

S6K (RPS6KB1) Antibody (S404) Blocking peptide
Synthetic peptide
Catalog # BP3289d**Specification**

S6K (RPS6KB1) Antibody (S404) Blocking peptide - Product InformationPrimary Accession [P23443](#)**S6K (RPS6KB1) Antibody (S404) Blocking peptide - Additional Information****Gene ID** 6198**Other Names**

Ribosomal protein S6 kinase beta-1, S6K-beta-1, S6K1, 70 kDa ribosomal protein S6 kinase 1, P70S6K1, p70-S6K 1, Ribosomal protein S6 kinase I, Serine/threonine-protein kinase 14A, p70 ribosomal S6 kinase alpha, p70 S6 kinase alpha, p70 S6K-alpha, p70 S6KA, RPS6KB1, STK14A

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP3289d](/product/products/AP3289d) was selected from the S404 region of human RPS6KB1. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

S6K (RPS6KB1) Antibody (S404) Blocking peptide - Protein Information**Name** RPS6KB1**Synonyms** STK14A**Function**

Serine/threonine-protein kinase that acts downstream of mTOR signaling in response to growth factors and nutrients to promote cell proliferation, cell growth and cell cycle progression (PubMed: [11500364](http://www.uniprot.org/citations/11500364), PubMed: [12801526](http://www.uniprot.org/citations/12801526), PubMed: [14673156](http://www.uniprot.org/citations/14673156), PubMed: [15071500](http://www.uniprot.org/citations/15071500), PubMed: [15341740](http://www.uniprot.org/citations/15341740), PubMed: [16286006](http://www.uniprot.org/citations/16286006)),

PubMed:17052453, PubMed:17053147, PubMed:17936702, PubMed:18952604, PubMed:19085255, PubMed:19720745, PubMed:19935711, PubMed:19995915, PubMed:23429703, PubMed:28178239, PubMed:22017876). Regulates protein synthesis through phosphorylation of EIF4B, RPS6 and EEF2K, and contributes to cell survival by repressing the pro-apoptotic function of BAD (PubMed:11500364, PubMed:12801526, PubMed:14673156, PubMed:15071500, PubMed:15341740, PubMed:16286006, PubMed:17052453, PubMed:17053147, PubMed:17936702, PubMed:18952604, PubMed:19085255, PubMed:19720745, PubMed:19935711, PubMed:19995915, PubMed:23429703, PubMed:28178239, PubMed:22017876). Under conditions of nutrient depletion, the inactive form associates with the EIF3 translation initiation complex (PubMed:16286006). Upon mitogenic stimulation, phosphorylation by the mechanistic target of rapamycin complex 1 (mTORC1) leads to dissociation from the EIF3 complex and activation (PubMed:16286006). The active form then phosphorylates and activates several substrates in the pre-initiation complex, including the EIF2B complex and the cap-binding complex component EIF4B (PubMed:16286006). Also controls translation initiation by phosphorylating a negative regulator of EIF4A, PDCD4, targeting it for ubiquitination and subsequent proteolysis (PubMed:17053147). Promotes initiation of the pioneer round of protein synthesis by phosphorylating POLDIP3/SKAR (PubMed:15341740). In response to IGF1, activates translation elongation by phosphorylating EEF2 kinase (EEF2K), which leads to its inhibition and thus activation of EEF2 (PubMed:11500364). Also plays a role in feedback regulation of mTORC2 by mTORC1 by phosphorylating RICTOR, resulting in the inhibition of mTORC2 and AKT1 signaling (PubMed:19720745, PubMed:19935711, PubMed:19995915). Also involved in feedback regulation of mTORC1 and mTORC2 by phosphorylating DEPTOR (PubMed:22017876). Mediates cell survival by phosphorylating the pro- apoptotic protein BAD and suppressing its pro-apoptotic function (By similarity). Phosphorylates mitochondrial URI1 leading to dissociation of a URI1-PPP1CC complex (PubMed:17936702). The free mitochondrial PPP1CC can then dephosphorylate

RPS6KB1 at Thr-412, which is proposed to be a negative feedback mechanism for the RPS6KB1 anti-apoptotic function (PubMed:17936702). Mediates TNF-alpha-induced insulin resistance by phosphorylating IRS1 at multiple serine residues, resulting in accelerated degradation of IRS1 (PubMed:18952604). In cells lacking functional TSC1-2 complex, constitutively phosphorylates and inhibits GSK3B (PubMed:17052453). May be involved in cytoskeletal rearrangement through binding to neurabin (By similarity). Phosphorylates and activates the pyrimidine biosynthesis enzyme CAD, downstream of MTOR (PubMed:23429703). Following activation by mTORC1, phosphorylates EPRS and thereby plays a key role in fatty acid uptake by adipocytes and also most probably in interferon-gamma-induced translation inhibition (PubMed:28178239).

Cellular Location

Synapse, synaptosome. Mitochondrion outer membrane. Mitochondrion. Note=Colocalizes with URI1 at mitochondrion [Isoform Alpha II]: Cytoplasm.

Tissue Location

Widely expressed..

S6K (RPS6KB1) Antibody (S404) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

S6K (RPS6KB1) Antibody (S404) Blocking peptide - Images

S6K (RPS6KB1) Antibody (S404) Blocking peptide - Background

RPS6KB1 is a member of the RSK (ribosomal S6 kinase) family of serine/threonine kinases. This kinase contains 2 non-identical kinase catalytic domains and phosphorylates several residues of the S6 ribosomal protein. The kinase activity of this protein leads to an increase in protein synthesis and cell proliferation. Amplification of the region of DNA encoding the gene for RPS6KB1 and overexpression of this kinase are seen in some breast cancer cell lines.

S6K (RPS6KB1) Antibody (S404) Blocking peptide - References

Adem, C., et al., Genes Chromosomes Cancer 41(1):1-11 (2004).Suzuki, Y., et al., Genome Res. 14(9):1711-1718 (2004).Gomez-Cambronero, J., et al., Leuk. Res. 28(7):755-762 (2004).Raught, B., et al., EMBO J. 23(8):1761-1769 (2004).Miyakawa, M., et al., Endocr. J. 50(1):77-83 (2003).