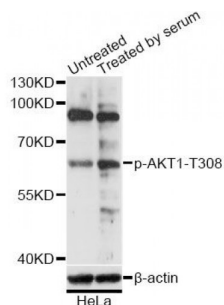


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## AKT1 (pT308) Antibody

Catalogue No.: abx000352



Western blot analysis of extracts of HeLa cells, using Phospho-AKT1-T308 antibody (abx000352) at 1/2000 dilution. HeLa cells were treated by 10% FBS for after serum-starvation overnight.

AKT1 (pT308) Antibody is a Rabbit Polyclonal antibody against AKT1 (pT308). 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.

**Target:** AKT1 (pT308)

**Reactivity:** Human, Mouse

**Host:** Rabbit

**Clonality:** Polyclonal

**Tested Applications:** WB

**Recommended dilutions:** WB: 1/500 - 1/2000. Optimal dilutions/concentrations should be determined by the end user.

**Immunogen:** Synthetic Peptide. A phospho specific peptide corresponding to residues surrounding T308 of human AKT1.

**Purification:** Affinity purified.

**Form:** Liquid

**Isotype:** IgG

**Conjugation:** Unconjugated

**Storage:** Aliquot and store at -20 °C. Avoid repeated freeze/thaw cycles.

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**Molecular Weight:** Calculated MW: 48 kDa/55 kDa  
Observed MW: 60 kDa

**Swiss Prot:** [P31749](#)

**GeneID:** [207](#)

**Gene Symbol:** AKT1

**Concentration:** > 1 mg/ml

**Buffer:** PBS, pH 7.3, 0.02% sodium azide, 50% glycerol.

**Note:** This product is for research use only.