



# Akt (phospho Ser473) Polyclonal Antibody

<b>Catalog No</b>	BYab-14292
<b>Isotype</b>	IgG
<b>Reactivity</b>	Human;Mouse;Rat;Chicken
<b>Applications</b>	IF;WB;IHC;ELISA
<b>Gene Name</b>	AKT1/AKT2/AKT3
<b>Protein Name</b>	RAC-alpha serine/threonine-protein kinase/RAC-beta serine/threonine-protein kinase/RAC-gamma serine/threonine-protein kinase
<b>Immunogen</b>	The antiserum was produced against synthesized peptide derived from human Akt around the phosphorylation site of Ser473. AA range:431-480
<b>Specificity</b>	Phospho-Akt (S473) Polyclonal Antibody detects endogenous levels of Akt protein only when phosphorylated at S473.
<b>Formulation</b>	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
<b>Source</b>	Polyclonal, Rabbit,IgG
<b>Purification</b>	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
<b>Dilution</b>	IF: 1:50-200 Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. ELISA: 1/40000. Not yet tested in other applications.
<b>Concentration</b>	1 mg/ml
<b>Purity</b>	≥90%
<b>Storage Stability</b>	-20°C/1 year
<b>Synonyms</b>	AKT1; PKB; RAC; RAC-alpha serine/threonine-protein kinase; Protein kinase B; PKB; Protein kinase B alpha; PKB alpha; Proto-oncogene c-Akt; RAC-PK-alpha; AKT2; RAC-beta serine/threonine-protein kinase; Protein kinase Akt-2; Protein kinase B
<b>Observed Band</b>	56kD
<b>Cell Pathway</b>	Cytoplasm . Nucleus . Cell membrane . Nucleus after activation by integrin-linked protein kinase 1 (ILK1). Nuclear translocation is enhanced by interaction with TCL1A. Phosphorylation on Tyr-176 by TNK2 results in its localization to the cell membrane where it is targeted for further phosphorylations on Thr-308 and Ser-473 leading to its activation and the activated form translocates to the nucleus. Colocalizes with WDFY2 in intracellular vesicles (PubMed:16792529).
<b>Tissue Specificity</b>	Expressed in prostate cancer and levels increase from the normal to the malignant state (at protein level). Expressed in all human cell types so far analyzed. The Tyr-176 phosphorylated form shows a significant increase in expression in breast cancers during the progressive stages i.e. normal to hyperplasia (ADH), ductal carcinoma in situ (DCIS), invasive ductal carcinoma

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(IDC) and lymph node metastatic (LNMM) stages.

**Function**

catalytic activity:ATP + a protein = ADP + a phosphoprotein.,disease:Defects in AKT1 are associated with breast cancer (BC) [MIM:114480]. BC is an extremely common malignancy, affecting one in eight women during their lifetime.,disease:Defects in AKT1 are associated with colorectal cancer (CRC) [MIM:114500].,disease:Defects in AKT1 are associated with susceptibility to ovarian cancer [MIM:604370]; also called susceptibility to familial breast-ovarian cancer type 1 (BROVCA1).,domain:Binding of the PH domain to the phosphatidylinositol 3-kinase alpha (PI(3)K) results in its targeting to the plasma membrane.,domain:The AGC-kinase C-terminal mediates interaction with THEM4.,enzyme regulation:Three specific sites, one in the kinase domain (Thr-308) and the two other ones in the C-terminal regulatory region (Ser-473 and Tyr-474), need to be phosphorylated for its full activation.,function:Gene

**Background**

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]

**matters needing attention**

Avoid repeated freezing and thawing!

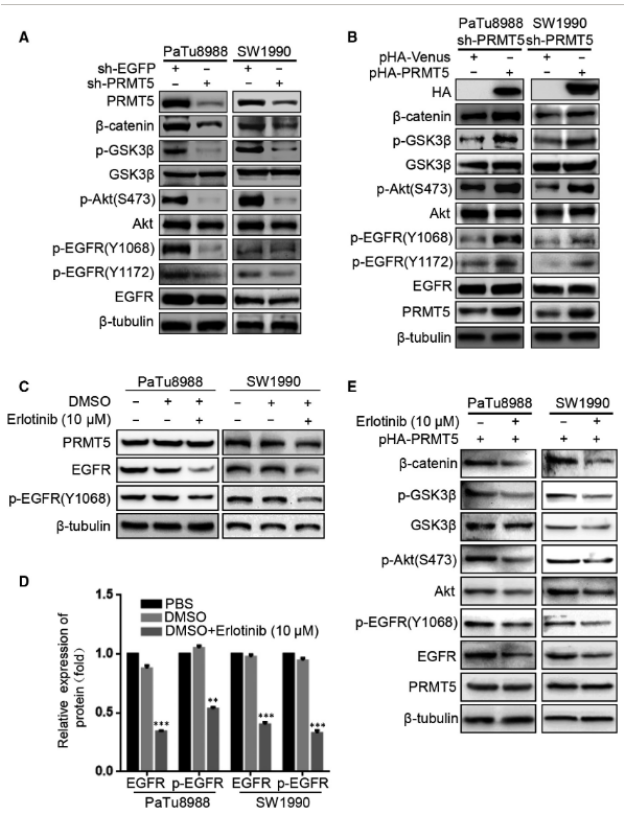
**Usage suggestions**

This product can be used in immunological reaction related experiments. For more information, please consult technical personnel.

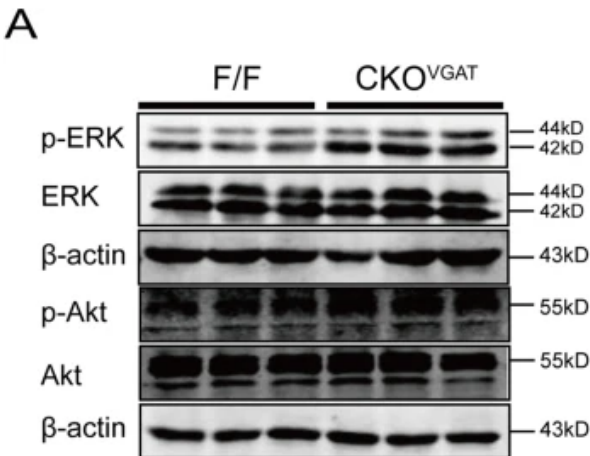
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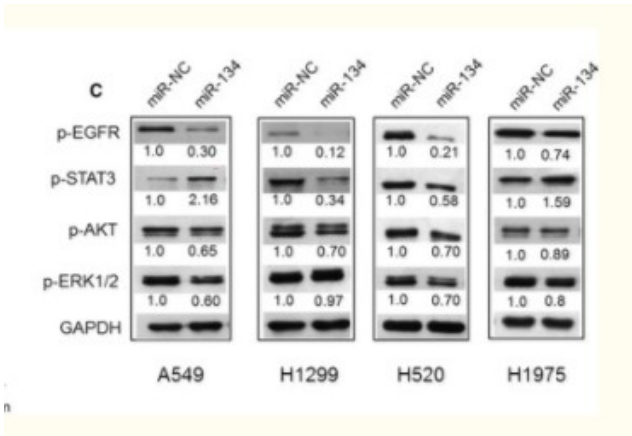
## Products Images



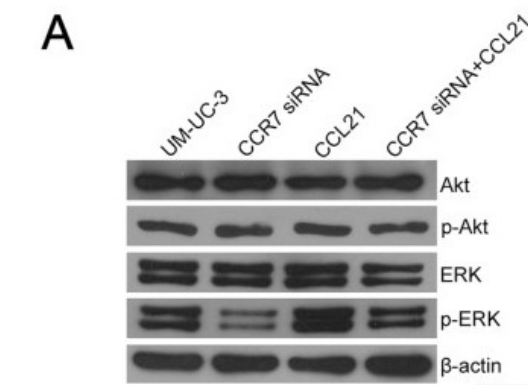
Ge, Lu, et al. "PRMT5 promotes epithelial-mesenchymal transition via EGFR-β-catenin axis in pancreatic cancer cells." *Journal of cellular and molecular medicine* 24.2 (2020): 1969-1979.



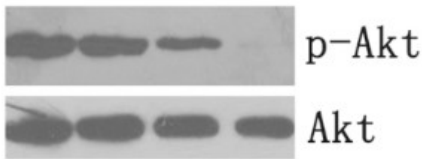
Guo, Moran, et al. "Deletion of FGF9 in GABAergic neurons causes epilepsy." *Cell death & disease* 12.2 (2021): 1-13.



Qin, Qin, et al. "miR-134 inhibits non-small cell lung cancer growth by targeting the epidermal growth factor receptor." *Journal of cellular and molecular medicine* 20.10 (2016): 1974-1983.



Xiong, Yang, et al. "CCL21/CCR7 interaction promotes cellular migration and invasion via modulation of the MEK/ERK1/2 signaling pathway and correlates with lymphatic metastatic spread and poor prognosis in urinary bladder cancer." *International journal of oncology* 51.1 (2017): 75-90.



Wang, Xinzhao, et al. "Ad-p53 enhances the sensitivity of triple-negative breast cancer MDA-MB-468 cells to the EGFR inhibitor gefitinib." *Oncology reports* 33.2 (2015): 526-532.