AKT Antibody – Rabbit Polyclonal

PRODUCT OVERVIEW

Product Name	Akt, Polyclonal Antibody
Full Product Name	Anti-Phospho-Akt (Thr308) Rabbit pAb
Product Gene Name	anti-Akt antibody
Product Synonym Names	AKT1; AKT1_HUMAN; AKT2; AKT3; PKB alpha; PKB; PKB beta; PKBalpha; PRKBA; PRKBB; PRKBG; Protein kinase B; Protein kinase B beta; Protein kinase B gamma; Proto-oncogene c-Akt; RAC alpha; RAC; RAC gamma; RAC PK alpha; RAC PK beta; RAC PK gamma; RAC-alpha serine/threonine-protein kinase; RAC-PK-alpha; STK2.
Clonality	Polyclonal
Host	Rabbit
Species Reactivity	Mouse
Purity/Purification	Affinity purified
Preparation and Storage	Aliquot and store at-20 degree C. Avoid repeated freeze/thaw cycles.
Molecular Weight	60
OMIM	114480
3D Structure	ModBase 3D Structure for P31749
Immunogen	DGATMKp-TFCGTPE
Storage Buffer	10mM PBS with 0.02% sodium azide, 50% glycerol.
ISO Certification	Manufactured in an ISO 9001:2015 Certified Laboratory.
Applications Tested/Suitable for anti-Akt antibody	Western Blot (WB)
Related Product Information for anti-Akt antibody	Plays a role as a 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.
Other Notes	Small volumes of anti-Akt antibody vial(s) may occasionally become entrapped in the seal of the product vial during shipment and storage. If necessary, briefly centrifuge the vial on a tabletop centrifuge to dislodge any liquid in the container`s cap. Certain products may require to ship with dry ice and additional dry ice fee may apply.
Searchable Terms for Akt purchase	MBS8533364 is an antibody targeting Akt. Also known as Anti-Phospho-Akt (Thr308) Rabbit pAb. Reactive in Mouse. Host: Rabbit.

ADDITIONAL DETAILS

NCBI/Uniprot data below describe general gene information for Akt. It may not necessarily be applicable to this product.

NCBI GI #	62241013
NCBI GeneID	207
NCBI Accession #	NP_001014431.1
UniProt Primary Accession #	P31749
UniProt Secondary Accession #	Q9BWB6; B2RAM5; B7Z5R1

UniProt Related Accession #	P31749
GenBank Nucleotide #	NM_001014432
NCBI Official Full Name	RAC-alpha serine/threonine-protein kinase
NCBI Official Synonym Full Names	AKT serine/threonine kinase 1
NCBI Official Symbol	AKT1
NCBI Official Synonym Symbols	AKT; PKB; RAC; CWS6; PRKBA; PKB-ALPHA; RAC-ALPHA
UniProt Protein Name	RAC-alpha serine/threonine-protein kinase
UniProt Synonym Protein Names	Protein kinase B; PKB; Protein kinase B alpha; PKB alpha; Proto-oncogene c-Akt; RAC-PK-alpha
Protein Family	AKT-interacting protein
UniProt Gene Name	AKT1
UniProt Synonym Gene Names	PKB; RAC; PKB; PKB alpha
NCBI Protein Information	RAC-alpha serine/threonine-protein kinase
NCBI Summary for Akt	The serine-threonine protein kinase encoded by the AKT1 gene is catalytically inactive in serum-starved primary and immortalized fibroblasts. AKT1 and the related AKT2 are activated by platelet-derived growth factor. The activation is rapid and specific, and it is abrogated by mutations in the pleckstrin homology domain of AKT1. It was shown that the activation occurs through phosphatidylinositol 3-kinase. In the developing nervous system AKT is a critical mediator of growth factor-induced neuronal survival. Survival factors can suppress apoptosis in a transcription-independent manner by activating the serine/threonine kinase AKT1, which then phosphorylates and inactivates components of the apoptotic machinery. Mutations in this gene have been associated with the Proteus syndrome. Multiple alternatively spliced transcript variants have been found for this gene. [provided by RefSeq, Jul 2011]

UniProt Comments for Akt	AKT1 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 PI3P-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 (PI3K) 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. Phosphorylates STK4/MST1 at 'Thr-120' and 'Thr-387' leading to inhibition of its: kinase activity, nuclear translocation, autophosphorylation and ability to phosphorylate FOXO3. Phosphorylates STK3/MST2 at 'Thr-117' and 'Thr-384' leading to inhibition of its: cleavage, kinase activity, autophosphorylation at Thr-180, binding to RASSF1 and nuclear translocation. Phosphorylates SRPK2 and enhances its kinase activity towards SRSF2 and ACIN1 and promotes its nuclear translocation. Phosphorylates RAF1 at 'Ser-259' and negatively regulates its activity. Phosphorylation of BAD stimulates its pro-apoptotic activity. Phosphorylates KAT6A at 'Thr-369' and this phosphorylation inhibits the interaction of KAT6A with PML and negatively regulates its acetylation activity towards p53/TP53.AKT1-specific substrates have been recently identified, including palladin (PALLD), which phosphorylation modulates cytoskeletal organization and cell motility; prohibitin (PHB), playing an important role in cell metabolism and proliferation; and CDKN1A, for which phosphorylation at 'Thr-145' induces its release from CDK2 and cytoplasmic relocalization. These recent findings indicate that the AKT1 isoform has a more specific role in cell motility and proliferation. Phosphorylates CLK2 thereby controlling cell survival to ionizing radiation.CautionIn light of strong homologies in the primary amino acid sequence, the 3 AKT kinases were long surmised to play redundant and overlapping roles. More recent studies has brought into question the redundancy within AKT kinase isoforms and instead pointed to isoform specific functions in different cellular events and diseases. AKT1 is more specifically involved in cellular survival pathways, by inhibiting apoptotic processes; whereas AKT2 is more specific for the insulin receptor signaling pathway. Moreover, while AKT1 and AKT2 are often implicated in many aspects of cellular transformation, the 2 isoforms act in a complementary opposing manner. The role of AKT3 is less clear, though it appears to be predominantly expressed in brain.
Research Articles on Akt	**1.** Studies indicate the role of the insulin-like growth factor 1 (IGF1) - phosphoinositide 3-kinase (PI3K)-Akt signaling pathway in regulating exercise-induced physiological cardiac hypertrophy and cardiac protection [Review].
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