

### **PRAS40 Polyclonal Antibody**

Catalog # AP72033

### **Specification**

#### **PRAS40 Polyclonal Antibody - Product Information**

Application WB
Primary Accession Q96B36

Reactivity Human, Mouse, Rat

Host Rabbit Clonality Polyclonal

#### PRAS40 Polyclonal Antibody - Additional Information

**Gene ID 84335** 

#### **Other Names**

AKT1S1; PRAS40; Proline-rich AKT1 substrate 1; 40 kDa proline-rich AKT substrate

#### Dilution

WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. ELISA: 1/5000. Not yet tested in other applications.

#### **Format**

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.

# **Storage Conditions**

-20°C

#### **PRAS40 Polyclonal Antibody - Protein Information**

Name AKT1S1 {ECO:0000312|EMBL:AAH16043.1}

#### **Function**

Negative regulator of the mechanistic target of rapamycin complex 1 (mTORC1), an evolutionarily conserved central nutrient sensor that stimulates anabolic reactions and macromolecule biosynthesis to promote cellular biomass generation and growth (PubMed:<a href="http://www.uniprot.org/citations/17510057" target="\_blank">17510057</a>, PubMed:<a href="http://www.uniprot.org/citations/17386266" target="\_blank">17386266</a>, PubMed:<a href="http://www.uniprot.org/citations/17277771" target="\_blank">17277771</a>, PubMed:<a href="http://www.uniprot.org/citations/29236692" target="\_blank">29236692</a>). In absence of insulin and nutrients, AKT1S1 associates with the mTORC1 complex and directly inhibits mTORC1 activity by blocking the MTOR substrate- recruitment site (PubMed:<a href="http://www.uniprot.org/citations/29236692" target="\_blank">29236692</a>). In response to insulin and nutrients, AKT1S1 dissociates from mTORC1 (PubMed:<a href="http://www.uniprot.org/citations/17386266" target="\_blank">17386266</a>, PubMed:<a href="http://www.uniprot.org/citations/18372248" target="\_blank">18372248</a>). Its activity is dependent on its phosphorylation state and binding to 14-3-3 (PubMed:<a



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href="http://www.uniprot.org/citations/18372248" target=" blank">18372248</a>). May also play a role in nerve growth factor-mediated neuroprotection (By similarity).

#### **Cellular Location**

Cytoplasm, cytosol {ECO:0000250|UniProtKB:Q9D1F4}. Note=Found in the cytosolic fraction of the brain. {ECO:0000250|UniProtKB:Q9D1F4}

#### **Tissue Location**

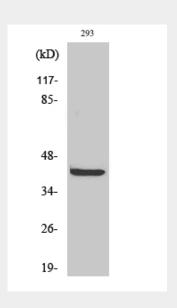
Widely expressed with highest levels of expression in liver and heart. Expressed at higher levels in cancer cell lines (e.g. A-549 and HeLa) than in normal cell lines (e.g. HEK293)

#### **PRAS40 Polyclonal Antibody - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

## PRAS40 Polyclonal Antibody - Images



#### PRAS40 Polyclonal Antibody - Background

Subunit of mTORC1, which regulates cell growth and survival in response to nutrient and hormonal signals. mTORC1 is activated in response to growth factors or amino acids. Growth factor-stimulated mTORC1 activation involves a AKT1-mediated phosphorylation of TSC1-TSC2, which leads to the activation of the RHEB GTPase that potently activates the protein kinase activity of mTORC1. Amino acid-signaling to mTORC1 requires its relocalization to the lysosomes mediated by the Ragulator complex and the Rag GTPases. Activated mTORC1 up-regulates protein synthesis by phosphorylating key regulators of mRNA translation and ribosome synthesis. mTORC1





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phosphorylates EIF4EBP1 and releases it from inhibiting the elongation initiation factor 4E (eiF4E). mTORC1 phosphorylates and activates S6K1 at 'Thr-389', which then promotes protein synthesis by phosphorylating PDCD4 and targeting it for degradation. Within mTORC1, AKT1S1 negatively regulates mTOR activity in a manner that is dependent on its phosphorylation state and binding to 14-3-3 proteins. Inhibits RHEB-GTP-dependent mTORC1 activation. Substrate for AKT1 phosphorylation, but can also be activated by AKT1-independent mechanisms. May also play a role in nerve growth factor-mediated neuroprotection.