Data Sheet (Cat.No.TMPY-04473)

TargetMol

ADK Protein, Human, Recombinant (His & GST)

General Information

Synonyms: adenosine kinase;AK

Protein Construction: A DNA sequence encoding the human ADK isoform short (AAH03568.1) (Met 1-His 345) was fused with the N-terminal polyhistidine-tagged GST tag at the N-terminus. Predicted N terminal: Met

Species: Human

Expression Host: Baculovirus Insect Cells

Accession: AAH03568.1

Molecular Weight: 68 kDa (predicted); 60 kDa (reducing conditions)

QC Testing

Biological Activity: Kinase activity untested

Purity: > 90 % as determined by SDS-PAGE

Endotoxin: < 1.0 EU/μg of the protein as determined by the LAL method.

Formulation: Supplied as sterile 50 mM Tris, 100 mM NaCl, pH 8.0, 10% gly, 0.3 mM DTT.

Preparation and Storage

Reconstitution:

A Certificate of Analysis (CoA) containing reconstitution instructions is included with the products. Please refer to the CoA for detailed information.

Stability & Storage:

It is recommended to store the product under sterile conditions at -20°C to -80°C. Samples are stable for up to 12 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

Proteins are shipped with blue ice.

Protein 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. It has been shown that the two ADK isoforms differ only in their first exons and the promoter regions; hence they arise via differential splicing of their first exons with the other exons common to both isoforms. 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.

Reference

Aronica E, et al. (2011) Upregulation of adenosine kinase in astrocytes in experimental and human temporal lobe epilepsy. Epilepsia.52 (9): 1645-55.

Kuettel S, et al. (2011) Crystal structures of T. b. rhodesiense adenosine kinase complexed with inhibitor and activator: implications for catalysis and hyperactivation. PLoS Negl Trop Dis. 5 (5): e1164.

Cui XA, et al. (2011) Molecular characterization of Chinese hamster cells mutants affected in adenosine kinase and showing novel genetic and biochemical characteristics. BMC Biochem. 12 (1): 22.