10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

ADAR2 Antibody

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP50962

Product Information

Application	WB, ICC, IHC-P
Primary Accession	P78563
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	80 KDa

Additional Information

Other Names	Double-stranded RNA-specific editase 1, RNA-editing deaminase 1, RNA-editing enzyme 1, dsRNA adenosine deaminase, ADARB1, ADAR2, DRADA2, RED1
Target/Specificity	KLH-conjugated synthetic peptide encompassing a sequence within the center region of human ADAR2. The exact sequence is proprietary.
Dilution	WB~~1:1000 ICC~~N/A IHC-P~~N/A
Format	0.01M PBS, pH 7.2, 0.09% (W/V) Sodium azide, Glycerol 50%
Storage	Store at -20 °C.Stable for 12 months from date of receipt

Protein Information

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.

References

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