

**RPS6KA3 Antibody**  
**Mouse Monoclonal Antibody (Mab)**  
**Catalog # AM2008b****Specification**

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**RPS6KA3 Antibody - Product Information**

Application	WB,E
Primary Accession	<a href="#">P51812</a>
Other Accession	<a href="#">P18654</a> , <a href="#">NP_004577.1</a>
Reactivity	Human
Predicted	Mouse
Host	Mouse
Clonality	Monoclonal
Isotype	IgG1
Calculated MW	83736

**RPS6KA3 Antibody - Additional Information****Gene ID** 6197**Other Names**

Ribosomal protein S6 kinase alpha-3, S6K-alpha-3, 90 kDa ribosomal protein S6 kinase 3, p90-RSK 3, p90RSK3, Insulin-stimulated protein kinase 1, ISPK-1, MAP kinase-activated protein kinase 1b, MAPK-activated protein kinase 1b, MAPKAP kinase 1b, MAPKAPK-1b, Ribosomal S6 kinase 2, RSK-2, pp90RSK2, RPS6KA3, ISPK1, MAPKAPK1B, RSK2

**Target/Specificity**

Purified His-tagged RPS6KA3 protein(Fragment) was used to produced this monoclonal antibody.

**Dilution**

WB~~1:500~1000

**Format**

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

**Storage**

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

**Precautions**

RPS6KA3 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**RPS6KA3 Antibody - Protein Information****Name** RPS6KA3**Synonyms** ISPK1, MAPKAPK1B, RSK2

**Function** Serine/threonine-protein kinase that acts downstream of ERK (MAPK1/ERK2 and MAPK3/ERK1) signaling and mediates mitogenic and stress-induced activation of the transcription factors CREB1, ETV1/ER81 and NR4A1/NUR77, regulates translation through RPS6 and EIF4B phosphorylation, and mediates cellular proliferation, survival, and differentiation by modulating mTOR signaling and repressing pro- apoptotic function of BAD and DAPK1 (PubMed:[16213824](#), PubMed:[16223362](#), PubMed:[17360704](#), PubMed:[9770464](#)). In fibroblast, is required for EGF-stimulated phosphorylation of CREB1 and histone H3 at 'Ser-10', which results in the subsequent transcriptional activation of several immediate-early genes (PubMed:[10436156](#), PubMed:[9770464](#)). In response to mitogenic stimulation (EGF and PMA), phosphorylates and activates NR4A1/NUR77 and ETV1/ER81 transcription factors and the cofactor CREBBP (PubMed:[16223362](#)). Upon insulin-derived signal, acts indirectly on the transcription regulation of several genes by phosphorylating GSK3B at 'Ser-9' and inhibiting its activity (PubMed:[8250835](#)). Phosphorylates RPS6 in response to serum or EGF via an mTOR-independent mechanism and promotes translation initiation by facilitating assembly of the preinitiation complex (PubMed:[17360704](#)). In response to insulin, phosphorylates EIF4B, enhancing EIF4B affinity for the EIF3 complex and stimulating cap-dependent translation (PubMed:[18508509](#), PubMed:[18813292](#)). Is involved in the mTOR nutrient-sensing pathway by directly phosphorylating TSC2 at 'Ser-1798', which potentially inhibits TSC2 ability to suppress mTOR signaling, and mediates phosphorylation of RPTOR, which regulates mTORC1 activity and may promote rapamycin- sensitive signaling independently of the PI3K/AKT pathway (PubMed:[18722121](#)). Mediates cell survival by phosphorylating the pro- apoptotic proteins BAD and DAPK1 and suppressing their pro-apoptotic function (PubMed:[16213824](#)). Promotes the survival of hepatic stellate cells by phosphorylating CEBPB in response to the hepatotoxin carbon tetrachloride (CCl4) (PubMed:[18508509](#), PubMed:[18813292](#)). Is involved in cell cycle regulation by phosphorylating the CDK inhibitor CDKN1B, which promotes CDKN1B association with 14-3-3 proteins and prevents its translocation to the nucleus and inhibition of G1 progression (By similarity). In LPS-stimulated dendritic cells, is involved in TLR4- induced macropinocytosis, and in myeloma cells, acts as effector of FGFR3-mediated transformation signaling, after direct phosphorylation at Tyr-529 by FGFR3 (By similarity). Negatively regulates EGF-induced MAPK1/3 phosphorylation via phosphorylation of SOS1 (By similarity). Phosphorylates SOS1 at 'Ser-1134' and 'Ser-1161' that create YWHAB and YWHAЕ binding sites and which contribute to the negative regulation of MAPK1/3 phosphorylation (By similarity). Phosphorylates EPHA2 at 'Ser- 897', the RPS6KA-EPHA2 signaling pathway controls cell migration (PubMed:[26158630](#)). Acts as a regulator of osteoblast differentiation by mediating phosphorylation of ATF4, thereby promoting ATF4 transactivation activity (By similarity).

**Cellular Location**

Nucleus. Cytoplasm

**Tissue Location**

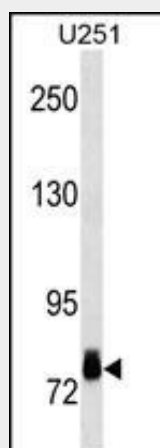
Expressed in many tissues, highest levels in skeletal muscle

**RPS6KA3 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 Cytometry](#)
- [Cell Culture](#)

**RPS6KA3 Antibody - Images**



RPS6KA3 Antibody (Cat. #AM2008b) western blot analysis in U251 cell line lysates (35µg/lane). This demonstrates the RPS6KA3 antibody detected the RPS6KA3 protein (arrow).

#### **RPS6KA3 Antibody - Background**

This gene encodes a member of the RSK (ribosomal S6 kinase) family of serine/threonine kinases. This kinase contains 2 non-identical kinase catalytic domains and phosphorylates various substrates, including members of the mitogen-activated kinase (MAPK) signalling pathway. The activity of this protein has been implicated in controlling cell growth and differentiation. Mutations in this gene have been associated with Coffin-Lowry syndrome (CLS).

#### **RPS6KA3 Antibody - References**

Peng, C., et al. FASEB J. 24(9):3490-3499(2010)  
Vigneron, S., et al. Oncogene 29(24):3566-3574(2010)  
Kang, S., et al. J. Clin. Invest. 120(4):1165-1177(2010)  
Yerges, L.M., et al. J. Bone Miner. Res. 24(12):2039-2049(2009)  
Doehn, U., et al. Mol. Cell 35(4):511-522(2009)