Recombinant Human ADK Protein (His &GST Tag)

Catalog Number: PKSH030331

Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

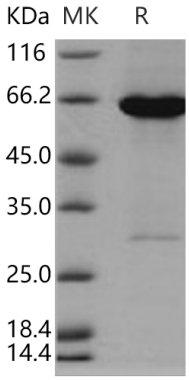
Description

Species	Human
Source	Baculovirus-Insect Cells-derived Human ADK protein Met 1-His 345, with an N-terminal His & GST
Calculated MW	68.0 kDa
Observed MW	60 kDa
Accession	AAH03568.1
Bio-activity	Not validated for activity

Properties

Purity	> 90 % as determined by reducing SDS-PAGE.
Concentration	Subject to label value.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles.
Shipping	This product is provided as liquid. It is shipped at frozen temperature with blue ice/gel packs. Upon receipt, store it immediately at < - 20°C.
Formulation	Supplied as sterile solution of 50mM Tris, 100mM NaCl, pH 8.0, 10% glycerol, 0.3mM DTT

Data



> 90 % as determined by reducing SDS-PAGE.

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.