# Recombinant Human AKT1 protein (SUMO,His Tag)

**Catalog Number: PDEH101093**

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

## Description

| | |
|---|---|
| **Species** | Human |
| **Source** | E.coli-derived Human AKT1 protein Met1-Ala480, with an N-terminal Sumo & His |
| **Calculated MW** | 72.7 kDa |
| **Observed MW** | 75 kDa |
| **Accession** | P31749 |
| **Bio-activity** | Not validated for activity |

## Properties

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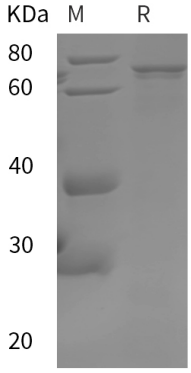
|---|---|
| **Purity** | > 95% as determined by reducing SDS-PAGE. |
| **Endotoxin** | < 10 EU/mg of the protein as determined by the LAL method |
| **Storage** | Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 ℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months. |
| **Shipping** | This product is provided as lyophilized powder which is shipped with ice packs. |
| **Formulation** | Lyophilized from a 0.2 μm filtered solution in PBS with 5% Trehalose and 5% Mannitol. |
| **Reconstitution** | It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis. |

## Data




> 95 % as determined by reducing SDS-PAGE.

## Background

v-akt murine thymoma viral oncogene homolog 1 (AKT1), or protein kinase B-alpha (PKB-ALPHA) is a serine-threonine protein kinase, belonging to the Protein Kinase Superfamily. AKT1 is a major mediator of the responses to insulin, insulin-like growth factor 1 (IGF1), and glucose. AKT1 also plays a key role in the regulation of both muscle cell hypertrophy and atrophy. AKT1 activity is required for physiologic cardiac growth in response to IGF1 stimulation or exercise training. In contrast, AKT1 activity was found to antagonize pathologic cardiac growth that occurs in response to endothelin 1 stimulation or pressure overload. AKT1 selectively promotes physiological cardiac growth while AKT2 selectively promotes insulin-stimulated cardiac glucose metabolism. AKT1 deletion prevented tumor initiation as well as tumor progression, coincident with decreased Akt signaling in tumor tissues. AKT1 is the primary Akt isoform activated by mutant K-ras in lung tumors, and that AKT3 may oppose AKT1 in lung tumorigenesis and lung tumor progression. A number of separate studies have implicated AKT1 as an inhibitor of breast epithelial cell motility and invasion. AKT1 may have a dual role in tumorigenesis, acting not only pro-oncogenically by suppressing apoptosis but also anti-oncogenically by suppressing invasion and metastasis.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