

Rabbit Polyclonal Antibody to Human ADK (Antigen Affinity Purified)



Catalog Number: 13149-RP02

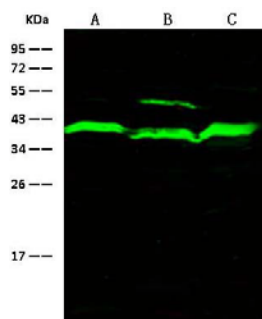
General Information	
Immunogen:	Recombinant Human ADK protein (Catalog#13149-H20B)
Ig Type:	Rabbit IgG
Applications:	WB, ELISA
Specificity:	Human ADK
Formulation:	0.2 µm filtered solution in PBS with 5% trehalose
Storage:	< -20° C

Preparation

Produced in rabbits immunized with purified, recombinant Human ADK (rh ADK; Catalog#13149-H20B; AAH03568.1; Met 1-His 345). ADK specific IgG was purified by Human ADK affinity chromatography.

Applications

Western blot – This antibody can be used at 2-5 µg/mL with the appropriate secondary reagents to detect Human ADK in WB.



Lanes	A	B	C
Items			
Sample (whole cell lysate)	RAW264. 7	293T	NIH/3T3
Sample Volume (cells/lane)	5×10^5	5×10^5	5×10^5
Gel	13% SDS-PAGE reducing gel		
Recommended Concentration	2-5 µ g/ml		
Secondary Antibody	Dylight 800-Labeled Antibody To Rabbit IgG (H+L), at 1:5000 dilution.		

Direct ELISA – This antibody can be used at 0.1-0.2 µg/mL with the appropriate secondary reagents to detect Human ADK. The detection limit for Human ADK is approximately 0.00975 ng/well.

Storage

This antibody can be stored at 2°C-8°C for one month without detectable loss of activity. Antibody products are stable for twelve months from date of receipt when stored at -20°C to -70°C. **Preservative-Free.**

Sodium azide is recommended to avoid contamination (final concentration 0.05%-0.1%). It is toxic to cells and should be disposed of properly. **Avoid repeated freeze-thaw cycles.**

Background

Adenosine kinase (ADK) belongs to the family of transferases. Adenosine kinase (ADK) is the key enzyme in adenosine metabolism and catalyzes ATP and adenosine into two products: ADP and AMP. Two isoforms of the enzyme adenosine kinase (AdK), which differ at their N-terminal ends, are found in mammalian cells. In adult brain, ADK is primarily present in astrocytes. Several lines of experimental evidence support a critical role of ADK in different types of brain injury associated with astrogliosis, which is also a prominent morphologic feature of temporal lobe epilepsy (TLE). It has been suggested that dysregulation of ADK in astrocytes is a common pathologic hallmark of TLE. Moreover, in vitro data suggest the existence of an additional layer of modulatory crosstalk between the astrocyte-based adenosine cycle and inflammation. ADK also contributes to CK homeostasis in vivo. Both siRNA and amiRNA mediated silencing of ADK led to impaired root growth, small, crinkled rosette leaves and reduced apical dominance.

Reference

1. Aronica E. et al., 2011, Epilepsia. 52 (9): 1645-1655.
2. Kuettel S. et al., 2011, PLoS Negl Trop Dis. 5 (5): 1164.
3. Cui XA. et al., 2011, BMC Biochem. 12 (1): 22.

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