兔抗 AKT1/3(Ab-437/434) 多克隆抗体

中文名称：兔抗 AKT1/3(Ab-437/434) 多克隆抗体

英文名称： Anti-AKT1/3(Ab-437/434) rabbit polyclonal antibody

别　名： AKT; PKB; RAC; CWS6; PRKBA; PKB-ALPHA; RAC-ALPHA/MPPH; PKBG; MPPH2; PRKBG; STK-2; PKB-GAMMA; RAC-gamma; RAC-PK-gamma

相关类别： 一抗

储　存： 冷冻（-20℃） 避光

宿　主： Rabbit

抗　原： AKT1/3(Ab-437/434)

反应种属： Human Mouse

标记物： Unconjugate

克隆类型： Unconjugate

技术规格

Background:	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 specificityhas 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-5

0' negatively modulates its phosphatase activity preve nting dephosphorylation of the insulin receptor and t he attenuation of insulin signaling. Phosphorylation of TBC1D4 triggers the binding of this effector to inhibit ory 14-3-3 proteins, which is required for insulin-stim ulated glucose transport. AKT regulates also the stora ge of glucose in the form of glycogen by phosphoryl ating GSK3A at 'Ser-21' and GSK3B at 'Ser-9', resultin g in inhibition of its kinase activity. Phosphorylation o f GSK3 isoforms by AKT is also thought to be one m echanism by which cell proliferation is driven. AKT reg ulates also cell survival via the phosphorylation of MA P3K5 (apoptosis signal-related kinase). Phosphorylation of 'Ser-83' decreases MAP3K5 kinase activity stimulate d by oxidative stress and thereby prevents apoptosis. AKT mediates insulin-stimulated protein synthesis by phosphorylating TSC2 at 'Ser-939' and 'Thr-1462', ther eby activating mTORC1 signaling and leading to both phosphorylation of 4E-BP1 and in activation of RPS6K B1. AKT is involved in the phosphorylation of member s of the FOXO factors (Forkhead family of transcriptio n factors), leading to binding of 14-3-3 proteins and cytoplasmic localization. In particular, FOXO1 is phosp horylated at 'Thr-24', 'Ser-256' and 'Ser-319'. FOXO3 a nd FOXO4 are phosphorylated on equivalent sites. AK T has an important role in the regulation of NF-kapp a-B-dependent gene transcription and positively regul ates the activity of CREB1 (cyclic AMP (cAMP)-respons e element binding protein). The phosphorylation of C REB1 induces the binding of accessory proteins that a re necessary for the transcription of pro-survival gene s such as BCL2 and MCL1. AKT phosphorylates 'Ser-4 54' on ATP citrate lyase (ACLY), thereby potentially re gulating ACLY activity and fatty acid synthesis. Activat es the 3B isoform ofcyclic nucleotide phosphodiestera se (PDE3B) via phosphorylation of 'Ser-273', resulting i n reduced cyclic AMP levels and inhibition of lipolysis. Phosphorylates PIKFYVE on 'Ser-318', which results in increased PI3P-5 activity. The Rho GTPase-activating p rotein DLC1 is another substrate and its phosphorylati on is implicated in the regulation cell proliferation an d cell growth. AKT plays a role as key modulator of t he AKT-mTOR signaling pathway controlling the temp o of the process of newborn neurons integration duri

	ng 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 hasa more specific role in cell motility and proliferation. Phosphorylates CLK2 thereby controlling cell survival to ionizing radiation.
Applications:	WB
Name of antibody:	AKT1/3(Ab-437/434)
Immunogen:	Synthesized non-phosphopeptide derived from human AKT1/3 around the phosphorylation site of tyrosine 437/434 (T-R-Y(p)-F-D).
Full name:	v-akt murine thymoma viral oncogene homolog 1/3
Synonyms :	AKT; PKB; RAC; CWS6; PRKBA; PKB-ALPHA; RAC-ALPHA/MPPH; PKBG; MPPH2; PRKBG; STK-2; PKB-GAMMA; RAC-gamma; RAC-PK-gamma

SwissProt:	P31749/Q9Y243
WB Predicted band size:	56 kDa
WB Positive control:	A549 cells lysate
WB Recommended dilution:	500-3000