PRODUCT INFORMATION

ADAR2 Long Isoform (human, recombinant)

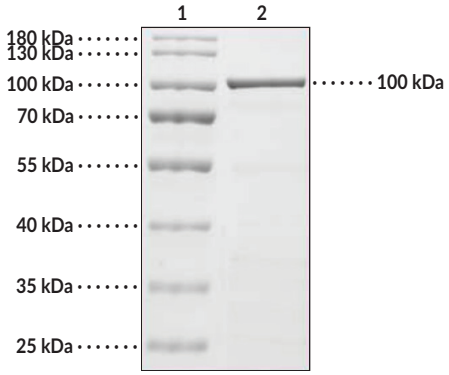
Item No. 42937

Overview and Properties

Synonyms:	ADARB1, Adenosine Deaminase RNA-specific B1, DRADA2B
Source:	Recombinant human N-terminal DYKDDDDK-tagged ADAR2L expressed in insect cells
Amino Acids:	2-741 (full length)
Uniprot No.:	P78563
Molecular Weight:	~85 kDa
Storage:	-80°C (as supplied)
Stability:	≥1 year
Purity:	≥85% estimated by SDS-PAGE
Supplied in:	50 mM Tris, pH 7.5, with 150 mM sodium chloride, 1 mM EDTA, 0.5 mg/ml DYKDDDDK-tag, 1 mM DTT, and 10% glycerol
Protein Concentration:	***batch specific*** mg/ml

Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Image



Lane 1: MW Markers
Lane 2: ADAR2 Long Isoform

SDS-PAGE Analysis of ADAR2 Long Isoform. This protein has a calculated molecular weight of approximately 85 kDa. It has an apparent molecular weight of approximately 100 kDa by SDS-PAGE.

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Description

Adenosine deaminase acting on RNA 2 (ADAR2) is an aminohydrolase and member of the ADAR protein family.[1,2] It is composed of an N-terminal nuclear export (NES) region, two RNA-binding domains, and a C-terminal deaminase domain.[2] ADAR2 is widely expressed and its two predominant isoforms, ADAR2 short (ADAR2S) and ADAR2 long (ADAR2L), form homodimers and localize primarily to the nucleus.[1,3] ADAR2L performs site-specific editing of dsRNA by converting adenosine to inosine, a post-transcriptional modification known as A-to-I editing that predominantly targets RNA encoding neuroreceptors and ion channels. ADAR2 has a variety of RNA substrates, including *Gria2*, which encodes the AMPA receptor GluA2 subunit, also known as GluR-B, and the adenosine at the Q/R site is edited by ADAR2 in nearly all transcripts, resulting in a codon change from CAG (glutamine) to CIG (arginine).[3-5] This edit reduces calcium permeability of the AMPA receptor and reduces trafficking of the receptor to the postsynaptic membrane.[3,5] Mice lacking *AdarB1*, the gene encoding Adar2, have reduced *Gria2* pre-mRNA editing, develop early-onset epilepsy, and are viable only up to three weeks.[5] Biallelic *ADARB1* variants are associated with intractable epilepsy, microcephaly, and intellectual disability.[3] *ADARB1* expression is decreased in ovarian cancer tumors and correlates with poor prognosis and shorter progression-free survival.[6] Cayman's ADAR2L (human, recombinant) protein has a calculated molecular weight of approximately 85 kDa. By SDS-PAGE, the apparent molecular mass of the protein is approximately 100 kDa.

References

1. Zinshteyn, B. and Nishikura, K. Adenosine-to-inosine RNA editing. *Wiley Interdiscip. Rev. Syst. Biol. Med.* **1(2)**, 202-209 (2009).
2. Rehwinkel, J. and Mehdipour, P. ADAR1: From basic mechanisms to inhibitors. *Trends Cell Biol.* **35(1)**, 59-73 (2025).
3. Tan, T.Y., Sedmík, J., Fitzgerald, M.P., *et al.* Bi-allelic *ADARB1* variants associated with microcephaly, intellectual disability, and seizures. *Am. J. Hum. Genet.* **106(4)**, 467-483 (2020).
4. Macbeth, M.R., Schubert, H.L., Vandemark, A.P., *et al.* Inositol hexakisphosphate is bound in the ADAR2 core and required for RNA editing. *Science* **309(5740)**, 1534-1539 (2005).
5. Higuchi, M., Maas, S., Single, F.N., *et al.* Point mutation in an AMPA receptor gene rescues lethality in mice deficient in the RNA-editing enzyme ADAR2. *Nature* **406(6791)**, 78-81 (2000).
6. Zhu, W., Xu, Z., Huang, M., *et al.* Downregulated ADARB1 facilitates cell proliferation, invasion and has effect on the immune regulation in ovarian cancer. *Front. Bioeng. Biotechnol.* **9:792911**, (2021).

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