Nucleus, nucleolus
Tissue Location	Highly expressed in brain and heart and at lower levels in placenta. Fair expression in lung, liver and kidney. Detected in brain, heart, kidney, lung and liver (at protein level)

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 GRIK2) and serotonin (HTR2C), GABA receptor (GABRA3) and potassium voltage-gated channel (KCNA1). Site- specific RNA editing of transcripts encoding these proteins results in amino acid substitutions which consequently alter their functional activities. Edits GRIA2 at both the Q/R and R/G sites efficiently but converts the adenosine in hotspot1 much less efficiently. Can exert a proviral effect towards human immunodeficiency virus type 1 (HIV-1) and enhances its replication via both an editing-dependent and editing-independent mechanism. The former involves editing of adenosines in the 5'UTR while the latter occurs via suppression of EIF2AK2/PKR activation and function. Can inhibit cell proliferation and migration and can stimulate exocytosis.

Images



Western Blot analysis of various cells using ADAR2 Polyclonal Antibody cells nucleus extracted by Minute TM Cytoplasmic and Nuclear Fractionation kit (SC-003,Inventbiotech,MN,USA).