10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

AKT2 Antibody (C-term)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7029B

Specification

AKT2 Antibody (C-term) - Product Information

Application	**IF, WB, IHC-P, FC,E**
Primary Accession	P31751
Other Accession	P47197, Q60823
Reactivity	**Human**
Predicted	**Mouse, Rat**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Calculated MW	**55769**
Antigen Region	**416-444**

AKT2 Antibody (C-term) - Additional Information

Gene ID 208

Other Names
RAC-beta serine/threonine-protein kinase, Protein kinase Akt-2, Protein kinase B beta, PKB beta, RAC-PK-beta, AKT2

Target/Specificity
This AKT2 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 416-444 amino acids from the C-terminal region of human AKT2.

Dilution
IF~~1:10~50
WB~~1:1000
IHC-P~~1:50~100
FC~~1:10~50

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
AKT2 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

AKT2 Antibody (C-term) - Protein Information

Name AKT2

Function AKT2 is one of 3 closely related serine/threonine-protein kinases (AKT1, AKT2 and AKT3) called the AKT kinase, and which regulate many processes including metabolism, proliferation, cell survival, growth and angiogenesis. This is mediated through serine and/or threonine phosphorylation of a range of downstream substrates. Over 100 substrate candidates have been reported so far, but for most of them, no isoform specificity has been reported. AKT is responsible of the regulation of glucose uptake by mediating insulin-induced translocation of the SLC2A4/GLUT4 glucose transporter to the cell surface. Phosphorylation of PTPN1 at 'Ser-50' negatively modulates its phosphatase activity preventing dephosphorylation of the insulin receptor and the attenuation of insulin signaling. Phosphorylation of TBC1D4 triggers the binding of this effector to inhibitory 14-3-3 proteins, which is required for insulin-stimulated glucose transport. AKT regulates also the storage of glucose in the form of glycogen by phosphorylating GSK3A at 'Ser-21' and GSK3B at 'Ser-9', resulting in inhibition of its kinase activity. Phosphorylation of GSK3 isoforms by AKT is also thought to be one mechanism by which cell proliferation is driven. AKT regulates also cell survival via the phosphorylation of MAP3K5 (apoptosis signal-related kinase). Phosphorylation of 'Ser-83' decreases MAP3K5 kinase activity stimulated by oxidative stress and thereby prevents apoptosis. AKT mediates insulin-stimulated protein synthesis by phosphorylating TSC2 at 'Ser-939' and 'Thr-1462', thereby activating mTORC1 signaling and leading to both phosphorylation of 4E- BP1 and in activation of RPS6KB1. AKT is involved in the phosphorylation of members of the FOXO factors (Forkhead family of transcription factors), leading to binding of 14-3-3 proteins and cytoplasmic localization. In particular, FOXO1 is phosphorylated at 'Thr-24', 'Ser-256' and 'Ser-319'. FOXO3 and FOXO4 are phosphorylated on equivalent sites. AKT has an important role in the regulation of NF- kappa-B-dependent gene transcription and positively regulates the activity of CREB1 (cyclic AMP (cAMP)-response element binding protein). The phosphorylation of CREB1 induces the binding of accessory proteins that are necessary for the transcription of pro-survival genes such as BCL2 and MCL1. AKT phosphorylates 'Ser-454' on ATP citrate lyase (ACLY), thereby potentially regulating ACLY activity and fatty acid synthesis. Activates the 3B isoform of cyclic nucleotide phosphodiesterase (PDE3B) via phosphorylation of 'Ser-273', resulting in reduced cyclic AMP levels and inhibition of lipolysis. Phosphorylates PIKFYVE on 'Ser-318', which results in increased PI(3)P- 5 activity. The Rho GTPase-activating protein DLC1 is another substrate and its phosphorylation is implicated in the regulation cell proliferation and cell growth. AKT plays a role as key modulator of the AKT-mTOR signaling pathway controlling the tempo of the process of newborn neurons integration during adult neurogenesis, including correct neuron positioning, dendritic development and synapse formation. Signals downstream of phosphatidylinositol 3-kinase (PI(3)K) to mediate the effects of various growth factors such as platelet- derived growth factor (PDGF), epidermal growth factor (EGF), insulin and insulin-like growth factor I (IGF-I). AKT mediates the antiapoptotic effects of IGF-I. Essential for the SPATA13-mediated regulation of cell migration and adhesion assembly and disassembly. May be involved in the regulation of the placental development. Involved in the inhibition of ciliogenesis associated with RAB8-dependent cilia growth (PubMed:31204173).

Cellular Location
Cytoplasm. Nucleus. Cell membrane; Peripheral membrane protein. Early endosome {ECO:0000250|UniProtKB:Q60823} Note=Localizes within both nucleus and cytoplasm of proliferative primary myoblasts and mostly within the nucleus of differentiated primary myoblasts. By virtue of the N-terminal PH domain, is recruited to sites of the plasma membrane containing increased PI(3,4,5)P3 or PI(3,4)P2, cell membrane targeting is also facilitared by interaction with CLIP3. Colocalizes with WDFY2 in early endosomes (By similarity) {ECO:0000250|UniProtKB:Q60823}

Tissue Location
Expressed in all cell types so far analyzed.

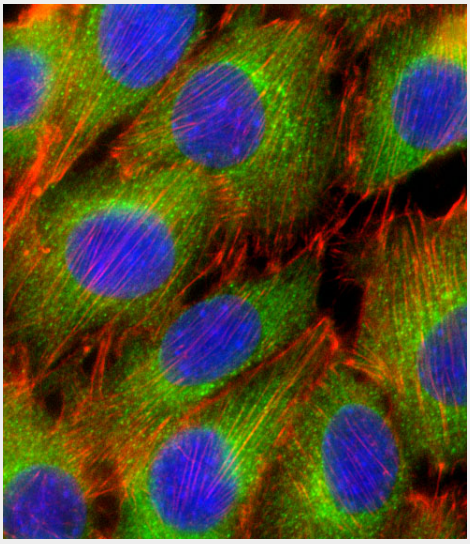
AKT2 Antibody (C-term) - Protocols

10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

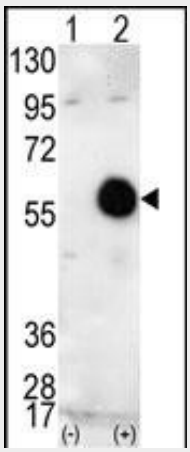
Provided below are standard protocols that you may find useful for product applications.

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

AKT2 Antibody (C-term) - Images

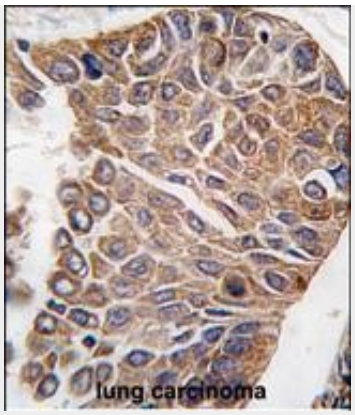


Fluorescent confocal image of C2C12 cell stained with AKT2 Antibody (C-term)(Cat#AP7029b).C2C12 cells were fixed with 4% PFA (20 min), permeabilized with Triton X-100 (0.1%, 10 min), then incubated with AKT2 primary antibody (1:25, 1 h at 37℃). For secondary antibody, Alexa Fluor® 488 conjugated donkey anti-rabbit antibody (green) was used (1:400, 50 min at 37℃).Cytoplasmic actin was counterstained with Alexa Fluor® 555 (red) conjugated Phalloidin (7units/ml, 1 h at 37℃). Nuclei were counterstained with DAPI (blue) (10 µg/ml, 10 min).AKT2 immunoreactivity is localized to Cytoplasm significantly.

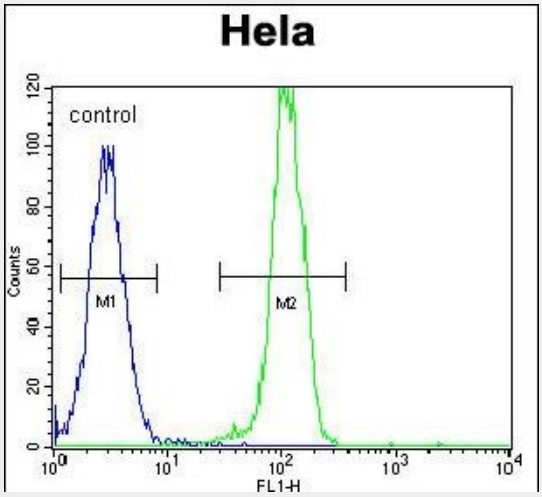


Western blot analysis of AKT2 (arrow) using AKT2 Antibody (C-term) (Cat.#AP7029b). 293 cell lysates (2 ug/lane) either nontransfected (Lane 1) or transiently transfected with the AKT2 gene (Lane 2) (Origene Technologies).

10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999



Formalin-fixed and paraffin-embedded human lung carcinoma tissue reacted with AKT2 antibody (C-term), which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated.



AKT2 Antibody (C-term) (Cat. #AP7029b) flow cytometric analysis of Hela cells (right histogram) compared to a negative control cell (left histogram).FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

AKT2 Antibody (C-term) - Background

AKT2 is a protein belonging to a subfamily of serine/threonine kinases containing SH2-like (Src homology 2-like) domains. AKT2 has been shown to be amplified and overexpressed in 2 of 8 ovarian carcinoma cell lines and 2 of 15 primary ovarian tumors. Overexpression of AKT2 contributes to the malignant phenotype of a subset of human ductal pancreatic cancers. AKT2 is a general protein kinase capable of phophorylating several known proteins.

AKT2 Antibody (C-term) - References

George, S., et al., Science 304(5675):1325-1328 (2004).
Xu, X., et al., Oncol. Rep. 11(1):25-32 (2004).
Shim, D., et al., Arch. Biochem. Biophys. 425(2):214-220 (2004).
Li, X., et al., Gastroenterology 126(1):122-135 (2004).
Vojtek, A.B., et al., Mol. Cell. Biol. 23(13):4417-4427 (2003).