

## Recombinant Human ADK Protein (His & GST Tag)

**Catalog Number:** PKSH030331

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

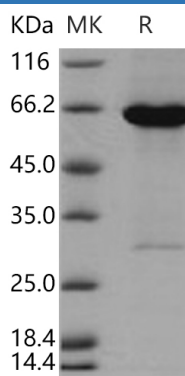
### Description

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

### Properties

|                      |   |
|----------------------|---|
| <b>Purity</b>        | > 90 % as determined by reducing SDS-PAGE.  |
| <b>Concentration</b> | Subject to label value.   |
| <b>Endotoxin</b>     | < 1.0 EU per µg of the protein as determined by the LAL method.   |
| <b>Storage</b>       | Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles.  |
| <b>Shipping</b>      | 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. |
| <b>Formulation</b>   | 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.

### For Research Use Only

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